Rhinology, XVII, 3-11, 1979

The role of the nose in the functional unit of the respiratory system

Börje Drettner, Stockholm, Sweden

SUMMARY

The nasal secretion produced by the goblet cells and glands in the normal mucosa, consists of a serous periciliary layer on which there are discrete droplets of mucus. The exchange of humidity to the respiratory air is facilitated by a principally turbulent nasal airflow with a laminar boundary layer. Normally, the capacity of the glands and goblet cells to humidify the air is so great that an unimpaired nasal mucociliary activity is possible even when the surrounding humidity is very low. The nasal secretion contains inhibitors which decrease during an acute upper respiratory infection.

Previous investigations on naso-pulmonary reflexes have recently been under discussion. These reflexes are probably principally changes of the respitarory breathing pattern and not changes in the intrinsic behaviour of the pulmonary airways. Several reports have been published showing that experimental nasal obstruction causes changes in the arterial blood gases. There are several reports too concerning serious cardiorespiratory complications in children due to naso-pharyngeal obstruction. Maximal voluntary nasal ventilation is closer to maximal voluntary oral ventilation in subjects with pulmonary insufficiency than in normal subjects.

Kartageners syndrome is due to a defect of the dynein arms in the cilia and is usually combined with sterility in male subjects due to the same defect in the spermatozoa.

When the respiratory function of a patient is investigated this is almost always performed during breathing through the mouth, which means that one important part of the normal respiratory system – the nose – is bypassed. When rhinologists investigate the nose they correspondingly concentrate their examination to the nose. It seems therefore required that the total respiratory passages

---

Paper presented at the 7th Congress of the European Rhinologic Society. Davos (Switzerland), September, 1978.

are looked upon as the unit they in reality are and not separated into too many specific parts.

THE NASAL MUCOSA

In the nose, the epithelium is ciliated, columnar and with goblet cells except for the anterior part of the nose, including the anterior part of the inferior and middle turbinate, where the epithelium is stratified, squamous and keratinized. The mucous layer has long been considered to consist of a continuous mucous blanket in which the surface is more richly mucous and the deeper layer more fluid (Lucas and Douglas, 1934).

Modern studies by scanning electron microscopy have shown, however, that there are discrete droplets of mucus above the periciliary layer which is serous (van As and Webster, 1972). The more viscid material is secreted by the goblet cells and passes through the periciliary fluid to lie in droplets on the surface.

The vascular system in the nasal mucosa has a special structure. There are large venous sinusoids situated between the capillaries and venous plexus. A similar system with large venous sinusoids has recently been described in the trachea (Nordin and Lindholm, 1977). In addition, the mucosal blood flow is pronounced in the mucosa of the nose, paranasal sinuses and trachea.

THE ROLE OF THE NOSE FOR THE LOWER RESPIRATORY SYSTEM

The nose as aerodynamic pathway

The normal airflow in the nose is principally turbulent which implies an irregular air stream with eddies. In a pure turbulent airflow, the driving pressure is proportional to the square of the airflow. In a series of experiments, it was shown that the exponent characterizing the nasal airflow as a mean is 1.7 which implies that there is a predominantly but not completely turbulent airflow (Drettner, 1961). This exponent was found to be principally independent of the nasal patency, but with lower airflows there is a change to a more laminar airflow (Masing, 1967) and a complete laminar airflow occurs at inspiration at airflows below 5 l. per minute (Fischer, 1969).

A boundary layer with low velocity and laminar airflow is present close to the nasal walls (Ingelstedt and Toremalm, 1960). The turbulence reaches according to Fischer (1969), a maximum immediately central to this boundary layer. The exchange of water vapour and heat energy is thus facilitated.

Airconditioning

Airconditioning by the nose comprises two functions: to warm and to humidify the respiratory air in its passage through the nose. Ingelstedt (1956) by recordings of the temperature and humidity of the subglottic air showed that the subglottic air is warmer and more humid during nasal breathing than during oral breathing, and that nasal breathing in a cold climate gives a better conditioning of the subglottic air than oral breathing at room temperature. The anatomical basis for this superiority is the narrow nasal cavities, the vascular system with a high blood flow, venous sinusoids and the rich distribution of glands and goblet cells and, finally, the turbulent airflow.

The nasal cavities have not only an excellent ability to warm and humidify the inspiratory air but also are able to regain heat energy and water vapour during expiration. The anterior part of the nasal cavity has a somewhat cooler surface than the posterior part, and a condensation and regain of heat energy occurs during expiration (Ingelstedt, 1956). This exchange of water vapour and heat energy is more effective in a cold climate than at ordinary room temperature.

Mucociliary transport

Mucociliary transport in the nose is dependent on two separate functions, the ciliary activity and the production of nasal secretion. The normal nasal secretion is generally considered to be produced by glands and goblet cells and no transudation appears to occur except in pathologic conditions such as allergic rhinitis (Ingelstedt and Ivstam, 1951). Modern studies of the mucociliary activity *in vitro* have shown that the optimal temperature for mucociliary activity lies close to body temperature. The activity decreases with decreasing relative air humidity. The time during which mucociliary activity persists *in vitro* is shorter when the relative humidity falls below 40–50% than with higher relative humidity (Mercke, 1975).

In contrast to observations *in vitro*, it has been found that even extreme variations of the relative humidity do not exert any influence upon the nasal mucociliary transport speed (Proctor et al., 1973; Andersen et al., 1975). The ability of the glands and goblet cells to humidify the air and the nose is so great that an intact mucociliary activity is possible even when the surrounding relative humidity is as low as 10% and the subjects are exposed and observed during 78 hours (Andersen et al., 1974).

An effect of an increased airstream and thereby increased demands on the mucociliary transport becomes obvious when an individual is forced to breathe through one nostril only. The transport speed is increased in the blocked nasal cavity while it is slightly reduced in that with increased airstream. It is also worthy of notice that decongestants in the nose decrease the mucociliary transport but whether this is a direct or a secondary effect of vasoconstriction, or wider nasal cavities, is not known. An inhibition of the transport speed occurs too during an upper inspiratory infection, and one week before the onset of such an infection.

Filtration

The nose has a highly effective filtrating capacity, which protects the lower respiratory system from noxious particles, gases and micro-organisms. Particles with a diameter of 8 μm or more remain almost entirely in the nose while particles of 2–3 μm in diameter are retained to about 50% and those of less than 1 μm pass through the nose to the lower airways almost inevitably (Hilding, 1976). But the very small particles of 0.5 μm in diameter leave the respiratory tract to a great extent on expiration, and are therefore not retained in the lower airways in contrast to the somewhat larger particles.

Immunology and enzymology of the nasal secretion

There are several protective mechanisms in nasal secretion which protect the host against different kinds of infection. Lysozyme is an enzyme with a bacteriolytic property and the ability to break down bacterial cell walls. Schorn and Hochstrasser (1975) have shown that the normal nasal secretion contains several enzymes belonging to the intermediary metabolism. These show characteristic changes during various nasal conditions; they are increased during a virus rhinitis but not during allergic rhinitis. The nasal secretion also contains specific protease inhibitors which act on, among others, trypsin and chymotrypsin. These inhibitors decrease during an acute upper respiratory infection and have their lowest value when the clinical symptoms are most pronounced and they may sometimes even disappear completely.

The immunoglobulins are also important for protection. IgG and IgA, which are produced by plasmacells, comprise most antibodies against vira and bacteria. IgA is the quantitatively dominating immunoglobulin in secretion in contrast to serum. In secretion, IgA is principally a dimer bound by so called secretory piece, while IgA in serum is a monomer. IgA and IgE in nasal secretion are mainly produced locally while IgG is not locally produced. The connection between IgE and allergy is well known. It is worth mentioning that hyposensibilization produces antibodies which belong to the IgG-group.

THE ROLE OF THE PARANASAL SINUSES FOR THE LOWER RESPIRATORY SYSTEM

So far, the function of the paranasal sinuses is unknown even though hypotheses have been published in order to try to explain the existence of the paranasal sinuses. One of these hypotheses discusses the paranasal sinuses as a reservoir of warm and humid air utilized during inspiration. It has been shown that the contribution in humidity from the paranasal sinuses is only about 1.5% of the total net supply of water vapour from the upper respiratory airways and that this is too small for paranasal sinus function in the conditioning of the respiratory air (Aust & Drettner, 1974).

NASO-PULMONARY REFLEXES

Much has been written concerning reflexes from the nose affecting the bronchii.

Many of these reports have shown contradictory results and there still remains a great deal of confusion on what actually is true in man concerning naso-pulmonary reflexes.

Sercer (1952), showed that mechanical, chemical and thermal stimulation of the nasal mucosa induced constriction of the bronchii but sometimes there was a dilatation especially with strong nasal stimulation. This naso-bronchial reflex is principally homolateral.

Ogura et al., (1966, 1970, 1971) and Ohnishi et al. (1969, 1971) in a series of papers established a relation between nasal obstruction and pulmonary function in human subjects. The pulmonary resistance increases with an increasing nasal obstruction and the functional compliance decreases with increasing grades of nasal obstruction. The functional compliance which expresses compliance divided by the functional residual capacity was found to increase with increased nasal obstruction. The ordinary compliance showed no statistical correlation with nasal obstruction. In addition it was found that changes in resistance, functional compliance and functional residual capacity were reversible after successful nasal operations. Experiments in dog which surgically obtained a partial or total obstruction of the nasal cavities also showed an increasing pulmonary airflow resistance after nasal obstruction. It is interesting to note that in a series of cats Nadel and Widdicombe (1962) and Tomori and Widdicombe (1969) found broncho-dilatation after stimulation of the nasal mucosa.

In a recently published report from the Mayo Clinic (Whicker et al., 1978) all previous investigations were questioned because the pulmonary breathing patterns and volumes were not well enough controlled in any one of these to allow an accurate conclusion to be drawn regarding changes in pulmonary resistance and compliance. Whicker et al. (1978) found an increase in pulmonary airflow resistance but no change in compliance by nasal stimulation with ice-water in anaesthetized non-paralyzed dogs. This response to nasal stimulation was abolished by interrupting either the trigeminal or the vagus nerves. In another group of anaesthetized paralyzed dogs with fixed flow rates, no changes in resistance or compliance were found after nasal stimulation. This paralyzing had no direct effect on broncho-constriction or broncho-dilatation since this is mediated by smooth muscles. The conclusion reached was that nasal stimulation produced changes in breathing pattern leading to changes in resistance but without an actual change in the intrinsic behaviour of the pulmonary airways. It was found that the respiratory pattern changes in non-paralyzed dogs after nasal stimulation and those dogs who showed a drastic change in resistance also tended to show increases in respiratory airflow. Due to the nonlinearity of the pressure-flow relationship, measurements taken at a resting respiratory flow, give a lower resistance than measurements taken at higher flow. (Figure 1)

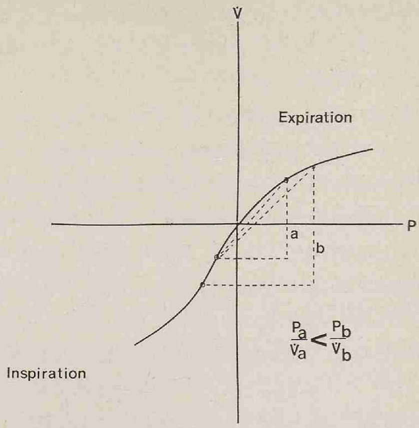

Figure 1. Pressure – flow curve of the lung during inspiration and expiration. When resistance is expressed as $P/\dot{v}$ the value at a will be smaller than the value at b. Modified after Whicker, Kern & Hyatt. Annals of Otology, 87, 1978.

Similarily, changes in volume can alter compliance due to the nonlinearity of the pressure-flow characteristics of the lung.

NASO-PULMONARY RELATIONSHIP

Effects of nasal obstruction on the lower respiratory system and blood gases

Nasal respiration is definitely more physiological than oral respiration and occurs in normal subjects, at least during rest. According to Uddströmer (1940), patients with septum deviation have a lower degree of nasal respiration than normal subjects. Oral breathing occurs in 36% of cases with septum deviation. Proctor et al. (1978) found that in some subjects inspired air can pass through the nasal airways without being sufficiently airconditioned, and they supposed that principally the smaller airways may be affected under these conditions, due to the fact that the passage of the air through these smaller airways takes a long time and their immense surface provides an optimal opportunity for the completion of the process left unfinished in the nose.

Several reports have also been published concerning changes in arterial blood gases after nasal obstruction, principally after nasal tamponade (Lüscher, 1930; Slocum et al., 1976; Cassisi et al., 1971; Cavo et al., 1975; Ogura et al., 1973) which all have found that the arterial oxygen tension was lower with packing than without.

Naso-pharyngeal obstruction in children causing cardiorespiratory complications

During the last decade, several reports have been published describing serious cardiovascular complications caused by partial obstruction of the upper respiratory tract, principally nasopharyngeal obstruction (Menashe et al., 1965; Noonan, 1965; Luke et al., 1966; Levy et al., 1967; Massumi et al., 1969).

Mainly negro children have been reported. They had cardio-respiratory complications ranging from moderate cardiomegaly and right ventricular hypertrophy to severe right cardiac failure and pulmonary oedema. The only cause disclosed after comprehensive investigation was the nasopharyngeal obstruction. Tonsillectomy and adenoidectomy performed as an emergency measure gave complete recovery with decrease in the heart size and normalization of the electrocardiogram. The mechanism was assumed to be hypoxia and hypercapnia causing a pulmonary vasoconstriction and elevated pulmonary arterial pressure which over a period of months resulted in cardiac hypertrophy and right cardiac failure. This syndrome is, however, very rare in comparison with the frequency of nasal obstruction in children.

Effects of pulmonary insufficiency on nasal breathing

Nasal breathing requires more energy than oral breathing. The total work during nasal breathing is more than double the respiratory work during oral breathing in normal subjects (Butler, 1960). Individuals with reduced pulmonary function can use nasal breathing to a higher percentage of their maximum volunatary ventilation than those with normal ventilation (Drettner, 1969). One explanation can be a bronchiolar collapse which can occur, for instance, in emphysema during forced oral breathing. If there is a terminal resistance to the expiratory airflow, as during nasal breathing, the pressure gradient in the bronchii will be less which will thus reduce the risk of bronchiolar collapse.

Several studies have shown that respiratory stress affects the patency of the nasal cavities. Already in 1923 Tatum showed that asphyxia and rebreathing in a bag causes a widening of the nasal cavities, and breathing of air with a high carbon dioxide content induces a widening of the nasal cavities (Tagaki et al., 1969). Physical exertion has an obvious effect on the nasal patency which becomes greater (Aschan et al., 1958). By using various pharmacological blockers, it has been found that this widening of the nasal cavities, during exercise is abolished, for instance, after stellate ganglion block which indicates a sympathetic nerve discharge as an explanation of this phenomenon (Richerson and Seebohm, 1968).

Interaction between the upper and lower respiratory airways

It is well known that allergy sometimes affects the nose, sometimes the bronchii, and sometimes both. Grass and birch which are relatively big pollen particles are deposited in the nose and thus cause nasal allergy while fungal spores are smaller and therefore pass through the nose to the bronchii more readily. It is also established that nasal polyps and asthma often coexist, which does not mean, however, that nasal polyps are necessarily of allergic origin.

Kartageners syndrome consists of chronic sinusitis and bronchiectasia, and sometimes also situs inversus. It has recently been shown that this syndrome is probably caused by an inherent defect of the cilia which causes a chronic infection both of the upper and lower respiratory system (Camner et al., 1975). Furthermore, these patients have an inherent defect of spermatozoa which causes sterility in male subjects due to the fact that the spermatozoa are not able to move. There is an ultrastructural defect of the cilia and the sperm tail which lack the dynein arms required for a normal movement of both cilia and spermatozoa. The "immotile cilia" syndrome is a better name than Kartageners syndrome for the respiratory symptoms which in half of the cases are combined with situs inversus. All the patients have impaired mucociliary transport in the lower airways.

ZUSAMMENFASSUNG

Das Nasensekret, welches von den Becherzellen und Drüsen in der Nasenschleimhaut produziert wird, besteht aus einer serösen, periciliaren Schicht, auf welcher sich kleine Schleimtropfen befinden. Der Feuchtigkeitsaustausch zur Atmungsluft wird erleichtert von dem hauptsächlich turbulenten Luftstrom in der Nase mit einer laminaren Grenzschicht. Normalerweise ist das Vermögen der Drüsen und Becherzellen die Luft anzufeuchten so gross, dass eine intakte, nasal-mukociliare Aktivität auch möglich ist, wenn die umgebende Luftfeuchtigkeit sehr niedrig ist. Das Nasensekret enthält unter anderem Inhibitoren, welche während einer akuten oberen Luftwegsinfektion abnehmen.

Frühere Untersuchungen von nasopulmonären Reflexen sind vor kurzem kritisiert worden. Diese Reflexe sind hauptsächliche Veränderungen in dem respiratorischen Atmungsmuster und nicht Veränderungen in dem inneren Verhalten der Luftwege. Mehrere Berichte sind publiziert worden, welche zeigen, dass experimentelle, nasale Obstruktion Veränderungen in den arteriellen Blutgasen verursachen. Es gibt auch mehrere Berichte über ernsthafte kardiorespiratorische Komplikationen bei Kindern, die auf nasopharyngeale Obstruktion beruhen. Maximale, volontäre, nasale Ventilation liegt näher der maximalen, volontären, oralen Ventilation bei Personen mit Lungeninsuffizienz als bei normalen Individuen.

Kartageners Syndrom beruht auf einen Defekt in den Dynein-armen bei den Cilien und ist meistens kombiniert mit Sterilität bei männlichen Individuen, welche auf den gleichen Defekt in den Spermatozoen beruht.

REFERENCES

References can be obtained from the author on request.

B. Drettner, M.D.
Dept. of O.R.L.
University Hospital
S-14186 Huddinge
Sweden

This investigation was supported by the Swedish Medical Research Council (Project 739).