THE NOSE, CAN YOU TRUST IT ?

David Bromwich
University of Queensland

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Occupational Hygienist, University of Queensland

ABSTRACT

The nose is a largely underestimated organ by occupational hygienists, yet it provides us with a sense of smell, warms, humidifies and filters air as well as producing copious secretions.

Workplace chemicals can directly affect the nose and enter the bloodstream through the nasal mucosa.

This paper discusses theories of the sense of smell, variability of smell thresholds, airflow, filtration, heat exchange and some occupational diseases of the nose. Workplace examples of how the nose can mislead are presented.

1 INTRODUCTION

Little attention is paid to the nose due to a lack of functional replacement for the nose and its minor role as an organ of communication. This contrasts with vision, hearing and teeth where function can often be restored mechanically or electronically. If you can't do much about a reduced function like that of smell, there is little general application of methods to measure it. Testing can be done, but unlike vision and hearing, the tests involve chemical rather than physical stimuli. The science of transducing and amplifying chemical signals with great sensitivity and specificity is still in its infancy,

If your nose is obstructed you simply breathe through your mouth. If your lungs are blocked you die, so research funds tend to go towards lung function. Some research into the function of the nose is sponsored by the food and cosmetics industry where there are obvious commercial gains. Relatively little current research has direct application to occupational hygiene.

Occupational hygiene texts tend to refer to the air modification function of the nose along with lung disease and the nose is quickly dismissed as a simple size selective air cleaner which is slightly better at warming, humidifying and filtering the air than the mouth.

This paper outlines two functions of the nose– air modification and olfaction but largely ignores the immune response, nasal allergies and the details of nasal secretions.

2 AIR MODIFICATION

The nose. may be described in terms of its ability to filter, humidify and warm air on the way to the lungs. Around 85% of adults are nose breathers, but if the mouth is open, 60% of the air still enters through the nose in quiet breathing, decreasing to around 40% with exercise (COLE 1982a). With the tongue depressed and the mouth

open the resistance to flow through the mouth decreases to near zero.

Laryngectomy patients lack the use of their nose and mouth to modify inspired air but over many months their trachea adapts to perform this function (HILDING 1976). Much of this function can be duplicated with 25cm long 20mm tube lined with damp blotter (COLE 1982b).

Laminar air flow may occur in the nose during quiet respiration. Turbulent air flow occurs first at the nasal valve as inspired air flows up the external nose and turns to flow towards the back of the throat. The nasal valve is the narrowest part of the nose and most of the resistance to breathing comes at this point. Air turbulence ensures good mixing of air, particularly of the boundary layer of air next to the nasal mucosa. This allows good exchange of heat and moisture on both inspiration and exhalation as well as absorption of gases and impaction of smaller dust particles with the moist epithelium of the nose. At high flow rates this turbulent regime moves towards the back of the nose and may even reach the seventh division of the bronchi (COLE 1982a).

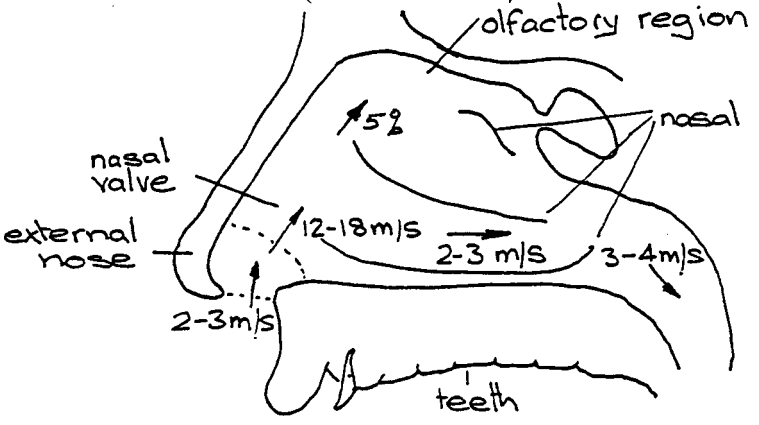

Figure 1 Pattern of airflow through nose (after COLE 1982a)

The visco-elastic properties of the mucus lining the nose may either promote or inhibit the transition from laminar to turbulent flow, but this subject is complex and there appears little published on it.

In its passage through the nose, inspired air first encounters nasal hairs in the vestibule or entrance to the nose. Two groups of hairs with diameters of 50 and 150 microns project to cover the airway and remove around 20% of airborne particles by impaction (HILDING 1976). There could be no coincidence that the diameter of this part of the nose is such as to facilitate digital removal of debris.

Further impaction occurs at the nasal valve as air accelerates from around 2 m/s to 12-18 m/s (COLE 1982a). Clearance from this area is poor since the terrain is too inhospitable for the survival of the microscopic cilia which propel a protective blanket of mucus. Particles are either cleared further into the nose and swallowed or expelled into the external nose. The limitation of clearance into the body makes sense since it reduces the amount of toxic material actively transported into the body.

As the airflow leaves the nasal valve, 5% of the air continues upwards to the olfactory region of the nose. Short sniffs increase

this proportion to 20% (BERGLUND 1982) and give an aerodynamic reason for sniffing.

Most of the air turns sharply and encounters the nasal turbinates which are very well supplied with blood. This blood supply appears much greater than is necessary for air warming and humidification. The tissue is crowded with secretory cells and cilia. Most humidification is done by capillary transudate. Examination of a human skull can give a misleading impression as to the function of the turbinates, since they apparently do not produce a swirling airflow: Most of the air flowing through the nose flows along the main air passages each side of the nasal septum and the airflow along the hidden parts of the turbinates is minimal. These hidden parts do however connect with various sinuses in the skull and allow them to drain into the nose and also allow for air pressures to equilibrate (PROCTOR 1982).

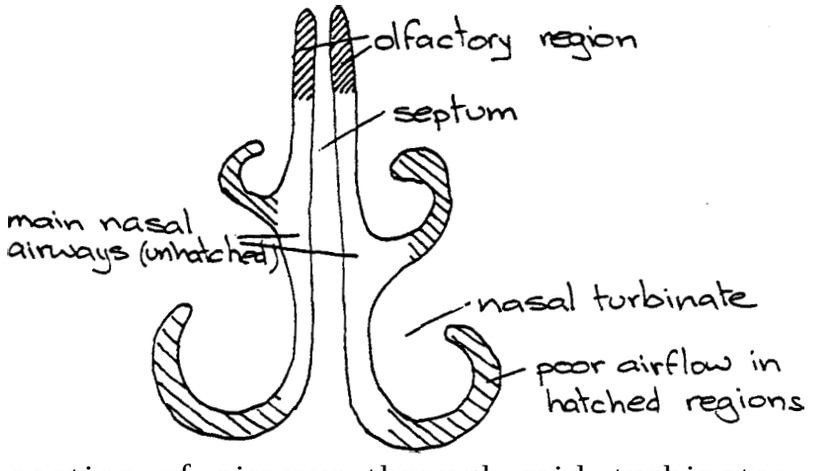

Figure 2 cross section of airways through mid-turbinates (after PROCTOR 1982)

Much of the impaction of particles and absorption of gases occurs at the front surfaces of the turbinates. The degree of absorption is relatively independent of the vapour concentration and actually increases with air velocity.

substance	percentage retention
ethanol	**43-70**
acetone	19-23
ammonia	>80
sulphur dioxide	70-96

Table I nasal absorption of vapours and gases (from HILDING 1976)

If the particles impacting in the nose are hygroscopic then the mucus may dry and the cilia die. This stops the cleansing action of the mucus flow and may explain the thousandfold increase in nasal cancers in wood workers, particularly with hardwoods where pyrolysis products from the heat of sawing may be expected (HILDING 1976). Toxic metals like cadmium also affect nasal clearance and likewise help explain the incidence of nasal cancers in workers exposed to cadmium fumes.

The flow past the turbinates is complicated by the presence of the "nasal cycle" which is present in about 80% of people (COLE 1982a). The cycle is of abour two hours duration during which the mucosa on each side of the nose swell alternately. We are unaware of it

because the overall airways resistance remains the same. The effect is more pronounced in some than others. Its function is unknown, but it would have some effect on air modification and olfaction since it cyclically varies the airflow through each side of the nose and the amount of nasal secretions, which increase as the mucous layer contracts (STOKSTED 1976).

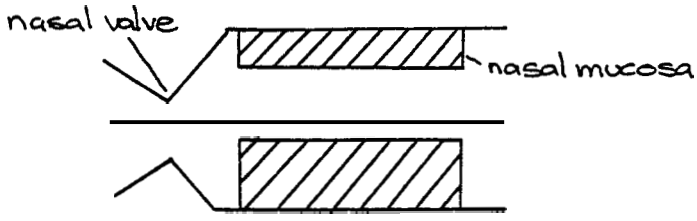

Figure 3 nasal air resistance schematic (after COLE 1982a)

Once clear of the nasal turbinates, the air again changes direction and accelerates down the trachea to join any air flow from the mouth. A minor amount of dust impaction occurs at this point and soluble gases are absorbed to a lesser extent.

The flow rates and patterns on inhalation and exhalation are somewhat different. On inspiration the flow rapidly plateaus as a result of the resistance to flow of the nasal valve and drops off sharply towards the end of the inspiratory cycle. On expiration the flow is markedly asymmetrical and tapers off at the end of exhalation. The pattern of flow is also very different, sluicing odour laden air from the olfactory region in preparation for the next inhalation.

The protective role of the nose in filtering air on its way to the lung have been known for some time, particularly in studies of silicosis (LEHMANN 1934, HILDING 1976) where the capacity of the nose to retain dust had a major bearing on the development of silicosis. One study showed the median nasal efficiency for filtering respirable dust was only 27.5% for silicotics but 56% for workers with 'normal' noses, In Australia we are still producing cases of silicosis in our dusty industries through inadequate control of dust so perhaps the early suggestions of selecting workers with a resistance to dust would have saved some lives.

3 OLFACTION

The strength of smell of a substance is determined by the water solubility of a substance and its volatility since it has to first become a vapour and then diffuse across the protective mucus covering the olfactory region of the nose. This explains some the variability of smell thresholds in different substances (RUTH 1986).

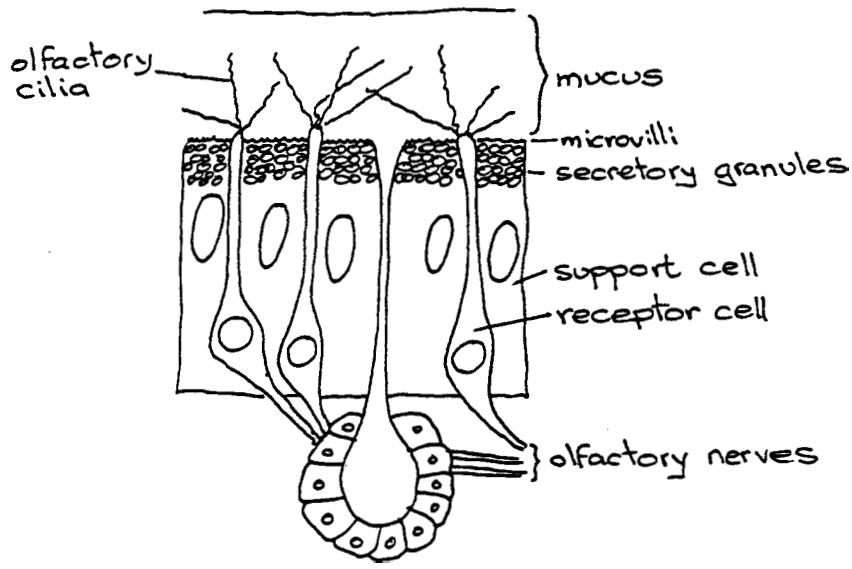

Figure 4 The olfactory receptor cell (after BERGLUND 1982)

The threshold of smell for different substances may be **absolute,** where the substance is detected but not identified. This is generally around 3 mg/m^3. At around **15** times this level (40mg/m^3) substances may be **identified** but a similar increase (550mg/m^3) is needed to **differentiate** the smell from other smells. The perception of smells is essentially logarithmic since a tenfold increase in the concentration of a substance only doubles the number of nerve impulses to the brain (NAUS 1983).

Olfactory fatigue can occur at a number of levels (NAUS 1983). The receptors may exhaust but recover within minutes on removal of the stimuli, usually with reduced sensitivity. For long exposures , the brain may fatigue and smell may be lost for hours but return on the next shift. This may be habituated and last for weeks or months even in the absence of the stimuli.

Example 1

A worker performing a preparation using chloroform in an unventilated coldroom held at 5°C relied on the smell of the chloroform to warn him to change his respirator cartridge. The operation was discovered and immediately suspended. If the vapour concentration was 10% of saturation or 10,000 ppm at 5°C, then protection by this method would limit the exposure to around 200 ppm at the best (CHEMINFO 1988). This is twenty times the Threshold Limit Value (TLV) of 10 ppm. Olfactory fatigue at 20–200 ppm could considerably dampen the workers sense of smell, so a chronic exposure to a time weighted average air concentration well in excess of the TLV could be possible.

The opinion on how to apply the threshold of smell to protection from solvent vapours using its TLV varies, from an implied figure of unity (SILK 1982) to a factor of 10% of the TLV (ANON. 1975). The published figures for smell thresholds are often based on the responses of a panel of around nine people, so unity would not protect all workers. Added to this is the effects of olfactory fatigue and diseases like colds which affect smell. A recent study (STEVENS 1987) showed that the normal concentration of the warning gas ethyl mercaptan of 14 ppb was sufficient to protect the young but 30% of the elderly could not smell it. This may become an increasing consideration in an aging population.

Causes of damage to the receptor cells include trauma or chemical. Traumatic damage may be caused by an accident where the nerve-fibres from the nose are sheared from the olfactory bulb. The result is a lack of smell or anosmia. Chemical damage is often followed by regeneration of receptor cells which then become sensitive to chemicals at one end and grows into the olfactory bulb at the other end (GESTELAND 1982). This neuronal repair by receptor cell replacement takes a month or so to complete. It is not known whether this is part of a continuing process or mainly a response to external damage (BREIPOHL 1986).

A number of theories of smell have been advanced over the years (PROETZ 1941, BERGLUND 1982). A theory still entertained in the 1940's was the wave theory of smell with 'olfactory waves' in the ultraviolet and violet part of the spectrum emanating from substances and causing pigmented granules noticed in the nasal epithelium to resonate at specific frequencies. Today the debate is about the details of how substances or their derivatives cause ion channels in the receptor cells to open and cause the receptor cell to fire impulses into the olfactory bulb and and from there to other parts of the brain. These theories all involve the presence of specific receptor molecules in the receptor cells.

Example 2

Workers in a small High Performance Liquid Chromatography (HPLC) laboratory reported sore eyes and throats and attributed this to formaldehyde from a ventilation duct. They said they could smell the formaldehyde, which has a smell threshold of around 0.5 ppm and a TLV of 1 ppm. Repeated measurements with Draeger tubes either failed to detect the formaldehyde or indicated very low levels. A number of measurements with a Lion Formaldemeter indicated levels up to 17 ppm. Inspection of the ventilation system did not show any obvious source of formaldehyde and levels inside and near the duct were not any higher. Draeger tube measurements of methanol levels gave the first clue to the problem and were an obvious measurement since this solvent is used extensively in HPLC. These measurements indicated air concentrations of methanol of 500 ppm, well above the methanol TLV of 200 ppm but below its threshold 'for smell of 2000-8000 ppm (CHEMINFO 1988). This also indicated the sensor in the Formaldemeter had a 2% sensitivity to methanol. There was still no indication of the source of the formaldehyde. Discussion of the laboratory back at the office revealed the vital clue- a bar radiator which was thermostatically controlled to control the humidity probably oxidized the methanol to formaldehyde. Since it was off during most of the investigations it was not considered important. Its cyclic nature also helped explain the contention that the formaldehyde levels seemed much worse on some days than others. Recommendations were made to limit the evaporation of methanol into the laboratory air and to discontinue the use of the bar radiator as a system of humidity control.

This example shows how easy it is to dismiss a problem when no obvious source of the problem can be found. Only repeated visits to the workplace and brainstorming produced the vital clue. The workers experienced symptoms and naturally attributed them to the substance they could smell, What they couldn't smell was causing the problems, but the initial report helped pinpoint the real problem.

4 SUMMARY

Only two aspects of the nose have been presented but they do show the vital role the nose can play in protecting the worker and assisting or misleading,the occupational hygienist. To be aware of the physics of nasal airflow and filtration and the chemical basis of olfaction can give the occupational hygienist a useful tool. We should be aware of individual variability of nasal function and changes with disease, chemical exposure and age. The nose is an organ which serves us well but cannot be trusted to perform unerringly.

5 ACKNOWLEDGEMENTS

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