ANOSMIA AND HYPOSMIA **MEDICAL APPENDIX**

DEFINITIONS

1. **Anosmia** refers to complete loss of sense of smell (olfaction); **hyposmia** refers to reduction in sense of smell. Either may be temporary or permanent. Other olfactory disturbances include distortions of normal smells (parosmia or dysosmia) and a heightened sense of smell to some or all odourants (hyperosmia).

PATHOPHYSIOLOGY

2. Taste and smell rely on chemical substances to stimulate their receptors and together form the *chemosensory system.* Their combination produces the sensation of flavour and dysfunction in one is often perceived as abnormality in the other.

3. There are two well-characterised nasal chemosensory systems: the free nerve endings of the trigeminal nerve and the sensory receptors of the olfactory nerve. A third, *vomeronasal,* organ exists but is of doubtful function in humans.

4. The free trigeminal nerve endings in the walls of the nasal passages respond non-selectively to a wide variety of volatile chemical substances, including high concentrations of most odourants. The olfactory receptors respond to chemical stimuli at lower concentrations and with far greater selectivity than trigeminal endings. In total anosmia, the capacity to distinguish between odours is lost, but the response to nasal irritation is usually preserved.

5. Olfactory function can be disrupted in three ways, the second and third of which are termed *neurogenic*:

    5.1. By nasal obstruction preventing volatile substances from reaching the receptors - *transport olfactory loss.*

    5.2. By impairment of receptor or cranial nerve function - *sensory olfactory loss.*

    5.3. By pathological processes affecting pathways from the olfactory bulb (the termination of the first cranial nerve) to the olfactory cortex and other parts of the brain.

6. Olfactory impairment is not always permanent. The receptor neurones have a lifespan limited to about 30 days, with continual replacement. This is an important factor in recovery from anosmia of certain causes, which may take months. Recovery of olfaction occurs in 2/3 of cases with cranial nerve palsy, sometimes as long as five years later.

CLINICAL MANIFESTATIONS

7. Qualitative changes in smell may be complained of in association with quantitative impairment and both may be accompanied by a perceived disturbance of taste.

8. Subjective tests for the sense of smell tend to be time-consuming and often imprecise. They rely on measuring minimal perceptible odour, identification, or adaptation. Even objective tests, using physiological measurements or evoked responses, may or may not be positive. In clinical practice, simple tests of identification of and discrimination between familiar substances. Appreciation of an odour, despite the inability to name it, excludes anosmia.

9. Unilateral non-obstructive loss of smell is not usually noticed by the patient but, if detected, may indicate a focal neurological lesion.

10. Cases of malingering can sometimes be exposed by comparing responses to odourants which differ in their propensity to stimulate trigeminal nerve endings. These are invariably stimulated by, for example, ammonia.

AETIOLOGY

11. Causes of anosmia and hyposmia can be classified thus:

    11.1. **Lesions of the Nose**

        11.1.1. **Deviated nasal septum** is a rare cause of disturbance of smell. Simple anatomical defects do not usually result in an abnormality of smell.

        11.1.2. **Nasal polyps**

        11.1.3. **Allergic and vasomotor rhinitis** are common causes of abnormalities of smell, but only rarely is the associated loss of smell total. In conditions such as hay fever and the common cold, the loss of smell is temporary.

        11.1.4. **Infective rhinitis** may damage considerable areas of olfactory mucosa if it becomes chronic and the affected areas do not regenerate.

        11.1.5. **Tumours**, including papilloma, adenoma, squamous cell carcinoma, esthesioneuroepithelioma and idiopathic midline granuloma.

        11.1.6. **Toxic fumes** may cause loss of smell, as may heavy smoking.

    11.2. **Lesions of the olfactory nerves.**

        11.2.1. **Injury**, either through a direct blow or from an occipital blow with shearing of nerve fibres, usually causes immediate and complete loss of smell.

        11.2.2. **Viral infections** such as influenza can cause great damage to the olfactory nerve fibres, replacing all the neuronal tissue with fibrous tissue.

        11.2.3. **Meningitis, sarcoid and neurosyphilis** may damage the olfactory tract.

        11.2.4. **Anoxia.**

11.2.5. **Degenerative conditions** include Parkinson's disease, Alzheimer's disease, motor neurone disease and multiple sclerosis (olfactory dysfunction occurs in about 40% of patients with MS)

11.3. **Intracranial Lesions.**

11.3.1. **Trauma** tends to give complete loss.

11.3.2. **Intracranial tumours** can affect the sense of smell in two ways, either by pressure on the olfactory nerve fibres or bulb, or by interference with the intracerebral pathways. Osteomas or meningiomas of the anterior fossa tend to diminish the sense of smell and at first this is unilateral. Frontal lobe tumours may do the same.

11.3.3. **Obstructive hydrocephalus**.

11.3.4. **Epilepsy**.

11.4. **Systemic diseases** in which the sense of smell may be impaired include diabetes mellitus, Paget's disease of bone, polyarteritis, cystic fibrosis and cirrhosis.

11.5. **Iatrogenic causes** include rhinoplasty, intracranial surgery, radiation therapy and certain drugs

11.6. **Psychogenic Disorders.**
Psychiatric disorders such as **psychoses** (including Korsakoff's), **depression** and **confusional states** can be accompanied by hallucination of smell (*phantosmia)*, and there is a specific olfactory reference syndrome. **Hysteria** and **malingering** have a place amongst these disorders. Olfactory hallucinations may also occur in Alzheimer's disease and alcohol withdrawal.

11.7. Anosmia is occasionally congenital, sometimes hereditary.

CONCLUSION

12. **Anosmia** and **hyposmia** are disturbances of the sense of smell which may be temporary or permanent. There are many possible causes, most of which are listed above. Many cases of anosmia and hyposmia resolve naturally, so no treatment is necessary. In others, treatment is that of the underlying cause, but the impairment is permanent in some cases.

REFERENCES

Finelli P.F. & Mair R.G. Disturbances of Taste and smell, in (eds) Bradley et al, Neurology in Clinical Practice, 3rd Ed. 2000, Boston Butterworth Heinemann, p.263-7.

Moore-Gillen V L. Abnormalities of smell. In: Eds. Mackay I S, Bull T R. Scott-Brown's Otolaryngology. Volume 4. Rhinology. 6th Ed. 1997. Oxford. Butterworth-Heinemann. p. 4/5/1-4/5/9.

Kennard C. in (ed) Donaghy M., Brain's Diseases of the Nervous System. 11th Ed. 2001. Oxford. Oxford University Press. p.312-3.

Leopold D. Disorders of olfactory perception: diagnosis and treatment. In Chem. Senses 2002 Sep;27(7):611-5.

October 2002