

Applied anatomy and physiology of the nose and paranasal sinuses

Authors' affiliations:

J.B. Watelet, P. Van Cauwenberge, Department of Otorhinolaryngology, University Hospital, Ghent, Belgium

Correspondence to:

Professor Paul Van Cauwenberge
ENT Department
University Hospital of Ghent
De Pintelaan 185
B-9000 Ghent
Belgium

Introduction

Breathing through the nose is vital for most animal species and in the early life of humans. The nose has a more complex role than just a simple tube-like airway: the anatomy of the nose is quite particular and its different functions are highly specific. Its close anatomical and functional relationships with the inferior airways urge the nose to develop a complete system of defense and air conditioning. It is possible not to breathe through the nose but the consequences of this are multiple, especially with regard to the protection of the lower respiratory tract.

Anatomy

Face

The face is made up of several bones. These osseous structures are necessary to sustain different intracranial and extracranial organs such as the brain, the eyes and the pituitary gland. They anchor all the facial muscles.

Importance of the facial structures

The rigidity of the bones and cartilages, and the strong connections with the mucosa avoid the collapse of the mucosa during inspiration. The particular organization of the bony and cartilaginous structures in the nasal cavities ensure close contact and an important surface area to the inspired air. These structures also play a role in voice resonance. The close relationship between nasal functions and skull base development has been confirmed by studies in rats, showing that nasal obstruction is able to cause anatomical changes of the maxilla, the skull base and the mandibula with abnormal skeletal growth (1).

Date:

Accepted for publication 13 August 1998

To cite this article:

Watelet J.B. & Van Cauwenberge P. Applied anatomy and physiology of the nose and paranasal sinuses. *Allergy* 1999; **54**, Suppl 57, 14–25.

Copyright © Munksgaard 1999

ISSN 0105-4538

External nose

The bony roof of the anterior part of the nasal cavity consists of the nasal bones and the ascending process of the maxillary bones. The covering is completed by the upper and lower lateral cartilages. The bony-cartilaginous roof is covered by muscles. Individual and racial variations in the external nose are numerous. Ohki, in 1991, showed that the dimensions of the nostrils were statistically different in healthy Caucasian, Oriental or black adults. The width of the nose was 34.0 mm in Caucasian, 38.7 mm in Oriental and 42.5 mm in black persons ($P < 0.05$) (2).

Importance of the external nose

The bony roof of the anterior part of the nasal cavities is essential to protect the fragile mucosa inside the nose, but its main function remains esthetic. The upper lateral, alar cartilages and fibrous tissue complete the covering of the anterior part of the nasal cavities and control the entrance of air into the respiratory tract. Examination of the alar anatomy and the alar base is indispensable in the exploration of nasal obstruction (3). Their ability to deform can create sufficient resistance against the transmural pressure of inspiration (4, 5), and can modify the cross-section of the vestibule and regulate the airflow before entering into the nasal cavity. The dilator naris muscle, the nasalis muscle and the apicis nasi muscle are strongly related to respiration, contributing to the prevention of collapse of the nasal valve (6). An external nasal dilator, by increasing the nasal valve area, decreases total airway resistance and can improve exercise performance (7). Cosmetic septorhinoplasty can alter nasal patency (8). In regulating the intensity and direction of the airflow, the external nose improves olfaction. Finally, the elastic characteristics, especially of the procerus muscle and levator labii superior alaeque nasi muscle, participate in facial expressions.

Nasal cavities

The anterior part of the nasal cavity opens anteriorly in the nostril while the nose communicates posteriorly with the rhinopharynx. Usually, authors divide the nasal cavity into three parts: the nasal vestibule, the olfactory region and the respiratory region. The junction of the vestibule with the nasal cavity is called the internal nasal valve. It is situated between the caudal end of the upper alar cartilage laterally, and the septum medially. Its apical angle has an angulation of less than 15° . It is the narrowest site of the nasal cavity, only 0.3 cm^2 on each side (9, 10).

Excluding the olfactory region, which consists of the upper part of the nasal cavity and the superior turbinates, the remainder of the nasal cavity constitutes the respiratory region. The total surface area of both nasal cavities reaches about 150 cm^2 and the total volume about 15 ml. However, here also some racial and pathological considerations have to be taken into account (11, 12).

Nasal septum

The septum divides the nasal cavity into two halves. Depending on the expansion of the perpendicular plate and the vomer, i.e. the bony parts of the septum, the cartilaginous septum reaches adult dimensions at the age of 2 years (13). The bony part of the septum consists of the perpendicular plate of the ethmoid bone and the vomer while the cartilaginous part is formed by the quadrilateral cartilage. The anterior part defines the columella and the postero-superior angle has contact with the sphenoid bone. The nasal septum lays in the crista nasalis of the bony palate.

Turbinates

The lateral nasal wall supports the three turbinates (inferior, middle, superior and sometimes there is even a supreme) that divide this lateral wall into three meatus (inferior, middle, superior). Before 9 weeks of gestation, three soft-tissue elevations (the preturbinates) can be identified within the nasal cavity; they are orientated both in size and position in a similar way to the inferior, middle and superior turbinates in the adult (14). The turbinates contain cartilage at 9 weeks of gestation. The inferior turbinate ossification appears to precede that of the middle turbinate (17 weeks vs 19 weeks of gestation) (15). The head of the inferior turbinate interferes directly with the entering airflow and its tail, in case of hypertrophy, can significantly reduce the choanal size. The middle turbinate covers the ostium of the major sinuses medially, while the supreme turbinate is not always present.

Paranasal sinuses (Fig. 1)

The paranasal sinuses are air-containing cavities in the facial bones and are connected to the nasal cavity. The maxillary sinus is the most important in size: its global volume can reach 15 ml. Asymmetries between the two sides and interindividual variations are frequent. Its ostium is placed at the upper part of the cavity, and opens into the middle meatus. The superior part of the maxillary sinus supports the orbit and the posterior part is also the anterior

wall of the pterygopalatine fossa containing the maxillary artery, the sphenopalatine ganglion and branches of the trigeminal nerve and the autonomic system. The variations in form and size of the frontal sinus are numerous but usually the volume reaches 7 ml. In 3–5 % of individuals the frontal sinus is completely absent in one or both sides. Its posterior wall is the anterior wall of the anterior cranial fossa and its floor covers the orbit. The ethmoid sinuses contain six to 10 cells subdivided into anterior and posterior cells; their ostia drain, respectively, into the middle and the superior meatus. They are localized at the medial part of the orbit and the inferior part of the skull base. The sphenoid sinus is placed at the junction of the anterior and the middle cerebral fossae. Here, also, interindividual variations are often noted. Sometimes, it may be completely absent. Its ostium lays in the superior meatus. It is surrounded by the pituitary gland, the optic nerves, the internal carotid arteries and the cavernous sinuses.

Importance of the paranasal sinuses

The nasal cavities and sinuses in the child differ from the adult in size and in proportion (16). The intrinsic functions of the paranasal sinuses are controversial. None of them has been proven. They may assure harmony in facial growth and make the skull lighter (17, 18). The sinuses can be a protector of the brain. Other hypotheses seem less valid: the paranasal sinuses probably do not contribute to efficient air conditioning by increase of the contact between mucosa and inspired air, nor to speech resonance (19), nor to smell perception (20).

Histology

The epithelial lining is a physical barrier to inhaled foreign materials. It entraps and clears foreign material through coordinated events of mucus secretion and ciliary activity. It participates in immune responses to inhaled antigens and it conditions inhaled air for the maintenance of optimal physiological conditions (21–23).

Embryology

Maturation of the epithelial lining on the septum and the lateral wall precedes that of the adjacent paranasal sinuses. Before the ninth week of gestation, the nasal cavity is lined by undifferentiated cells (24). The pseudostratified ciliated epithelium appears at 9 weeks of gestation, while the process of differentiation continues throughout the next 14 weeks. The lamina propria of the nasal mucosa becomes

increasingly vascularized from the ninth week of gestation. The glandular acini and the goblet cells start to develop around the eleventh week (25). The process of this progressive differentiation is completed at the twenty-fourth week.

Epithelium

The nasal epithelium lies on a basement membrane and a lamina propria. Different types of epithelium can be found in the nasal cavity:

- 1) A *pseudostratified columnar epithelium* (respiratory epithelium), which is composed of four major types of cells: ciliated (columnar) cells, nonciliated (columnar) cells, goblet cells and basal cells. This epithelium is found in the posterior two-thirds of the nasal cavity.
- 2) A *squamous and transitional epithelium* (stratified epithelium containing cuboidal cells with microvilli) is found in the first third of the nasal cavity (26).
- 3) The epithelium of the paranasal sinuses is of the simple ciliated columnar type with a few goblet cells and glands.

Epithelial cells

The epithelial cells protect the upper and lower airways directly by mucociliary clearance. The epithelial layer of nasal mucosa is composed of four types of cells: ciliated columnar cells and nonciliated columnar cells, goblet cells and basal cells. Eighty per cent of the cell population are ciliated cells. The ratio of columnar to goblet cells is about 5:1.

The apical part of the ciliated cells is covered by cilia originating from basal bodies that also serve to anchor them to the cell. The cytoplasm contains many mitochondria. They are necessary for the energy supply of the ciliary beating. The cilia in the human nose are 0.3 µm in diameter, 7–10 µm long; there are about 100 per cell (27). A cilium is composed of a ring of nine doublet microtubules that surround two single central microtubules. Each pair of microtubules has two subfibers, A and B. Dynein arms project from subfiber A toward the next microtubule subfiber B (28, 29). Cilia beat with a frequency of 1000 strokes per minute. The beat of a cilium consists of a rapid forward beat (effective stroke) and a slow return beat (recovery beat) (30).

Goblet or mucus-secreting cells

Goblet or mucus-secreting cells produce an acidic mucin. The production of the correct amount and the viscoelasti-

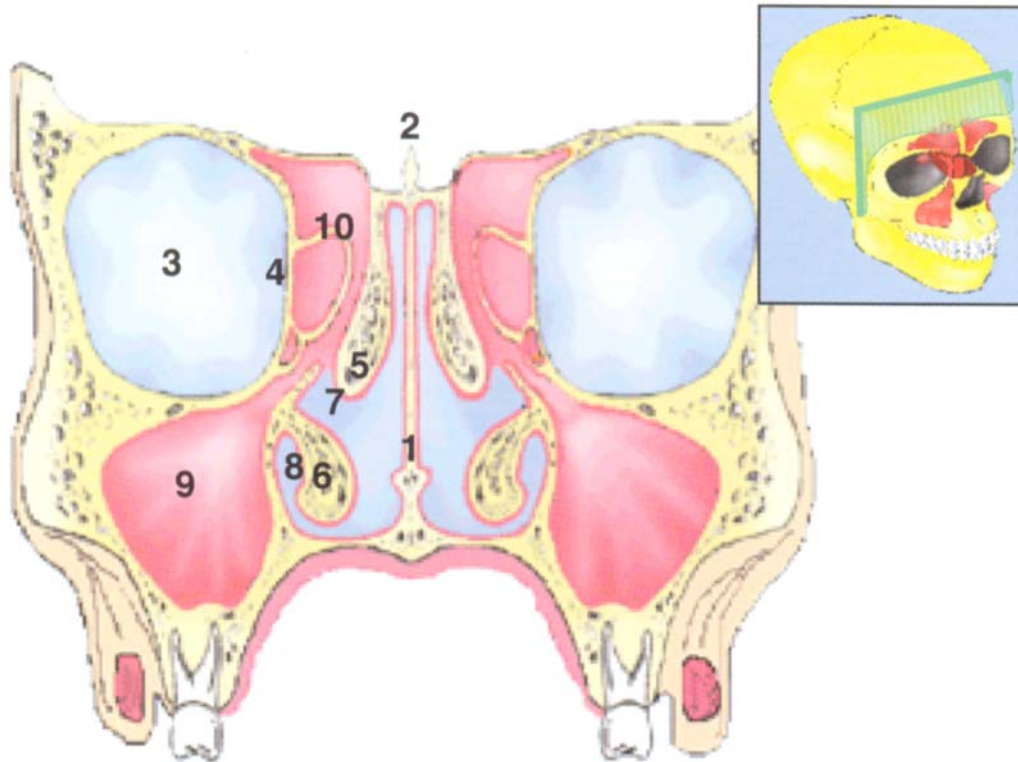


Figure 1. Anatomy of the nasal and paranasal cavities (frontal section). 1, Nasal septum; 2, crista galli; 3, orbit; 4, lamina papyracea; 5, middle turbinate; 6, inferior turbinate; 7, middle meatus; 8, inferior meatus; 9, maxillary sinus; 10, ethmoid sinus.

city of mucus are important in the maintenance of mucociliary clearance.

Basal cells

Basal cells are the progenitors of the specialized cells which populate the luminal border. Classically, they lay on the basement membrane but, on histological slides, they can give the impression of laying higher, between the other epithelial cells.

Importance of the mucociliary function

The mean velocity of the mucus flow and particle transport in a normal nose is about 5 mm/min, ranging from 0 to more than 20 mm/min [31]. Different factors can affect the ciliary function of the epithelial cells. Congenital abnormalities in the structural constitution or function of the cilia (Kartagener's triad, primary ciliary dyskinesia) are better known nowadays, but also other conditions can interfere easily with normal ciliary activity. According to Proetz, in 1956, dryness affects ciliar activity significantly. At 50% relative humidity of inspired air, ciliary action stops after 8–10 min and at 30% relative humidity of inspired air, it stops after 3–5 min [32]. Regarding the temperature, ciliary activity ceases at between 7 and 12°C. Other factors such as

locally applied drugs [33, 34], inhaled gases [35], environmental exposure to large amounts of wood dust and chromium vapors, tobacco smoke or infection [33, 36] can severely impair the ciliary function.

Epithelial cell junctions

Surface epithelial cells are in contact with each other by three types of junction: adhering junctions, tight junctions and gap junctions.

Adhering junctions include the zonula adherents, macula adherents and hemidesmosomes, which join cells to their basal lamina.

Tight junctions form a belt around the apicolateral borders of the epithelial cells and act to form a selectively permeable resistive barrier to the paracellular movement of ions, macromolecules and water.

Gap ("nexus") junctions allow cells to communicate directly; for example in the coordination of ciliary beat.

Importance of the epithelial junctions

The epithelium controls the movement of water by regulating the translocation of ions, in particular the absorption of sodium and secretion of chloride [37]. The movement of water and serum proteins through the mucosa

to the lumen (down the hydrostatic pressure gradient) is dependent on endothelial integrity and on the permeability of the epithelial reticular basement membrane to these molecules and on the epithelial tight junctions (zonula occludens) which serve as selective resistive barriers to the paracellular movement of water, ions and macromolecules.

Basement membrane

The membrane is permeable not only to fluids but also to particulate matter that penetrates the mucociliary blanket (38). It would be more accurate to describe the membrane as semipermeable. The basement membrane is penetrated by capillaries. Fluids can pass through these capillaries directly onto the mucosal surface without passing through the membrane. There is a fundamental difference between the basement membrane in the nose and basement membranes found elsewhere, due to difference in permeability.

Lamina propria (Fig. 2)

The lamina propria contains not only all the glandular, nervous and vascular structures but also organizes the exchanges between the epithelium and the organism.

Glands

They are abundant on the septum and on the floor of the nasal cavities. The lamina propria contain two layers of glands: the superficial layer is situated just under the epithelium, and the deep layer under the vascular layer. The glandular components of the lamina propria consist of serous, mucous or mixed glands, all connected with the epithelium by an excretory canal. The glandular acini are surrounded by myoepithelial cells, favoring the excretion of mucus. The mucus contains 95% of water, 3% of organic elements and 2% of mineral elements. The daily secretion quantity is about 0.3 ml/kg/day (39). The main organic element is mucin, a glycopeptide secreted by the goblet cells. As for tears, the electrolytical composition of the mucus is hyperosmotic compared to plasma. Albumin is the most prominent protein in the nasal mucus. In addition secretory IgA, lactoferrin, lysozyme and kallikrein are synthesized in the cells of the respiratory tract and can be found in the secretion. Interestingly, there seems to exist a circadian modification in the production of sIgA with higher values during the night than during the day (40). Epithelial-derived secretions include glycosaminoglycans, antioxidants and antibacterial substances (41). Other proteins with enzymatic properties have been detected, such as lactic dehydrogenase, several proteolytic enzymes and

protease inhibitors. Amino acids participate in mucus formation with a concentration between 0.4 and 1.3 $\mu\text{M}/\text{ml}$ (42).

Nasal secretions are a mixture of plasma exudate, mucus from goblet cells and seromucous material from the seromucous glands. Expelled epithelial cells and immunocompetent cells can be seen in normal nasal secretions.

Immunocompetent cells in the epithelium

In addition to the structural cells of the surface, there are cells which migrate or "home" into the epithelium. These cells normally include mast cells and intra-epithelial lymphocytes (43). A major function of these lymphocytes is to recognize "nonself" and to bring about, directly or indirectly, the removal of such antigenic particles from the body. They may be either T or B cells. T-cell surface markers include CD3, CD4 (helper), and particularly CD8 (suppressor/cytotoxic) (44). Intra-epithelial lymphocytes recognize antigens as such only when they are expressed on the surface of antigen-presenting cells (APC) bound to the surface molecules encoded by genes of the major histocompatibility complex (MHC) (44). Mast cells form up to 2% of surface epithelial cells and may share some, but not all, of the morphologic and functional characteristics of the subepithelial mast cells and release a variety of preformed and granule-derived mediators of inflammation, some of which affect epithelial tight-junction permeability (45).

Immunocompetent cells in the lamina propria

The depth of the lymphoid layer varies from one region to another; it is particularly important in the middle turbinate. Lymphocytes and plasma cells are the most represented. Histiocytes and macrophages can also be seen. Finally, neutrophils are not frequently found in normal conditions. B-cells of relatively immature memory clones with a potential for J-chain expression are stimulated initially in mucosa-associated lymphoid tissue (MALT) and migrate thereafter through lymph and blood to glandular sites, where they are subjected to terminal differentiation and become Ig-producing lymphocytes (46).

Importance of the secretory and immune functions

Mucus and secretions form a fragmented or continuous mucinous sheet, which acts as a nonspecific barrier to entrap antigenic and potentially noxious particles before they can enter the lower airways. In addition, peroxidases and interferon may act nonspecifically to maintain the sterility of the lower respiratory tract (47). Deficiency in local sIgA production or other local enzymatic factors can

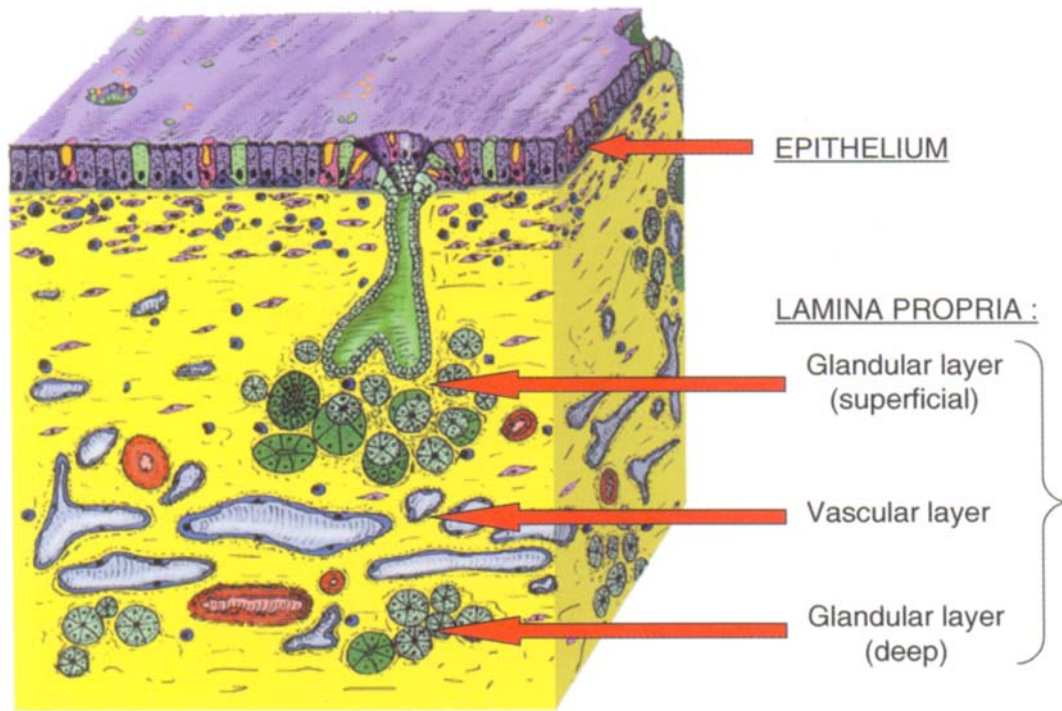


Figure 2. Histology of the nasal mucosa: the lamina propria.

influence directly the efficacy of immunological protection of the mucus. Finally, the viscosity of the mucus has an important impact on mucociliary clearance. If the mucus is too fluid, transport against gravity is impossible. If the mucus is too viscid, as in cystic fibrosis, its evacuation becomes difficult and infections can occur more easily in the mucus plugs.

Vascularization and innervation

>Burnham found, in 1935, that the arterial supply to the inferior and the middle turbinate comes from the sphenopalatine artery (48). Anteriorly, the blood supply comes from the anterior ethmoidal artery. The veins accompany the arteries.

The vasculature of the nose consists of at least four groups of blood vessels: precapillary resistance vessels, capillaries, veins and venous erectile tissue, and arteriovenous anastomoses (49, 50).

The subepithelial and periglandular capillaries are served by a network of arterioles. The various veins drain into large venous sinusoids which make up the venous erectile tissue of the mucosa (51). They form the major component of the mucosa on a volume basis. They are particularly well developed at the anterior part of the inferior turbinate and

on the nasal septum. Its filling determines the state of congestion and thus nasal resistance to airflow.

Blood flow through the nasal blood vessels is controlled by autonomic innervation of the nasal mucosa (52). Nasal blood vessels receive dense sympathetic innervation and stimulation of the sympathetic nerves to the nose causes reduction in nasal blood flow and a pronounced decongestion of nasal venous erectile tissue (53, 54). There is a continuous resting sympathetic tone to the nasal blood vessels and interruption of this activity by nerve section or local anesthesia of the stellate ganglion causes swelling of the venous erectile tissue and nasal congestion (55, 56). This sympathetic tonus to the nasal venous erectile tissue is normally asymmetrical and exhibits cyclic reciprocal activity, which causes a regular nasal cycle of changes in resistance to airflow (57–59).

The parasympathetic fibers to the nose relay in the sphenopalatine ganglion before distribution to the nasal glands and blood vessels. Both acetylcholine and vasoactive intestinal polypeptide (VIP) are involved as neurotransmitters with acetylcholine initiating mainly secretion and VIP mainly vasodilation (60).

Trigeminal sensory innervation of the nose is also involved in the initiation of vasodilatation by means of an axon reflex. Antidromic stimulation of this pathway has been shown to cause vasodilation and substance P has

been implicated as being released from sensory nerve endings (61).

Importance of these structures

The metabolic demands on the nasal mucosa are immense and a variable and adequate local control of the blood flow is crucial (62, 63). In vasomotor and allergic rhinitis, the major pathophysiological changes are congestion of the veins and increase of the permeability of the capillaries. Edema is less important in the swelling process than vasodilation.

Physiology

Even if anatomical and histological structures can have specific functions by themselves they can, when they cooperate, improve the quality of the defense systems for the lower airways. They control the airway size, filtration, air conditioning and smell.

Respiratory function of the nose

Phylogenetically and embryologically, the nose is an essential respiratory organ. Nasal patency can be measured by different exploration methods, which can provide us with much information about nasal physiology (64, 65) (Fig. 3).

Airflow (Fig. 4) (2)

Inspired air penetrates into the nostrils with a 60° angulation and splits into different flows following the different meatus and the space under the turbinates. Following Masing (9), only turbulent airflow enters the ostia. The speed at the entrance of the nasal cavity is between 2 and 3 m/s. At the posterior part of the vestibule – the internal nasal valve – airflow becomes more horizontal. Because this is the narrowest part of the nose, the speed is high and reaches 12–18 m/s. In the region of the turbinates, the speed diminishes to 2–3 m/s (66) (Fig. 5). The total airflow through the nose, normally around $382 \pm 50 \text{ cm}^3/\text{s}$ in adults, increases with age during childhood (67–69). During sniffing, the airflow is deviated in the direction of the superior turbinate and the olfactory epithelium.

Resistance

Nasal airway resistance accounts for 40% of total airway resistance. Different parameters influence nasal resistance: the direction of the nostrils and nares, the shape and size of the nasal cavities, in which the turbinates play an important role, and flow velocity. The most sensitive parameter to

change is the size of the nasal cavities. The size of the turbinate mucosa can be modified by several factors: exercise, emotions, vasomotor response to hormones, environment, pharmacotherapy, etc. (70).

Nasal cycle (Fig. 4) (1)

Another factor affecting the turgescence of the turbinates is the nasal cycle. The description of cyclic changes was described for the first time by Kayser in 1895. He used the term “nasal cycle” for the congestion and decongestion cycle of the cavernous tissue of the nasal turbinates that occurs over a period of 4 h. It was only in 1967 that Guillermin, using rhinomanometry, found that the total nasal resistance remains constant in spite of the continuous diameter changes in left and right nasal cavities (71). This finding can be seen in about 80% of the population (72). With the passive anterior rhinomanometry technique, it was shown that children aged 3–6 years did not have an alternating nasal cycle (73). It seems to be more active during adolescence and decreases in adulthood (74). The cycle is not influenced by anesthetizing the nose or the larynx, and by mouth breathing, but is absent after laryngectomy (75). The nasal cycle seems to depend upon a central regulator (76), even if it was thought to be maintained by peripheral autonomic centers, and the sphenopalatine and stellate ganglia with interconnections between them both (77).

Filtering function of the nose

Inspired air contains a high concentration of particulate matter, which could be aggressive for the fragile structures and slow clearance of the alveoli. To avoid penetration of particles into the lower airways, they are trapped in the mucus film covering the nasal epithelium and transported to the oropharynx, from where they can be swallowed. Proetz has shown, in 1953, that the major role of an anatomically and functionally normal nose is the filtering function (30). The efficacy of the nose filter also depends on the diameter of the particles. During normal breathing, only a few particles greater than 10 μm can enter the lower respiratory tract after the nasal filtration. Irritants around 1 μm of diameter are normally less trapped in the nasal mucous blanket (10, 78, 79). In some environmental and professional conditions, in the presence of large amounts of airborne particles, nasal respiration is particularly important.

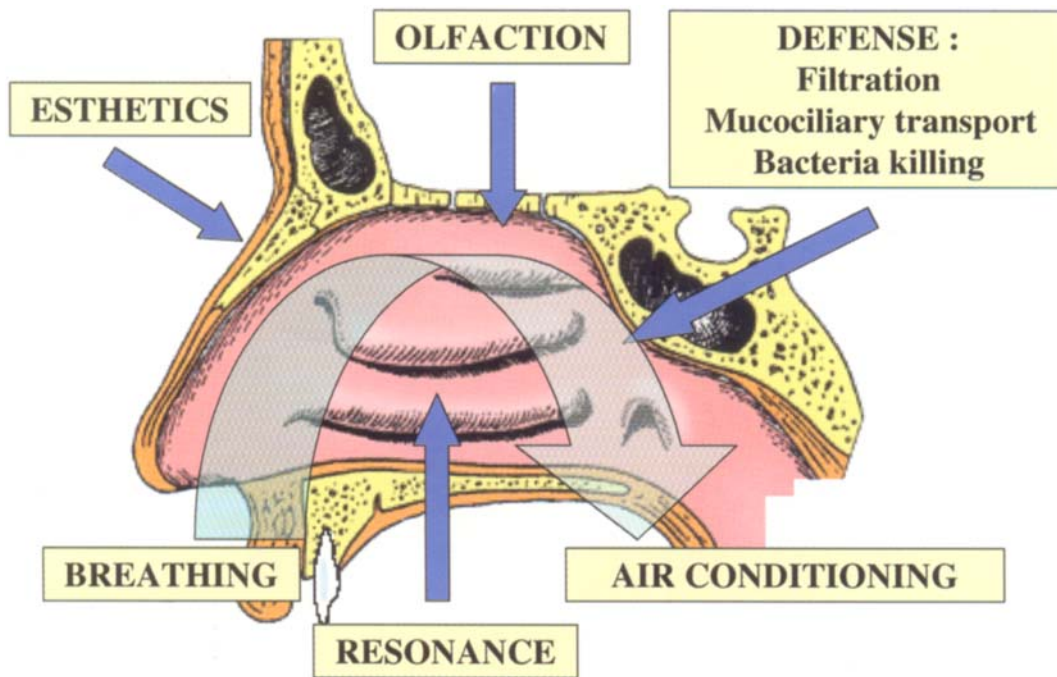


Figure 3. Functions of the nose.

Importance of the filtering function

In the case of oral breathing or tracheostomy, filtration is poorer than in normal nasal breathing. Reduced wall contact by partial or complete blockage of the airflow diminishes contact with the mucus gel-layer and some of the particles can reach the inferior airways more easily. Atrophic rhinitis, septoplasty or radical turbinectomy transforming the nasal vault into a tube-like cavity can have the same effect (80). Pharyngeal, laryngeal or tracheo-bronchial complaints are more often encountered in these situations.

Air conditioning function of the nose

The role of the nose is extremely important in air conditioning (heating and humidification) of inspired air. Ingelstedt, in 1956, analysed the changes in temperature and relative humidity of the air and proved the efficacy of the nose in conditioning inspired air. At room temperature (23°C) and 40% relative humidity, in normal breathing conditions, inhaled air was heated to 30°C and humidified at 98% relative humidity, as could be measured in the pharynx. After 10 min in cold air (−4°C–0°C), the air was conditioned to 31°C and 98% relative humidity. Mouth breathing is less effective.

Importance of the air conditioning function

Unconditioned inspired air alters the respiratory epithelium. Cessation of nasal breathing produces changes in the epithelium. The respiratory tracheal mucosa in the first 5 cm of a patient with a tracheostomy and the surface of a nasal polyp exposed to the impact of inspiratory air are changed into squamous epithelium (81, 82). Crusting and infections are two important complications of a deficiency in air conditioning. Increasing ventilation required in exercise or extreme atmospherical conditions can induce deeper penetration of particles and incomplete air conditioning. Essential in assuring this delicate function of air conditioning are the quantity of the seromucous glands, of goblet cells, the beating quality of the cilia and the microvilli, the ability to change the nasal internal diameter, the efficacy of the vascular network in the lamina propria, the contribution of watery secretion, and the surface contact between inspired air and mucosa.

Olfaction

Olfactory placodes are apparent at the fourth week of embryonic development. The olfactory epithelium covers the superior turbinate and the adjacent septum. Its aspect is yellowish because of the phospholipid pigments that it contains. The pseudostratified epithelium contains olfac-

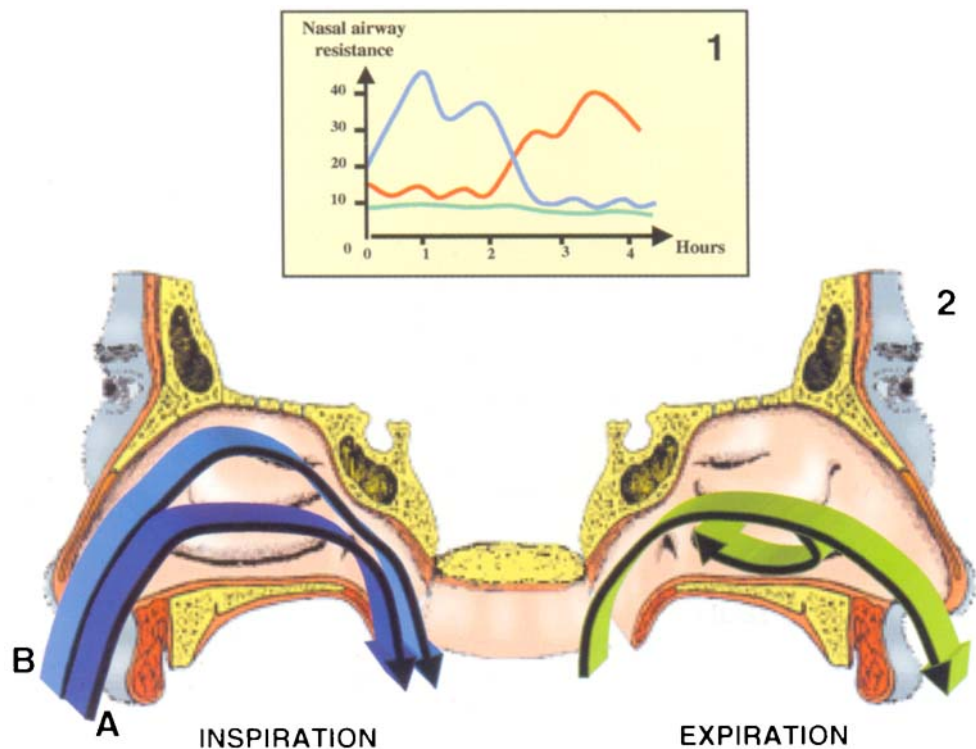


Figure 4. Respiratory function of the nose. 1, Nasal airways resistance; 2, inspiratory and expiratory airflow: A) slower flow, normal breathing, B) rapid flow, sniffing.

tory cells, supporting cells, basal cells and Bowman’s glands. The olfactory receptor cells are bipolar neurons acting as peripheral receptors and first-order ganglia. Connections of the olfactory system go to the limbic system, the reticular

formation of the brain stem for odor-alerting reponse, to the hippocampus, thalamus and hypothalamus and to the frontal lobe.

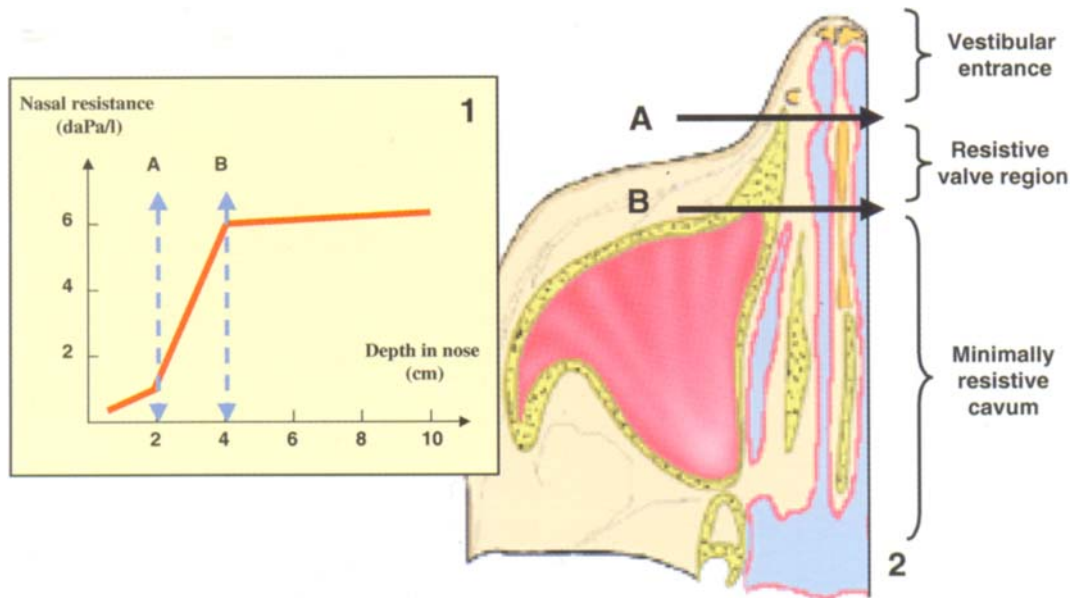


Figure 5. Respiratory function of the nose. 1. Resistive areas in the nasal cavities; 2, coronal section of the nasal and paranasal cavities: A) nasal valve, B) head of the inferior turbinate.

Importance of the olfactory function

The numerous interconnections between the primary olfactory centers and other central structures underline the implications of smell in several physiologic functions, such as reproduction, feeding and visceral reactions. Smell can also protect the lower airways in recognizing several irritants and inducing conscious or nonconscious responses of defense [83]. Anosmia is a frequent complaint in ENT practice, but a distinction must be made between “conductive” anosmia, where an anatomic obstruction keeps the inspired air from reaching the olfactory epithelium, and “perceptive” anosmia due to a dysfunction in the reception or the transmission of the olfactory information. The most common cause of conductive anosmia is the common cold, followed by nasal polyposis. Long-lasting or permanent anosmia can be seen in viral infections, intoxication with airborne agents, e.g. fumes, trauma of the skull base or after ethmoid sinus surgery.

Conclusions

Anatomical and histological characteristics of the nasal and paranasal cavities can have specific implications in the defense system which protects the lower airways. When these structures act together, not only is the efficacy of each increased but new defense systems become apparent. The interaction between all these processes is particularly important, and any modification in the system can induce a cascade of disorders and affect directly the protective properties of the nose. It is possible to live without a nose but the clinical implications are very obvious in patients with poor nasal function. No other organ can assume all the complex functions so well and protect the lower respiratory tract so effectively.

References

- Paludetti G, Alinadori G, Scarano E, Deli R, Laneri de Bemart A, Maurizi M. Nasal obstruction and skull base development: experimental study in the rat. *Rhinology* 1995;33:171–173.
- Ohki M, Naito K, Cole P. Dimensions and resistances of the human nose: racial differences. *Laryngoscope* 1991;101:276–278.
- Becker DG, Weinberger MS, Greene BA, Tardy ME. Clinical study of alar anatomy and surgery of the alar base. *Arch Otolaryngol Head Neck Surg* 1997;123:789–795.
- Cole P, Haight JSJ, Love L, Oprysk D. Dynamic components of nasal resistance. *Am Rev Respir Dis* 1985;132:1229–1232.
- Mann DG, Sasaki CT, Suzuki M, Fukuda H, Hernandez JR. Dilator naris muscle. *Ann Otol Rhinol Laryngol* 1977;86:362–370.
- Bruintjes TD, Van Olphen AF, Hillen B, Weijs WA. Electromyography of the human nasal muscles. *Eur Arch Otorhinolaryngol* 1996;253:464–469.
- Griffin JW, Hunter G, Ferguson D, Sillers MJ. Physiologic effects of an external nasal dilator. *Laryngoscope* 1997;107:1235–1238.
- Constantinides MS, Adamson PA, Cole P. The long-term effects of open cosmetic septorhinoplasty on nasal air flow. *Arch Otolaryngol Head Neck Surg* 1996;122:41–45.
- Masing H. Pathophysiology of the nasal air flow. *Int Rhinol* 1967;5:63–67.
- Proctor DF, Andersen I, Lundquist G. Clearance of inhaled particles from the human nose. *Arch Intern Med* 1973;131:132–136.
- Morgan NJ, Mac Gregor FB, Birchall MA, Lund VJ, Sittampalam Y. Racial differences in nasal fossa dimensions determined by acoustic rhinometry. *Rhinology* 1995;33:224–228.
- Drake AF, Davis JU, Warren DW. Nasal airway size in cleft and noncleft children. *Laryngoscope* 1993;103:915–917.
- Van Loosen J, Van Zanten GA, Howard CVI, Verwoed-Verhoef HL, Van Velzen D, Verwoerd CGA. Growth characteristics of the human nasal septum. *Rhinology* 1996;34:78–82.
- Bingham B, Wang RG, Hawke M, Kwok P. The embryonic development of the lateral nasal wall from 8 to 24 weeks. *Laryngoscope* 1991;101:992–997.
- Wang RG, Jiang SC. The embryonic development of the human ethmoid labyrinth from 8–40 weeks. *Acta Otolaryngol (Stockh)* 1997;117:118–122.
- Wolf G, Anderhuber W, Kuhn F. Development of the paranasal sinuses in children. *Ann Otol Rhinol Laryngol* 1993;102:705–711.
- Laurenzo JF, Canady JW, Zimmerman MB, Smith RJH. Craniofacial growth in rabbits. *Arch Otolaryngol Head Neck Surg* 1995;121:556–561.
- Mair EA, Bolger WE, Breisch EA. Sinus and facial growth after pediatric endoscopic sinus surgery. *Arch Otolaryngol Head Neck Surg* 1995;121:547–552.
- Chen MY, Metson R. Effects of sinus surgery on speech. *Arch Otolaryngol Head Neck Surg* 1997;123:845–852.
- Friedman M, Caldarelli DD, Venkatesan TK, Pandit R, Lee Y. Endoscopic sinus surgery with partial middle turbinate resection, effects on olfaction. *Laryngoscope* 1996;106:977–981.
- Breeze RG, Wheeldon EB. The cells of the pulmonary airways. *Am Rev Respir Dis* 1977;116:705–777.
- Rasp G, Hochstrasser K. Tryptase in nasal fluid is a useful marker of allergic rhinitis. *Eur J Allergy Clin Immunol* 1993;48:72–74.
- Swift DL, Proctor DF. Access of air to the respiratory tract. In: Brain JD, Proctor DF, Reid LM, editors. *Respiratory defense mechanisms, Part I*. New York: Marcel Dekker, 1977:488–498.
- Wake M, Takeno S, Hawke M. The early development of sino-nasal mucosa. *Laryngoscope* 1994;104:850–855.
- Helfferrich F, Viragh S. Histological investigations of the nasal mucosa in human fetuses. *Eur Arch Otorhinolaryngol* 1997;254(Suppl. 1):S39–S42.
- Mygind N, Bretlau P. Scanning electron microscopic studies of the human nasal mucosa in normal persons and in patients with perennial rhinitis. 1: Cilia and microvilli. *Acta Allerg (Kbh)* 1974;28:19.
- Breeze RG, Wheeldon EB. The cells of the pulmonary airways. *Am Rev Respir Dis* 1977;116:705–777.

28. Satir P. How cilia move. *Sci Am* 1974;**231**:44–52.
29. Dustin P. *Microtubules*. New York: Springer-Verlag, 1978:452–468.
30. Proetz AW. *Applied physiology of the nose*. St Louis: Annals Publishing Company, 1953.
31. Andersen I, Lundquist GR, Proctor DF. Human nasal function in a controlled climate. *Arch Environm Hlth* 1971;**23**:408–411.
32. Proetz AW. Humidity, a problem in air conditioning. *Ann Otol* 1956;**65**:374–384.
33. Simon H, Drettner B, Jung B. Messung des Schleimhauttransporters in menschlichen Nase mit ⁵¹Cr markierten Harzkilgelschen. *Acta Otolaryngol (Stockh)* 1976;**83**:378–385.
34. Cervin A, Bende M, Lindberg S, Mercke U, Olsson P. Relations between blood flow and mucociliary activity in the rabbit maxillary sinus. *Acta Otolaryngol (Stockh)* 1988;**105**:350–356.
35. Andersen I, Lundquist GR, Jensen PL, Proctor DF. Human response to controlled levels of sulfur dioxide. *Arch Environm Hlth* 1974;**28**:31–38.
36. Toskala E, Westrin KM, Stierna P, Rautiainen M. Ciliary ultrastructure in experimental sinusitis. *Acta Otolaryngol (Stockh)* 1997;**Suppl. 529**:137–139.
37. Welsh MJ. Electrolyte transport by airway epithelia. *Physiol Rev* 1987;**67**:1143–1184.
38. Munzel M. The permeability of intercellular spaces of the nasal mucosa. *J Laryngol Rhinol Otol* 1974;**51**:794.
39. Melon J. Contribution a l'étude de l'activité sécrétoire de la muqueuse nasale. *Acta Otolaryngol (Belg)* 1968;**22**:5–244.
40. Passali D, Bellussi L. Circadian changes in the secretory activity of nasal mucosa. *Acta Otolaryngol (Stockh)* 1988;**106**:281–285.
41. Jeffery PK. Morphology of airway surface epithelial cells and glands. *Am Rev Respir Dis* 1983;**128**:S14–S20.
42. Hilding AC, Cowles EJ, Hilding-Stuart J. Amino acid composition of human nasal mucus. *Ann Otol* 1973;**82**:75–79.
43. Pawankar R, Okuda M. A comparative study of the characteristics of intraepithelial and lamina propria lymphocytes of the human nasal mucosa. *Eur J Allergy Clin Immunol* 1993;**48**:99–105.
44. Strober W, James SP. Mucosal immune system. In: Stites DP, et al, editors. *Basic and clinical immunology*. Norwalk: Appleton and Lange, 1994:541–552.
45. Holgate ST, Robinson C, Church MK. Mediators of immediate hypersensitivity. In: Middleton E, editor. *Allergy, principles and practice*. St Louis: Mosby, 1988:135–163.
46. Brandtzaeg P. Immunobarrriers of the mucosa of the upper respiratory and digestive pathways. *Acta Otolaryngol (Stockh)* 1988;**105**:172–180.
47. Jeffery PK. Morphology of airway surface epithelial cells and glands. *Am Rev Respir Dis* 1983;**128**:S14–S20.
48. Bumham H. An anatomical investigation of blood vessels of the lateral nasal wall. *J Laryngol* 1935;**50**:569–594.
49. Widdicombe J. Microvascular anatomy of the nose. *Eur Allergy Clin Immunol* 1997;**52**(Suppl. 40):7–11.
50. Cauna N. Blood and nerve supply of the nasal lining. In: Proctor DF, Andersen I, editors. *The nose, upper airway physiology and the atmospheric environment*. Amsterdam: Elsevier, 1982:45–66.
51. Temesrekasi D. Mikroskopischer Bau und Funktion des Schwellgewebes der Nasenmuschel des Menschen. *Z Mikrosk-Anat Forsch* 1969;**80**:219–229.
52. Lund VJ. Nasal physiology, neurochemical receptors, nasal cycle and ciliary action. *Allergy Asthma Proc* 1996;**17**:179–184.
53. Eccles R, Wilson H. The autonomic innervation of the blood vessels of the cat. *J Physiol* 1974;**238**:54–60.
54. Malm L. Stimulation of sympathetic nerve fibres to the nose in cats. *Acta Otolaryngol (Stockh)* 1973;**75**:519–526.
55. Rooker DW, Jackson RT. The effects of certain drugs, cervical sympathetic stimulation and section on nasal patency. *Ann Otol Rhinol Laryngol* 1969;**78**:403–414.
56. Stocksted P, Thomsen KA. Changes in the nasal cycle under stellate ganglion blockade. *Acta Otolaryngol (Stockh)* 1953;**Suppl. 109**:176–181.
57. Eccles R. The central rhythm of the nasal cycle. *Acta Otolaryngol (Stockh)* 1978;**86**:464–485.
58. Hasegawa M, Kern EB. The human nasal cycle. *Mayo Clin Proc* 1977;**52**:28–34.
59. Riederer A, Fischer A, Knipping S, Unger J, Lange W, Kastanauer E. Basic innervation pattern and distribution of classic autonomic neurotransmitters in human nasal mucosal vasculature. *Laryngoscope* 1996;**106**:286–291.
60. Malin L, Sundler F, Uddman R. Effects of vasoactive intestinal polypeptide on resistance and capacitance vessels in the nasal mucosa. *Acta Otolaryngol (Stockh)* 1980;**90**:304–308.
61. Lundblad L, Ånggård A, Lundberg JM. Vasodilatation in the cat nasal mucosa induced by antidromic trigeminal nerve stimulation. *Acta Physiol Scand* 1982;**115**:517–519.
62. Hogg JC. Mediator release from the airway mucosa. *Eur J Respir Dis* 1983;**64**(Suppl. 128):3–6.
63. Konno A, Togawa K, Fujiwara T. The mechanisms involved in onset of allergic manifestations in the nose. *Eur J Respir Dis* 1983;**64**(Suppl. 128):155–166.
64. Malm L. Measurement of nasal patency. *Eur J Allergy Clin Immunol* 1997;**52**(Suppl. 40):19–23.
65. Cole P, Roithmann R, Roth Y, Chapnik JS. Measurement of airway patency. *Ann Otol Rhinol Laryngol* 1997;**106**(Suppl. 171):7–23.
66. Proctor DF. The nose, upper airway physiology and atmospheric environment. In: Proctor DF, Andersen I, editors. Amsterdam: Elsevier Biomedical Press, 1982.
67. Mennella JA, Beauchamp GK. Developmental changes in nasal airflow patterns. *Acta Otolaryngol (Stockh)* 1992;**112**:1025–1031.
68. Edelstein DR. Aging of the normal nose in adults. *Laryngoscope* 1996;**106**(Suppl. 81):1–23.
69. Laine-Alava M, Minkkinen UK. Variation of nasal respiratory pattern with age during growth and development. *Laryngoscope* 1997;**107**:386–390.
70. Ohki M, Hasegawa M, Kurita N, Watanabe I. Effects of exercise on nasal resistance and nasal blood flow. *Acta Otolaryngol (Stockh)* 1987;**104**:328–333.
71. Guillermin R, Badre R, Riv R, Le Den R, Fallot P. La rhinorheographie. *Rev Laryngol (Bordeaux)* 1967;**3**(Suppl.):45–60.
72. Stocksted P, Thomsen KA. Changes in the nasal cycle under stellate ganglion block. *Acta Otolaryngol (Stockh)* 1953;**109**(Suppl.):176–181.
73. van Cauwenberge P, Deleaye L. Nasal cycle in children. *Arch Otolaryngol* 1984;**110**:108–111.
74. Mirza N, Kroger H, Doty RL. Influence of age on the nasal cycle. *Laryngoscope* 1997;**107**:62–66.
75. Havas TE, Cole P, Gullane PJ, Kassel R, Kamino D. The nasal cycle after laryngectomy. *Acta Otolaryngol (Stockh)* 1987;**103**:111–116.
76. Eccles R. The central rhythm of the nasal cycle. *Acta Otolaryngol (Stockh)* 1978;**86**:464–468.
77. Connell JT. Reciprocal nasal congestion-decongestion reflex. *Trans Am Acad Ophthalmol Otolaryngol* 1968;**72**:18–25.
78. Cole P, Savard P, Miljeteig H, Haight JSJ. Resistance to respiratory airflow of the extrapulmonary airways. *Laryngoscope* 1993;**103**:447–450.
79. Muir DCF. *Clinical aspects of inhaled particles*. London: Heinemann, 1972.

80. Schwab JA, Zenkel M. Filtration of particulates in the human nose. *Laryngoscope* 1998;**108**:120–124.
81. Shah JT, Kamik PP, Chitale AR, Nadkarni S. Partial or total closure of the nostrils in atrophic rhinitis. *Arch Otolaryngol* 1974;**100**:196–200.
82. Mygind N, Bretlau P, Sørensen H. Scanning electron microscopic studies of nasal polyps. *Acta Otolaryngol (Stockh)* 1974;**78**:436–441.
83. Warren DW, Odont D, Waker JC, Drake AF, Lutz R. Effects of odorants and irritants on respiratory behavior. *Laryngoscope* 1994;**104**:623–626.