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Smell disorders in ENT clinic

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Abstract. Olfactory disorders may have several causes. Nasal polyposis or chronic sinusitis can result in nasal obstructions that block the access of odorants to the olfactory epithelium, and this can explain the development of olfactory disorders. On the other hand, when nasal endoscopy has revealed that the nasal cleft is free of inflammatory or tumoural disease, olfactory disorders may be explained by neuroepithelial or central nervous system disturbances. This paper will provide information about current approaches to smell disorders in otorhinolaryngology. Major causes will be reviewed as outcomes after medical or surgical treatment. An algorithm will also be given to standardise clinical investigations, including psychophysical olfactory testing, imaging and electrophysiological examinations.

Introduction

Clinical olfaction disorders are sensorineural diseases that can be seen in daily ENT practice. They can have a variety of causes and have a serious impact on quality of life. These symptoms are more frequently present in the elderly; the incidence in the population aged over 65 years is 50%.[1] Olfactory disorders linked to sinonasal disease are the main type of presentation for otolaryngologists. Impaired olfactory function is also found in neurodegenerative disorders such as Alzheimer's disease or Parkinson's disease.[2] This chapter deals exclusively with disorders of the physiological sensation of olfaction. It offers a guide to diagnostic and therapeutic practice based on a review of the literature up to June 2004 using Medline. By contrast with the situation for other ENT pathologies, this review did not identify any "evidence-based medicine" articles for the various sensorineural disorders.

Low prevalence and multiple aetiologies explain the lack of evidence-based medicine in the field of sensorineural olfactory disorders.

Anatomy and Physiology

The olfactory neuroepithelium is a pseudostratified columnar epithelium. This neuroepithelium is situated in the superior aspect of the nasal vault in the cribriform plate medially to the middle turbinate and has a surface area of 1.5 to 2.5 cm^2 in adults in each nasal fossa (Figures 1a-d). Some areas of the olfactory epithelium are also present in the superior turbinate, the superior septum and the middle turbinate.[3] The neuroepithelium represents the main olfactory receptor organ and is linked to the main olfactory system (cranial nerve I), which mediates odour sensation and is responsible for determining flavours (Figure 1b). Somatosensory sensations from the nasal mucosa (burning, cooling, irritation...) are transported by the trigeminal system (Trigeminal intranasal somatosensory system, cranial nerve V).
Intact somatosensory and olfactory sensations are essential for olfactory acuity.

The nervus terminalis (Vomeronasal organ, Jacobson's organ, cranial nerve O) is located in the anterior aspect of the septum. Its function and exact incidence in humans are a matter of debate (Figure 1e).

There are many distinct cell types in the olfactory epithelium: olfactory receptor neurons (ORN), microvillar cells, supporting cells, globose basal cells, horizontal basal cells and the cells surrounding the Bowman's glands[3] (Figure 1c). The ORN is a bipolar sensory receptor neuron and is the main receptor organ, transforming odorant-receptor interaction into electric signals. There are approximately 10-15,000,000 ORNs in the adult neuroepithelium in each nasal fossa. The olfactory receptors are located in the ciliated dendritic ends of the ORNs. Cilia are

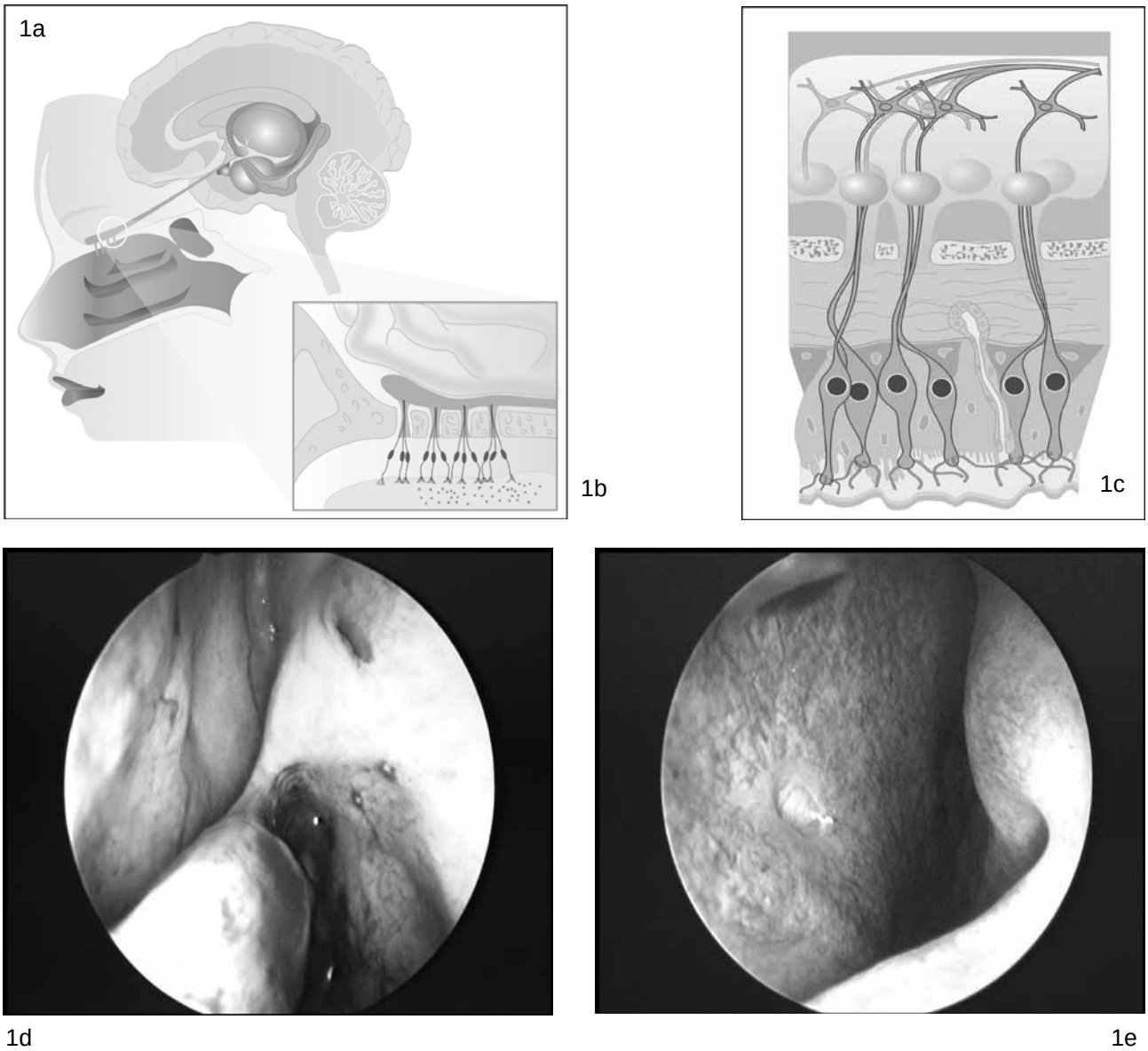


Figure 1

a. Schematic diagram representing a sagittal view of the olfactory pathways.
b. Schematic diagram of the olfactory neuroepithelium with odorants stimulating olfactory receptor neuron and olfactory axons to the olfactory bulb.
c. Schematic diagram of the neuroepithelium with olfactory receptor neuron, olfactory axons with connexion into the olfactory bulb; glomeruli and mitral cells.
d. Endoscopic view of the olfactory cleft (left).
e. Endoscopic view of Vomero-nasal organ or Jacobson's organ (left).

located on the exposed dendritic tips of ORNs and it is hypothesised that they increase the sensory surface area available for contact with odours. ORN cilia are different from respiratory cilia at the morphological level and are immotile. In the mucus, olfactory binding proteins (OBP) may help to amplify the sensory response by trapping odorants and carrying them with inspiration to ORN. Each receptor cell expresses a single odorant receptor gene. Until now, approximately 1,000 genes and 350 classes of receptors have been identified in the adult human neuroepithelium, accepting a range of molecular entities. The microvillar cells and the cells around the Bowman's gland play a role in mucus production and serve as stem cells for supporting cells. Supporting cells play a role in ion and water regulation, participate in protein degradation and in the metabolism of xenobiotic

compounds, and have a glial-like function in the regulation of K+ in the extracellular fluid compartment. They provide structural support and play a guiding role in the development of olfactory receptor neurons. Basal cells (globose and horizontal) are stem cells for postnatal neurogenesis. It should be pointed out that ORNs are capable of regenerating[3] from basal cells after damage. This phenomenon is hyperinduced after bulbectomy in animals.

ORNs produce olfactory marker protein (OMP), which modulates the olfactory signal cascade and olfactory neurogenesis. ORNs send unmyelinated axons through the basal lamina and the cribriform plate of the ethmoid bone to terminate in glomeruli on mitral and tufted neurons in the olfactory bulb of the brain (Figure 1b). The olfactory transduction of an odorant stimulus into a electric signal occurs at the external surface of the immotile cilia located in the ORNs. The odorant-receptor interaction is located in the seven transmembrane-spanning domain receptors of the G-protein-coupled receptors. This produces an electric signal through a biochemical messenger (cAMP) which is transported by the ORN axons. The axons of ORNs expressing the same odorant receptors converge onto defined glomeruli in the olfactory bulb. A wealth of data indicates that individual glomeruli are the convergence points of ORNs that display a single response type and that therefore probably express the same receptor protein. Coding is therefore the result of spatial processing. But there are also temporal codes (encoding time) and combined spatio-temporal codes (Figure 2). Mitral cells then project this alphabet of features into the piriform cortex where the features are combined into perceptually whole odours initiated in the periphery. At this point, orbitofrontal cortical projections, with or without thalamo-cortical relay, are finally present. The piriform cortex, the hippocampus, the amygdala and the orbitofrontal cortex combine to allow the association of odorants with sensory cortex, memory and hedonic reactions. Efferent stimuli can then

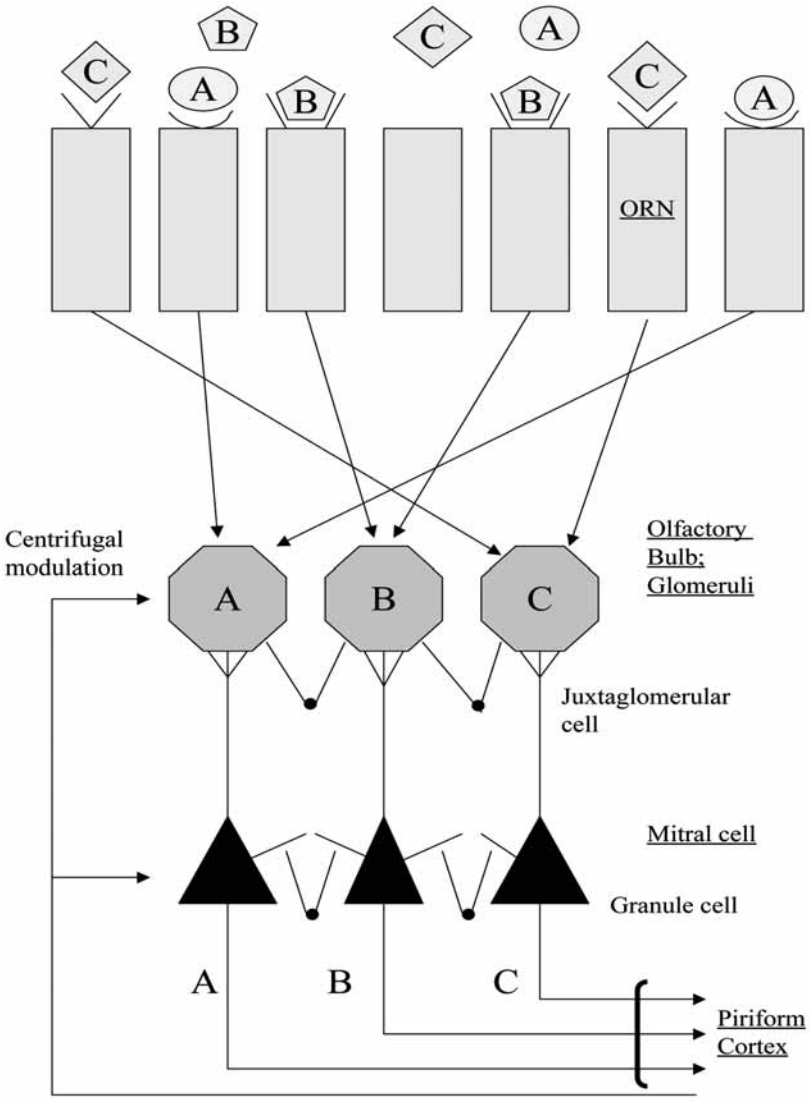


Figure 2

Basic schematic neural connectivity of the main olfactory bulb and piriform cortex. Odorant molecules are broken down into informational features by binding with specific receptors in the nose. Olfactory receptor neurons located in the neuroepithelium express one of 1000 different receptor proteins and are randomly scattered. Receptors expressing the same receptor protein converge onto a small number of exclusive glomeruli in the olfactory bulb (receptor types are labelled A, B and C in this example). The receptors are hypothesised to be responsive to individual odorant features rather than odorant molecules as a whole. Mitral cells receive receptor input from a single glomerulus and then project to the piriform cortex. Within the olfactory bulb, a neuron lateral inhibition is mediated by juxtaglomerular and granule cells. Modulatory and descending inputs also converge in the olfactory bulb to modulate the sensory responses.

shape behaviour appropriate for the given stimulus.

Classification and definitions

Olfactory disorders can be broken down into quantitative or qualitative disorders.

Quantitative disorders include anosmia (the inability to detect olfactory sensations (i.e., absence of smell function)), hyposmia or microsmia (decreased sensitivity to odorants), partial anosmia (ability to perceive some but not all odorants), hyperosmia (abnormally acute smell function).

Qualitative disorders include dysosmia (distorted or perverted smell perception of odorants stimulation (cacosmia, parosmia)), phantosmia (dysosmic sensation perceived in the absence of an odour stimulation (olfactory hallucination)), olfactory agnosia (inability to recognise an odour sensation).

Presbyosmia is the term sometimes used to designate the age-related impairment of smell perception.

It is also interesting to classify smell dysfunction into three general classes:

a. conductive or transport impairment as a result of the obstruction of the nasal passage
b. sensorineural impairment as a result of damage to the neuroepithelium and
c. central olfactory neural impairment as a result of central neural damage.

This classification may appear confusing, for instance because the olfactory nerve is both peripheral and central. It should be pointed out that a classification based on cause seems to be more reliable. The definitive classification of a patient into a category is sometimes difficult as many patients experience different smell dysfunctions (for example, patients with atrophic rhinitis often present with both hyposmia and cacosmia). On the other hand, the characterisation of the olfactory trouble based on the precise location in the olfactory pathways is also difficult (for example, patients with postviral anosmia often present with damage both to the neuroepithelium and to central elements of the system).

Table 1

Causes of olfactory disturbance

Drugs and Medications
Endocrine/Metabolic
Adrenocortical insufficiency
Cushing's syndrome
Hypothyroidism
Pseudohypoparathyroidism
…
Industrial products, dusts, metals, volatile compounds
Acetone
Benzene
Chromium
Paint solvents
Spices
Trichloroethylene,…
Rhinologic disease
Inflammatory disease
Allergic rhinitis
Chronic rhinosinusitis with nasal polyposis
Atrophic rhinitis
Post-viral olfactory loss
Post-traumatic olfactory loss
Post-surgical
Tumoural
Intracranial neoplasms
Gliomas
Olfactory meningioma,…
Intranasal neoplasms
Esthesioneuroblastoma
Adenocarcinoma,…
Neurological disease
Alzheimer's disease
Parkinson's disease,…
Psychiatric
Nutritional, metabolic
Cirrhosis of liver
Renal insufficiency
Gout,…

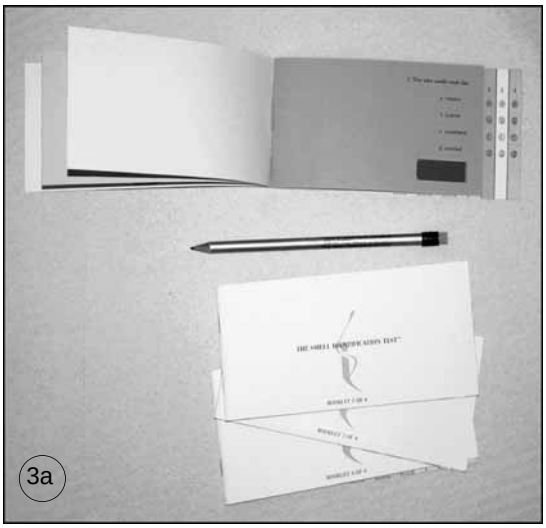
Clinical evaluation

The aetiology of most smell disorders can be ascertained by questioning the patient about the characteristics of the disorder (Table 1). The nature, timing, onset, duration, presence of fluctuation, antecedent events, medical history, and the associated symptoms are of primary importance and help to identify the cause of the sensory deficit (Table 2). The clinician must then evaluate the extent of the smell disturbance using psychophysical testing as a semi-objective measure. It is not sufficient to accept patient reports about the sensory deficit without verifying its presence and assessing magnitude.[4,5] Standardised quantitative olfactory testing is using to characterise the olfactory loss, to establish the validity of the patient's complaint, to detect malingering, to monitor changes over time (natural evolution, post-

Table 2

Checklist for clinical management in olfactory disturbance

1. Is taste also affected?
2. Description: anosmia, hyposmia, parosmia, dysosmia,…
3. Is trigeminal sensation preserved?
4. Localisation: right, left, both sides.
5. Timing? Persistent or intermittent, fluctuation?
6. Onset? Sudden, gradual,…
7. Precipitating event? Surgery, trauma, toxic,…
8. Medications? and past medical health?
9. Rhinologic evaluation and endoscopic endonasal evaluation.
10. Psychophysical testing of olfaction (semi-objective measure).
11. Chemosensory event-related potential (objective measure) if available.
12. Imaging technique; CT scan and/or MRI.



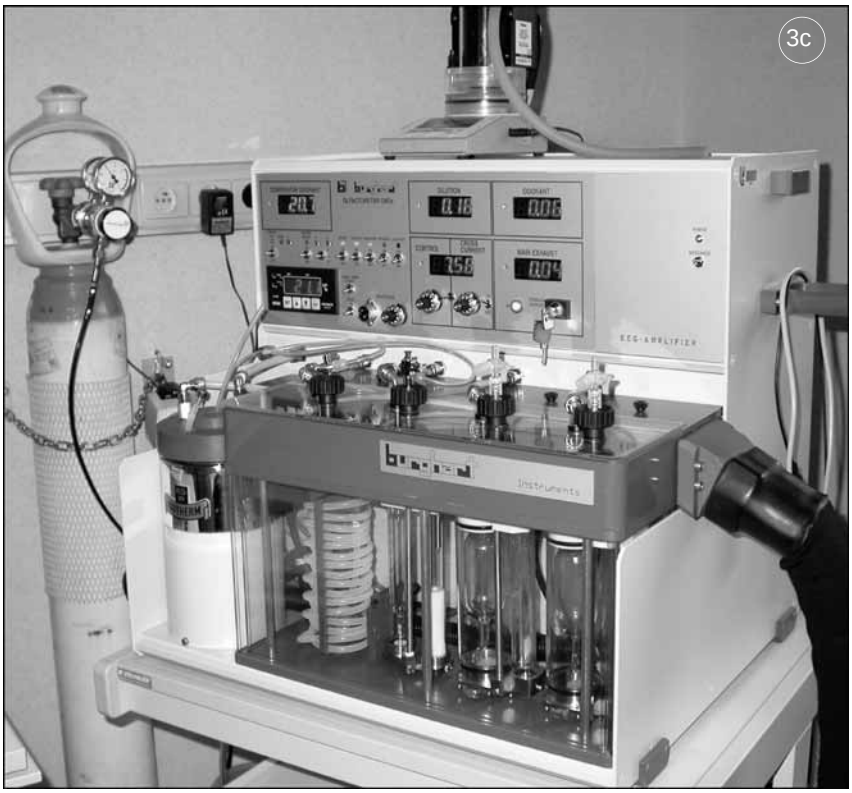


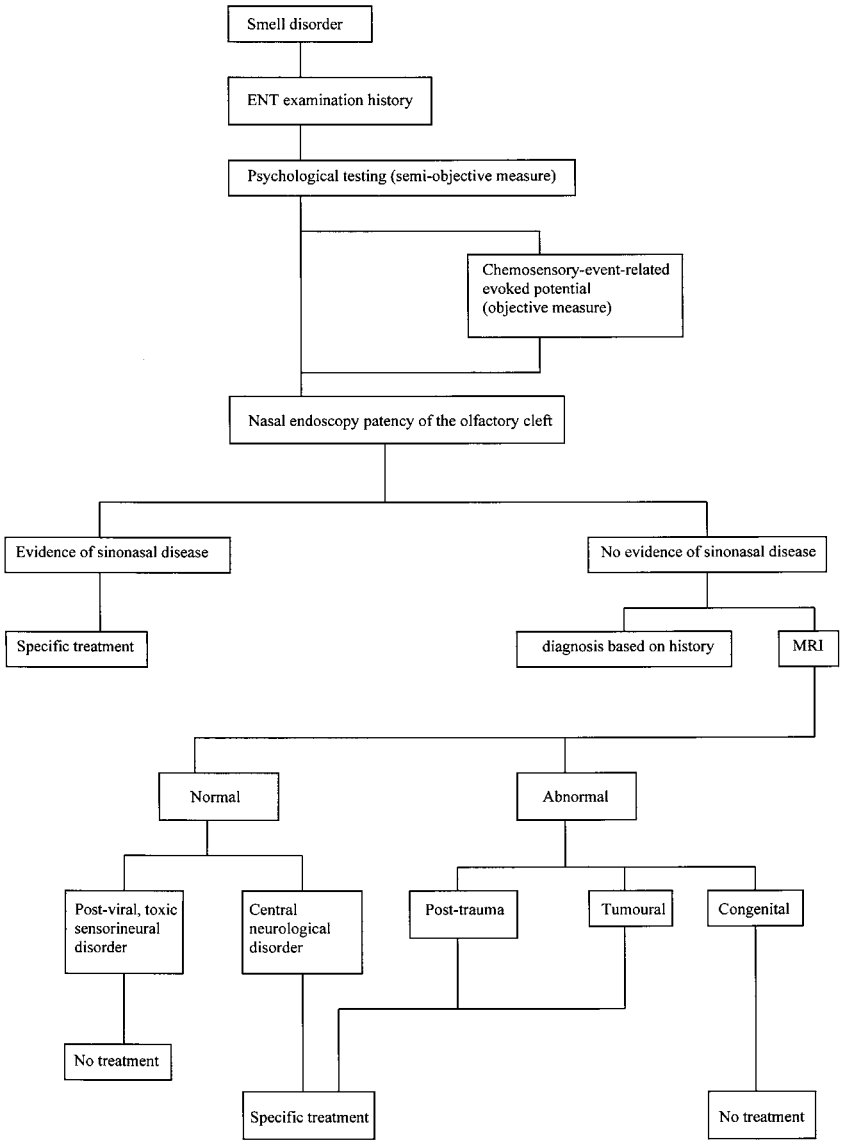
Figure 3
a. Standardised quantitative olfactory testing by scratching microencapsular label: the 40-odor University of Pennsylvania Smell Identification Test (UPSIT).
b. Standardised quantitative olfactory testing using odorant impregnated felt tip pen: the Sniffin'Sticks Test.
c. Olfactometer delivering olfactory stimulus for electrophysiological study using chemosensory event-related potential.

therapeutic effect…) and to provide data for establishing disability compensation. Odours are presented to the subject using an odorant-impregnated felt-tip pen (the Sniffin'Sticks test) or by scratching a microencapsular label (the 40-odor University of Pennsylvania Smell Identification Test; UPSIT) (Figures 3a, 3b).[6,7] This evaluation includes olfactory threshold tests, odour identification tests using multiple-choice identification and odour discrimination testing. Others devices have been proposed such as glass sniffing bottles, odorised strips of paper or plastic squeeze bottles. Psychophysical testing of olfaction has revealed that scores are higher in women than in men and that scores decrease after the age of 65 and in smokers.[5-7]

A more objective way to analyse olfactory disturbances is to use chemosensory event-related potentials.[8-10] Electrophysiological techniques are only available in specialised centres with well-designed olfactometers to administer the appropriate olfactory stimulus. This technique is well documented in research, and it can be useful in detecting malingering, in forensic tests and in some clinical situations where there is a loss of olfactory acuity but not trigeminal sensitivity (for example, in postviral anosmia) (Figure 3c).

The next step for the ENT practitioner is to perform an otorhinolaryngological examination focusing particularly on the nasal fossa and the olfactory cleft. An endoscopic evaluation of the meati (middle and superior) and of the olfactory cleft is mandatory. The

Table 3 (Algorithm)



See text for MRI aspect in postviral olfactory loss

vomeronasal organ can also be visualised during this examination despite the fact that the percentage of humans with this organ is not well established. If there is evidence of sinonasal disease (nasal polyposis, inflammatory disease, tumour…), specific treatment must be initiated. If there is no evidence of sinonasal disease and if history has not revealed any possible explanations for the sensory deficit, imaging of the olfactory bulb, olfactory tracts, and cortical brain parenchyma are mandatory (level III).

Functional MRI is used in research. MRI may reveal the absence of the olfactory bulb in congenital olfactory disorder, contusion in the area of the brain dedicated to olfactory pathways in posttraumatic olfactory disorder and tumoural lesions such as esthesioneuroblastoma. Central neurological disorders, toxic olfactory disorders and idiopathic olfactory loss do not usually result in pathological changes in the olfactory pathways revealed by MRI. This is also true of postviral olfactory loss, although some data have revealed a decrease in size of the olfactory bulb compared to controls.[11] Some laboratory tests are helpful in detecting underlying medical conditions (nutritional deficiencies, endocrine disorders, liver disease…) (Table 3).

A combination of different factors must also be taken into account, since patients with allergic disease, for instance, are more susceptible to olfactory loss from viral aggression due to cumulative damage of the neuroepithelium. Finally, some patients with an olfactory disturbance may be considered idiopathic since no aetiology has been found for their sensorineural disorder.

Olfactory disorders related to sinonasal disease

Sinonasal diseases encompass many different diagnoses that may include smell disorders with different incidence rates and different characteristics (Table 4). The degree of olfactory loss is usually linked to the severity of the sinonasal disease, with most loss being found in patients with rhinosinusitis and nasal polyposis. Olfactory loss is not usually related to decreases in nasal patency measured with rhino(mano)metry studies but it is related to the patency of the olfactory cleft. The relation between olfactory loss and sinonasal disease has been studied in different ways: patients with the symptom and the sinonasal disease have been compared to controls, and there have been medication trials for patients with olfactory loss and a rhinologic diagnosis.

Table 4

	Incidence	Olfactory trouble	Characteristics	Treatment
CRS –NP	20-30%	Hypo > anosmia	Fluctuation	Corticoid
CRS+NP	70-90%	Hypo = anosmia	Fluctuation	Corticoid
Allergic rhinitis	15-20%	Hyposmia	Fluctuation	Corticoid and anti-histamines
Atrophic rhinitis	60-80%	Hyposmia = Cacosmia	Persistent	No
Postviral olfactory loss	Difficult to establish	Hyposmia >(slightly) anosmia + Parosmia	Persistent	No, restitutio in 40–60%, mostly for hyposmia within the first year
Posttraumatic olfactory loss	Related to the severity of the trauma: Mild; 10 % Severe; 50-60%	Parosmia = Anosmia = Hyposmia	Persistent	No, restitutio for mild trauma, in 20–30%, within the first year

Evidence-based medicine lacks any supporting data because the vast majority of these studies have been conducted in a non-randomised and uncontrolled way. But there is a tendency to believe that corticoid nasal spray and corticoid per os are the best candidates to restore olfactory disorders related to sinonasal disease (level III).[12-14]

Allergic rhinitis, chronic rhinosinusits with or without nasal polyposis, and atrophic rhinitis are medical conditions that may involve impaired smell. Psychophysical olfactory testing has revealed that scores decline in the following sequence: Normal > allergic rhinitis > non-allergic rhinitis[12-20] and Normal > chronic rhinosinusits without nasal polyposis > chronic rhinosinusitis with nasal polyposis[21-27] (Table 4).

Allergic patients have impaired olfactory acuity compared to controls, with 23.1% having a threshold at or above the 2.5 percentile of the controls.[12] It has also been demonstrated that olfactory function is worsened after allergen challenge in allergic patients, and that this effect is not related to the decrease in nasal patency. Acute viral-related rhinitis or rhinosinusitis, hypertrophied adenoids and rhinitis medicamentosa are also medical conditions involving olfactory disturbances.[28] On the other hand, structural abnormalities such as deviated septum or alar cartilage dysfunctions are not associated with impaired olfactory performance.[29] Smell disorders are one of the most important symptoms in nasal polyposis; they are related to the evolution of the disease, are progressive and seem to be related to the stage of the disease.[27] The causes of olfactory loss in nasal polyposis are regional, mechanical and inflammatory factors rather than sensorineural deficit. The fact that the number of patients with impaired smell is lower that the number of patients with lower scores for the psychophysical testing is explained by the progressive pattern of the sensitive deficit in nasal polyposis. Smell and taste are both compromised in nasal polyposis. Taste is the perception of flavours and aromas. It includes the activation of gustatory, trigeminal and olfactory components. However, a large number of patients with a loss of smell have no taste problem. This is explained by the higher percentage of retronasal olfactory function in patients with nasal polyposis, which is more related to the taste than orthonasal olfaction.[30]

Smell problems may benefit from corticoid treatment for nasal polyposis. Oral corticoids usually result in a marked but limited benefit and topical corticoids usually produce a moderate effect that is more persistent since these medications may be taken for a longer period due to the lack of major side-effects[12,13,23,24,31] (level III). When medications fail, functional endoscopic sinus surgery (FESS) is proposed to the patient and the effects of surgery on smell disturbance have also been studied. However, these studies are difficult to compare because of the dissimilarity of the type of surgery (polypectomy, functional surgery, radical surgery, preservation of the middle turbinate…), the type of medications used before and after surgery, the type of psychophysical testing (when present) and the type of patients included (allergic, smoker, asthma …).[22,23,26,32-37] On the basis of a well-designed study without any psychophysical testing (olfactory disturbance was assessed by the patient and self-

rating using a visual analogue scale), it can be concluded that olfactory disturbance is improved after radical surgery and that this improvement persists when topical corticoids are prescribed after surgery.[26] The score after surgery is close to the score obtained after oral corticoid treatment prescribed before surgery. FESS is an efficient mode of treatment for olfactory disturbance in nasal polyposis; the more extensive the surgery, the more beneficial this treatment is for this symptom (level III). It should also be pointed out that patients with nasal polyposis with severe olfactory loss undergo an improvement in olfactory acuity after therapy but that the vast majority of them remain in the hyposmia range.[26]

When surgery fails for this symptom, this may be due to neuroepithelial alterations secondary to chronic inflammation, to nasal polyposis recurrence or to causes related to the surgical procedure itself. Mechanisms of olfactory injury resulting from sinonasal surgery are: direct trauma to the neuroepithelium, traction on the olfactory nerve due to cribriform motion, vascular compromise, secondary atrophic rhinitis, nasal packing and infection.[38]

Postoperative smell dysfunction may also be related to the presence of Staphylococcus aureus as it has been demonstrated that secreted toxins could damage olfactory receptor neurons.[39]

Septal and/or rhinoplasty surgery are proposed to the patients to increase nasal patency and the effect of these procedures on smell perception have been studied using psychophysical testing. One can conclude that these procedures have neither an adverse effect nor a positive effect on olfactory performance and that an olfactory acuity increase must not be the primary goal of them.[29]

Table 5

Drugs influencing smell perception

Analgesic
Antipyrine
Local anaesthetics
Cocaine HCL
Procaine HCL
Tetracaine HCL
Antimicrobials
Fluoroquinolon
Macrolides
Griseofulvin
Neomycin
Tetracyclines
Antirheumatics
Mercury/gold salts
D-Penicillamine
Antithyroids
Propylthiouracil
Thiouracil
Cardiovascular, hypertensives
Angiotensin conversion enzyme inhibitors
Nifedipine
Amlodipine
Gastric medication
Cimetidine
Hyperlipoproteinaemia medications
Intranasal saline solutions
With Acetylcholine, Menthol, Zinc sulphate
Opiates
Sympathicomimetics

Postviral olfactory loss[14,40-42]

Acute viral rhinitis or common cold is often associated for a limited period with olfactory disturbance. In some cases, the sensory trouble is more pronounced and subsides over time, leaving the patient with a noticeable olfactory deficit. The distinction between smell disorder due to oedema during viral infection and smell disorder due to neuroepithelial viral damage is difficult to establish and incidence is difficult to evaluate. Viral agents include influenza and parainfluenza, for example, and there is no advantage to monitoring antibodies with serology in the management of postviral olfactory loss because the viral infection will no longer be present at the time of diagnosis. What the predisposing factor is for viral damage remains unclear but one could argue that such losses become manifest in middle or older age, therefore suggesting that cumulative insults to the neuroepithelium may be involved. Neuroepithelial regeneration is impaired in older age, when there is a decrease in the proliferation of basal cells and immature neurons The pathophysiology of neuroepithelial damage due to a viral agent is based on different mechanisms: periciliary event, toxic attack of the neuroepithelium, direct insult to the olfactory epithelium and even inflammation of the olfactory tract with intracerebral viral inflammation. It has been shown that many viruses are capable of invading the central nervous system via the olfactory epithelium.

Typically, patients with postviral olfactory loss are women aged between 40 and 60 who present an olfactory deficit after a severe upper airway infection. The interval between the upper airway infection and awareness of the olfactory problem is usually 2–3 months, although viral destruction is present before this. In cohort studies, hyposmia is slightly more prevalent than anosmia. It is also interesting to note that parosmia is frequent. MRI studies reveal that olfactory bulb volumes are reduced compared to controls in patients with postviral olfactory loss, especially those with parosmia.[11] It has not yet been determined whether this feature is a consequence or a cause of the disease. There is no medical treatment that provides any objective benefit in postviral olfactory loss,

even though many medications have been used in the past: examples being oral corticoids, vitamin B, zinc sulphate… (level III). As soon as the viral infection is over, there is absolutely no advantage to using medication to restore olfactory acuity.[42,43]

Recently, the role of alphalipoic acid in a dosage of 600 mg/day has been emphasised in an unblinded and prospective clinical trial. Alphalipoic acid stimulates the expression of nerve growth factor, enhances motor nerve conduction and perinervous microcirculation and has a potent antioxidative effect with less neural damage due to free radicals.[44] It remains unclear when the treatment must be started and whether there is any advantage to initiating the treatment during a common cold in order to prevent sensory deficit.

Restitutio ad integrum after a postviral olfactory loss is not the rule, although significant improvement has been found in 40–60% of patients within the first three years (Table 4). Higher chances of recovery are found in younger patients, in patients with hyposmia, when parosmia is not present for a long period of time and when there are no other predisposing factors to olfactory disturbance.[45]

Posttraumatic olfactory loss

Olfactory loss is a frequent sequella of traumatic head injury. Its incidence is difficult to estimate as patients are unaware of their loss until some time after injury.[45,46] This incidence may be estimated at 50–60% in severe trauma. The olfactory disorder is usually an anosmia, although hyposmia may be present if the trauma is moderate. Post-traumatic olfactory loss may be explained by sinonasal contusions or fractures with or without direct damage to the olfactory apparatus, by tearing or shearing of olfactory nerve elements or by contusion or haemorrhage within the olfactory-related brain regions. It is important to consider rhinoliquorrhoea in this condition. Restitutio ad integrum is a feature of 20–30% of cases, mostly involving slight to moderate trauma, when imaging (MRI) does not reveal any contusion and takes place a few months after the trauma. There is no advantage to proposing any medication or surgical procedure.

Other Causes

Anosmia is rarely the consequence of the absence of any olfactory bulb and/or olfactory tract as revealed by MRI examination.[47] In association with endocrine dysfunction, this deficit is described as a Kallman's syndrome, a hereditary isolated condition with a hypogonadotrophic hypogonadism (cryptorchidism, deafness, craniofacial abnormalities, renal anomalies…).

Olfactory loss may also be due to the presence of a tumour. Intracerebral tumours (glioma, olfactory meningioma…) or intranasal tumours (esthesioneuroblastoma, adenocarcinoma…) may be associated with a sensorineural disorder. Neurological disorders and olfactory disturbances are combined in Alzheimer's disease and in Parkinson's disease. Finally, industrial toxics, dusts, metals, volatile compounds and internal medicine conditions such as hypothyroidism may predispose to olfactory disturbances. Finally smell disorders may be related to drugs (see Table 5).

Conclusion

In many patients, history and ENT examination are adequate to determine the aetiology of the olfactory disturbance. Before treatment and specific management, it is advisable to measure olfactory function, especially if olfactory disorder is the patient's main complaint. Imaging and electrophysiological examinations are of primary interest, not only for research but also to validate specific treatment and for legal purposes. It should be also pointed out that medical treatment has not been shown to produce clear benefits in olfactory disorder when the olfactory cleft is free of any inflammatory processes, as in postviral olfactory loss. In these cases, spontaneous recovery is sometimes seen, especially in younger patients and when olfactory loss is recent. On the other hand, in nasal polyposis or chronic sinusitis patients, systemic corticosteroid treatment and/or surgery seem to result in an improvement of olfactory function.

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Patient information

Olfactory function depends on odorant molecules interacting with olfactory receptor neurons located in the neuroepithelium in

each nasal fossa. Olfactory acuity is higher in women than in men, and decreases with advancing age and in smokers. Smell is associated with taste and both sensory systems (known as flavour) are essential to our lives.

Olfactory disorders have a negative impact on quality of life.
Rhinologic disease in the form of allergic rhinitis, chronic rhinosinusitis or nasal polyposis may interfere with olfactory acuity. An olfactory loss may persist after an acute upper viral infection or after a head trauma. Toxic agents, medication, neurological disease and rare tumoural disease may also be associated with a smell problem. The management of olfactory loss includes a detailed history, a rhinologic evaluation, psychophysical testing of olfactory performance and, in some cases, imaging (CT scan or MRI).

Management and prognosis depend on the cause of olfactory loss.

The effective treatment of sinonasal-disease-related olfactory loss is possible, although not always successful, and it includes medication (systemic or topical steroid) and surgical approaches.

Olfactory loss that is not related to sinonasal disease is difficult to treat since the clinical benefit of medication has not yet been demonstrated.

The objective assessment of olfactory performance is based upon electroencephalographic evoked response (chemosensory event-related potentials).

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CME questions

1. What statement is incorrect? The olfactory neuroepithelium is located:

   A - In the superior aspect of the nasal vault medially to the middle turbinate.
   B - In each nasal fossa
   C - With some area in the superior turbinate
   D - With some area in the middle turbinate
   E - With some area in the inferior turbinate

2. What is incorrect? The olfactory receptor neuron (ORN)

   A - Is a bipolar sensory receptor neuron
   B - Is the main receptor organ transforming an odorant-receptor interaction into an electric signal
   C - Is a sensory neuron capable of regenerating
   D - Has cilia at the dendritic end which are similar to respiratory cilia
   E - Sends an unmyelinated axon to the olfactory bulb

3. In which situation is magnetic resonance imaging ineffective for patients with olfactory loss?

   A - Olfactory meningioma
   B - Esthesioneuroblastoma
   C - Idiopathic olfactory loss
   D - Nasal polyposis
   E - Congenital olfactory disorder

4. The incidence of olfactory problems in chronic rhinosinusitis with nasal polyposis is between

   A - 0-20%
   B - 20-40%
   C - 40-70%
   D - 70-90%
   E - > 90%

5. What is incorrect? In the scores obtained by the psychophysical testing of olfaction

   A - Normal > allergic rhinitis > non-allergic rhinitis
   B - Women > men
   C - Smoker > non-smoker
   D - Normal > chronic rhinosinusitis without nasal polyp > chronic rhinosinusitis with nasal polyp
   E - Normal > postviral olfactory loss

6. Olfactory problems are present in all these circumstances but one.

   A - Septal deviation
   B - Atrophic rhinitis
   C - Rhinitis medicamentosa
   D - Nasal polyposis
   E - Common cold

7. Postviral olfactory loss is characterised by all these features but one.

   A - Parosmia
   B - More frequent in men
   C - Restitutio in 40–60% within the first year, mostly for hyposmic patient
   D - Secondary to viral agents such influenza, parainfluenza,…
   E - Secondary to viral attack of the neuroepithelium

8. What statement concerning posttraumatic olfactory loss is incorrect?

   A - 50-60% incidence in severe trauma
   B - No medication helping to restore olfaction
   C - Restitution in 20-30% within the first year for mild trauma
   D - Important to consider rhinoliquorrhoea
   E - It is helpful to propose surgical decompression of the olfactory bulb

9. A CT scan of the nose and of the paranasal sinuses is helpful in the diagnosis of olfactory problems in one circumstance. Which?

   A - Congenital olfactory disorder
   B - Post-medication olfactory problems
   C - Post-toxic olfactory problems
   D - Alzheimer’s disease
   E - Chronic rhinosinusitis

10. Which statement about olfactory performance is incorrect?

    A - It is due to retronasal olfaction in patients with nasal polyposis.
    B - It is secondary to both olfactory and trigeminal information.
    C - It is decreased when taste is impaired.
    D - It is related to nasal patency measured with rhinomanometric studies.
    E - Deficit is most pronounced in older males.

Answers: 1E; 2D; 3C; 4D; 5C; 6A; 7B; 8E; 9E; 10D