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Review Article

Diagnosis and management of olfactory disorders: survey of UK-based consultants and literature review

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Abstract

Background: The diagnosis and management of olfactory disorders is an often neglected topic in otolaryngology. This article evaluates current clinical practice within the United Kingdom, and provides a literature-based review of the diagnosis, management and prognosis of olfactory pathology.

Design: A questionnaire was sent to consultant and associate specialist members of the British Association of Otolaryngologists and Head and Neck Surgeons. The responses were documented to gain an impression of how olfactory disorders are managed in the United Kingdom. The literature relating to olfactory dysfunction was then evaluated and the findings summarised.

Conclusions: Management of olfactory pathology varies across the United Kingdom. The literature suggests that chemosensory testing is optimal and that both forced-choice and threshold testing should be applied if objective evaluation is required. Imaging can be of value but the appropriate technique should be used. Olfactory function can recover following head injury, viral infection and chronic sinonasal disease, although varying degrees of dysfunction are likely to persist. There is a role for the use of corticosteroids, particularly when administered systemically. More research is needed to establish the appropriate dose and length of treatment.

Key words: Olfaction Disorders; Smell; Diagnosis; Therapy

Introduction

Olfaction is the sensation arising from the nasal cavity, following stimulation of the olfactory epithelium by volatile compounds. A normal sense of smell is often undervalued; it plays a vital role in the enjoyment of food and the detection of environmental hazards. Some professions depend heavily on an intact sense of smell, e.g. chefs and wine tasters. Olfactory perception is heavily associated with memory and emotion, due to projections to the limbic system.[1] Olfactory symptoms may be the primary manifestation of serious intracranial pathology. However, this area of otolaryngology remains under-investigated.

Olfactory disorders affect 2 000 000 per annum in the USA; the UK incidence is poorly documented. Men perform less well in olfactory testing than women, and olfactory sensitivity deteriorates with age.[2]

Olfactory disorders may manifest as hyposmia or anosmia (i.e. reduced or absent sense of smell, respectively) or as distorted olfaction (i.e. parosmia, a distorted quality of a perceived odorant; phantosmia, a perceived smell in the absence of an olfactory stimulant; or cacosmia, perception of an unpleasant smell). Analogies are drawn with causes of hearing loss.[3] 'Conductive' disorders result from odorant molecules failing to access the olfactory mucosa (e.g. nasal polyps or rhinosinusitis). 'Sensory' losses are caused by damage to the olfactory mucosa (e.g. chemical exposure, viruses or neoplasms). 'Neural' causes result from defects in the peripheral or central neural pathways (e.g. head injury). Iatrogenic causes are important, as it has been shown that olfactory mucosa extends below the anterior middle turbinate, more anteriorly and inferiorly than originally thought.[4] Up to 22 per cent of olfactory disorders are idiopathic.[5]

There are areas in which the evidence base for the management of olfactory disorders remains undetermined. A survey carried out by the authors on the management strategies of consultant and associate specialist otolaryngologists demonstrated wide variation in clinical practice (see Appendix 1). The use of radiological investigations and formal chemosensory smell testing varied greatly between clinicians. There was little consistent information provided to patients regarding the prognosis for olfactory dysfunction following head injury, chronic rhinosinusitis or upper respiratory infections. It should be noted

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that there was a relatively poor response rate to the questionnaire (266 of 590 questionnaires returned, a 45 per cent response rate). This may reflect the fact that olfactory disorders are an often neglected aspect of otolaryngology.

In view of such variation in clinical practice, we aimed to provide an evidence-based review of the diagnosis, management and prognosis of various olfactory disorders.

Methods

A literature search was performed, searching Medline, Embase and Cochrane databases from 1966 to the present, using the keywords 'olfaction', 'anosmia', 'hyposmia', 'dysosmia', 'diagnosis', 'imaging' and 'prognosis'. Articles were reviewed and the selection limited to English articles on human subjects. Descriptive comparisons have been made and the findings summarised.

Literature review

Clinical examination

Evaluation of the anosmic patient should always involve clinical assessment. The duration, speed of onset and pattern of olfactory disturbance should be determined (e.g. progressive, fluctuant). A thorough clinical history should be carried out, including presence of associated nasal symptoms, taste disturbance and allergy. A history of head injury is relevant, and details of the nature of the injury should be elicited, particularly regarding loss of consciousness, direction of impact and radiological findings.[6–8] A full medical history should be taken, including neurological, psychiatric and metabolic disorders (Tables I and II).[9–11] Occupational history may reveal exposure to noxious chemicals (e.g. cadmium and benzene), and a smoking history is important.[12] Iatrogenic causes must be considered, including medication, neurosurgical intervention, radiotherapy and previous nasal surgery. Family history should be elicited. Some medications are implicated in altered smell and taste, including anticonvulsants and antihypertensives (Table III).[13]

TABLE I

CAUSES OF OLFACTORY LOSS

Aetiology	Patients (%)
Head injury	19
Post-URTI	17
Nasal or sinus disease	16
Idiopathic (nasal)	17
Toxic exposure (nasal)	5
Multiple	5
Congenital	2
Age	1
Idiopathic (oral)	9
Miscellaneous	6
Toxic exposure (oral)	1

Reprinted with permission.[9] URTI = upper respiratory tract infection

TABLE II

MEDICAL DISEASES CAUSING OLFACTORY DYSFUNCTION

Category	Disease
Neurological	Alzheimer's disease
	Down syndrome
	Epilepsy
	Multiple sclerosis
	Parkinson's disease
Congenital	Kallman syndrome
	Choanal atresia
Nutritional & metabolic	Chronic renal failure
	Liver disease
	Vitamin B12 deficiency
Endocrine	Diabetes
	Adrenal cortex insufficiency
	Hypothyroidism
	Cushing's disease
Trauma	Head injury
	Laryngectomy
Inflammatory	Rhinosinusitis or nasal polyposis
	Sarcoid
	Wegener's disease
Neoplastic	Olfactory neuroblastomas
	Anterior skull base tumours
Degenerative	Age
Infective	Acute viral hepatitis
	HIV
	Influenza-like
Other	Adenoid hypertrophy
	Familial
	Psychiatric

Reprinted[10] from adapted material,[11] with permission. HIV = human immunodeficiency virus

The patient's general habitus may reveal clues as to the nature of the olfactory disorder. Congenital disorders of smell, including isolated absence or hypoplasia of the olfactory bulbs, are associated with Kallmann syndrome, Turner syndrome and premature baldness.[14] Nasendoscopy may show evidence of rhinosinusitis and polyposis, or reveal no abnormality. Cranial nerve examination should be included to assess for underlying neurological causes.

TABLE III

MEDICATION CAUSING OLFACTORY DYSFUNCTION

Class	Drug
Local anaesthetic	Cocaine hydrochloride
Antihypertensive	Nifedipine
Antimicrobial	Streptomycin
	Amphotericin B
Antithyroid	Carbimazole
	Thiouracil
Opiate	Codeine
	Morphine
Antidepressant	Amitryptilline
Radiation therapy	To head
Sympathomimetic	Amphetamines
Vasodilator	Diltiazem
Amoebicide	Metronidazole
	Nidazole
Immunosuppressant	Methotrexate
	Azathioprine
Antirheumatic	Gold
	Colchicine
	Allopurinol

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Olfactory testing

Cain describes three criteria necessary to maximise odour recognition in olfactory testing.[15] Odours must be familiar to the patient, with a longstanding association between the odour and its name, and help should be given to recall the name. Reliability is improved using both threshold testing and odour discrimination assessment. 'Forced-choice' procedures reduce response bias. Patients scoring less than chance are likely to be malingering, as are those who fail to identify trigeminal nerve stimulants such as ammonia or 4 per cent butanol.

The University of Pennsylvania smell identification test (UPSIT) system was most commonly used by UK respondents to our questionnaire. This is a forced choice test, with 40 microencapsulated odours, acting as a 'scratch and sniff' test. This test can indicate a level of olfactory function (i.e. mild to total anosmia) and has a score ranking for age and gender.[16] However, the UPSIT system has not been validated in a UK population, relies on suprathreshold testing and is relatively expensive. The Cross-Cultural Smell Identification test is a12-item test based on the UPSIT, which can be carried out in five minutes and is self-administered.[17] 'Sniffin' Sticks' is a test of olfactory function based on felt-tip pens and assesses odour threshold, discrimination and identification.[18] Doty *et al.* evaluated 10 different olfactory tests, noting that there was considerable variation in their reliability, related to the length of the tests. They suggested that the results from different testing methods should not be compared, as variations may be a result of differing reliabilities rather than reflecting clinical findings.[19]

Currently, the only test validated in a UK population is the Combined Olfactory Score.[20] The suprathreshold test consists of nine odours and ammonia, while the threshold test uses dilutions of 1-butanol from 0.00061 per cent to 4 per cent in nine steps. The scores are combined and the average of the two is the final score. The test also allows differentiation between severities of olfactory dysfunction.

As well as a diagnostic and quantitative role, formal olfactory testing allows monitoring of progression or resolution of dysosmia, particularly following surgical or other therapeutic intervention.

Radiological evaluation of olfactory dysfunction

Imaging may be required in the evaluation of olfactory disorders. Computerised tomography is the most appropriate technique for patients with sinonasal disease, regarding surgical planning. However, magnetic resonance imaging is more useful for diagnosis of olfactory apparatus abnormalities and parenchymal disease, particularly in congenital disease. Absent olfactory bulbs and hypoplastic olfactory sulci are particularly noted in Kallmann syndrome, along with loss of temporal and/or frontal lobe volume.[21,22] Decreased volume of the olfactory bulbs is noted with increasing age.

Patients with olfactory groove or frontal lobe meningiomas are likely to present with hyposmia.[23] These may reach a significant size (>4 cm) before presentation, due to a gradual deterioration in olfactory function and preservation of unilateral olfactory function.[23] Welge-Leussen *et al.* suggest that all patients with lateralised dysosmia should undergo radiological evaluation, after noting that 50 per cent of patients with olfactory meningiomas had unilateral dysosmia on formal testing. Recovery of olfaction following intracranial surgery is not well documented, although Ishimaru *et al.* report improved olfactory function in a patient following decompression of a right frontal lobe meningioma.[24]

Accurate diagnosis of skull fracture site and associated parenchymal injuries may allow prediction of the likelihood of recovery of smell.[7] A positive correlation has been shown between number of plaques and olfactory function in patients with multiple sclerosis. Functional assessment of hyposmia with single photon emission computed tomography (SPECT) imaging demonstrates reduced frontal blood flow in patients with schizophrenia.[22]

Although multiple pathologies can be demonstrated on neuroradiological assessment, there are no guidelines regarding the indications for imaging, particularly relating to the sensitivity and specificity of imaging techniques. In our survey, 81 diagnoses of anterior cranial fossa tumours were made by 51 respondents, one surgeon reporting four cases alone. However, a number of respondents commented that they had never seen a single case, in a long established career. Busaba studied 28 patients with anosmia and negative endoscopy results, finding that imaging did not add any further information and concluding that it should not be requested routinely.[25] This appears to be the only study evaluating this aspect of radiological assessment. However, it was a small retrospective, unblinded study of 20 patients and the conclusions should be interpreted with caution.

Olfactory dysfunction following head injury

Head injuries account for 18 per cent of olfactory disturbances.[5] Olfactory insult results from damage to nasal mucosa, shearing of olfactory fibres due to cribriform plate fracture, and oedema of the olfactory tracts and bulbs. Damage to the peripheral olfactory apparatus results in anosmia, whereas central olfactory damage manifests as an inability to discriminate odours.[26,27] The anterior temporal lobes and orbitofrontal poles are most vulnerable to damage, where reduced orbitofrontal perfusion is demonstrated on SPECT.[28,29] The severity of post-traumatic olfactory impairment is more pronounced, with less chance of recovery than in cases of infection or chronic rhinosinusitis.

Potential predictive factors may allow identification of the likelihood of recovery. Green *et al.* retrospectively analysed 367 patients with post-traumatic olfactory disorders, finding a sharp decrease in olfactory function proportional to injury severity.[6] Patients with a Glasgow Coma Scale score of less than 13 at presentation, post-traumatic amnesia and radiological abnormalities were markedly less likely to recover their sense of smell. Ogawa and Rutka noted a similar relationship, with a reduced chance

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of smell recovery following loss of consciousness of more than one hour and occipital, frontal and skull base fractures.[7] Callahan and Hinkebein noted that 40 per cent of such patients suffered an olfactory deficit, which only manifested on formal testing.[8]

Recovery of a normal sense of smell following a head injury is unlikely, although improvement can occur over a longer time period that previously realised. Recovery has been noted up to 18 months post-injury, whereas Duncan and Seiden demonstrated improved olfactory scoring up to five years post-injury, with 35 per cent of patients improving in total.[28,30] Doty *et al.* evaluated 268 patients between one and 13 years following a head injury;[31] no patient with post-traumatic anosmia returned to normosmia, but patients' olfaction improved over time in 36 per cent, particularly in those with parosmia (dropping from 41 to 15 per cent over eight years). Recovery, again, was proportional to severity of head injury.

Olfactory dysfunction following upper respiratory tract infection

Temporary anosmia can occur with an upper respiratory tract infection, when oedema prevents odorant molecules reaching the olfactory cleft. Viral upper respiratory tract infection accounts for 20–30 per cent of identified cases of olfactory loss,[32] typically caused by the parainfluenza 3 virus.[33] In a small percentage, olfaction remains permanently distorted, particularly in women (70–80 per cent) and in those aged 40–60 years.[34] This is partly due to cumulative degeneration of the olfactory apparatus with age.[16] It is thought that viral infections cause a reduction in the number of olfactory receptors, with replacement by respiratory epithelium.[32,35] However, stem cells with the potential for regeneration may persist.[36] The reported prognosis for upper respiratory tract infection induced hyposmia varies. Hummel found that the majority of patients recovered function within six months, whereas Cullen and Leopold noted that some patients continued to recover up to three years following the initial olfactory insult.[36,37] Duncan and Seiden found that 19 of 21 patients with viral-induced hyposmia had markedly improved UPSIT scores at three years, although Doty and Mishra documented minimal recovery in such patients.[30,38]

Olfactory dysfunction after rhinosinusitis

If olfactory dysfunction secondary to nasal disease is due to odorant molecules failing to reach the olfactory apparatus, it seems logical that treating mucosal oedema and polyposis would result in a symptomatic improvement.

The effectiveness of surgical intervention in treating hyposmia secondary to chronic rhinosinusitis is open to debate (Table IV). A small study by Damm *et al.* found a significant correlation between nasal airflow and odour identification in patients with chronic rhinosinusitis, suggesting that surgery to improve nasal airflow and eliminate mucosal disease would be helpful.[39] A non-blinded, retrospective study by Iro *et al.* reported the success of endoscopic sinus surgery (ESS) in reducing nasal symptoms, including anosmia.[40] They reported a 92 per cent success rate over three years, although this was based purely on subjective data. Rowe-Jones and Mackay collected prospective data on 115 patients, evaluating subjective symptoms and olfactory detection thresholds, prior to and six weeks following ESS.[41] All parameters significantly improved, including nasal volume on acoustic rhinometry. Improvement in olfactory scores was proportional to the increase in nasal volume. However, Kimmelman found no improvement in olfaction scores in patients undergoing nasal polypectomy.[42]

Landis *et al.* note that, although 76 per cent of post-ESS patients improved their olfactory function, 13 per cent demonstrated a deterioration on formal testing.[43] In an extensive review of the literature, Deems *et al.* comment that both surgical and medical interventions do not result in return to normosmia.[5] Similarly, the relationship between airway patency and olfactory function is questionable.[38] This could be explained by the findings of Lee *et al.*, who noted lower levels of olfactory epithelium and replacement with normal respiratory mucosa in patients with chronic rhinosinusitis.[44] Inflammatory changes within the olfactory mucosa may account for hyposmia, independent of airflow alteration.[45] The likelihood of recovery of olfaction in patients with chronic rhinosinusitis or polyposis seems to be time-dependent, with prolonged disease resulting in degeneration of olfactory mucosa and persistent olfactory dysfunction.

Pharmacological therapy for olfactory disorders

Clinical experience often suggests that a patient's sense of smell may return following treatment with corticosteroids. Hotchkiss described a subjective improvement in olfaction in patients with chronic rhinosinusitis treated with 70 mg prednisolone.[46] However, no formal olfaction testing was carried out as confirmation. Jafek *et al.* describe two patients in whom high dose steroids temporarily restored a normal sense of smell, prior to surgery for nasal disease, followed by long-term, low dose nasal steroids.[47] These authors felt that a trial of steroids was worthwhile in patients with allergic rhinitis, nasal polyposis and anosmia (Table IV). In the study by Tos *et al.*, steroid nasal sprays were given to hyposmic patients with rhinosinusitis.[48] Olfaction scores improved but significant persistent hyposmia was noted in a number of subjects. Similarly, Mott *et al.* noted an improvement to the mid-hyposmic range in patients treated with flusenolide, although this was an unblinded trial without placebo control.[49]

Golding-Wood *et al.* performed UPSIT scoring on patients with perennial rhinitis treated with intranasal betamethasone.[50] Patients with scores in the hyposmic range improved but failed to reach normosmia. There was no improvement in anosmic patients. Blomqvist *et al.* describe a significant improvement in olfactory thresholds in 48 consecutive patients with anosmia or hyposmia receiving a 10-day course of oral prednisolone (40 mg for 3 days, followed by a daily reduction of 5 mg) and fluticasone nasal spray.[51] However, the improvement in olfactory scores was maintained equally well whether patients continued with nasal

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TABLE IV

LEVELS OF EVIDENCE FOR MEDICAL AND SURGICAL MANAGEMENT OF OLFACTORY DISORDERS

Reference	Description	*n*	Level of evidence	Conclusions
Damm *et al.* 2000[39]	Prospective cohort study	30	2b	Significant correlation between nasal airflow & odour identification & threshold screening
Iro *et al.* 2004[40]	Retrospective cohort study	208	3	41% of patients after complete ethmoidectomy & 32% of patients after pansinus surgery described complete resolution of all nasal symptoms, including anosmia
Rowe-Jones & Mackay 1997[41]	Prospective cohort study of consecutive patients	115	2b	Symptom scores, olfaction scores & nasal volume all significantly improved following endoscopic sinus surgery Increase in nasal volume proportional to symptom scores
Kimmelman 1994[42]	Prospective cohort study	93	2b	93 patients underwent various nasal procedures incl ethmoidectomy, polypectomy & rhinoplasty Post-operatively, 66% had improved or unchanged UPSIT scores, 34% had worsened scores One patient became anosmic
Landis *et al.* 2004[43]	Prospective cohort study	203	2b	Patients underwent endoscopic sinus surgery with pre- and post-operative olfactory testing Olfactory function improved in 76% & worsened in 13% (but with no spontaneous complaints)
Hotchkiss 1956[51]	Prospective cohort study	30	4	Improved sense of smell after short course of high-dose oral steroids No formal olfactory testing
Jafek *et al.* 2002[47]	Case series	2	4	Normal sense of smell restored in 2 patients receiving high-dose steroids prior to nasal surgery
Tos *et al.* 1998[48]	Prospective, randomised, placebo-controlled study	138	2b	Nasal symptom scores (incl hyposmia) significantly more reduced with intranasal budesonide compared with placebo Aqueous formula more effective than powdered
Mott *et al.* 1997[49]	Prospective, non-randomised, non-blinded study No placebo group	39	4	Improvement in olfactory scores after 8 weeks' nasal flusinolide in patients with anosmia due to sinonasal disease Multiple confounding factors due to concurrent medication
Golding-Wood *et al.* 1996[50]	Prospective, interventional, non-blinded cohort study	15	2b	Patients with perennial rhinitis & hyposmia treated with 6 weeks' intranasal betamethasone Post-treatment UPSIT & VAS scores significantly improved compared with pre-treatment scores
Blomqvist *et al.* 2003[51]	Randomised, double-blind, placebo-controlled study of consecutive patients	48	1b	Initial 10-day treatment with prednisolone, 40 mg for 3 days then decreasing dose, with nasal fluticasone Significant improvement in olfactory scores Randomised to receive fluticasone, placebo or control Scores maintained in steroid & placebo groups
Heilmann *et al.* 2004[52]	Prospective, non-blinded, non-randomised study	92	2b	37 patients with olfactory loss given mometasone nasal spray for 3 months, compared with 55 patients receiving 40 mg oral prednisolone decreasing over 3 weeks Significant improvement in olfactory function tests with prednisolone (not seen with topical mometasone) Improvement most marked in idiopathic and presumed viral olfactory loss

Continued

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TABLE IV *Continued*

Reference	Description	*n*	Level of evidence	Conclusions
Stevens 2001[53]	Prospective, non-blinded, cohort study	24	2b	Patients with sinonasal disease who remained anosmic after surgery were treated with nasal steroids followed by oral corticosteroids No improvement in olfactory scores with topical therapy, but normosmia restored in most patients with oral therapy
Aiba *et al.* 1998[54]	Prospective, non-randomised, non-blinded study No placebo group	426	4	Patients given topical corticosteroids & vit B12, zinc sulphate, or both treatments Significant improvement in post-traumatic anosmia with zinc No improvement in post-viral or anosmia of unknown aetiology Zinc levels only measured in zinc treatment group
Henkin *et al.* 1976[55]	Randomised, double-blinded, placebo-controlled, crossover study	106	1b	Zinc sulphate equivalent to placebo in management of taste and smell disorders
Hummel *et al.* 2002[56]	Prospective, non-randomised, non-blinded study No placebo group		4	23 patients given 600 mg/day oral α-lipoeic acid 35% showed 'remarkable' increase in olfactory discrimination & threshold testing
Quint *et al.* 2002[57]	Prospective, non-randomised, non-blinded, controlled study	77	4	51 patients given 120 mg caroverine daily, compared with 400 mg zinc sulphate as control Significant improvement in odour thresholds & identification in treatment group

UPSIT = University of Pennsylvania smell identification test; incl = including; VAS = visual analogue scale; vit = vitamin

fluticasone or placebo, when compared with controls. These authors felt that the improvement was probably due to a reduction in mucosal oedema, even though a conductive olfactory loss may not be apparent. Heilmann *et al.* were unable to demonstrate an improvement in olfaction following use of topical mometasone nasal spray in patients with sinonasal disease or upper respiratory tract infection related hyposmia, but they noted a significant increase in olfactory scoring following treatment with systemic corticosteroids.[52] Similar findings were noted by Stevens; patients who remained anosmic following ESS or polypectomy were found to respond well to oral corticosteroids but not to topical application.[53]

The evidence suggests that a trial of oral corticosteroids may be useful, although there is little information on the required dose or length of treatment. However, long-term use of corticosteroids is likely to have undesirable side effects which outweigh the handicap of hyposmia.

A small number of studies have assessed substances other than steroids for the treatment of olfaction disorders. Zinc deficiency has been postulated as a contributing factor in hyposmia. Henkin *et al.* found no therapeutic effect in a randomised, placebo-controlled trial of zinc sulphate in hyposmic patients; however, Aiba *et al.* described significant improvement in patients with post-traumatic hyposmia, following zinc replacement.[54,55] This appears to be based on retrospective, unblinded evidence. Jafek *et al.* reported that topical application of zinc gluconate resulted in permanent destruction of the olfactory epithelium.[58]

A significant improvement in olfaction was described by Hummel *et al.* in patients treated with alpha-lipoeic acid following upper respiratory tract infection.[56] This trial involved 23 patients and was unblinded, with no crossover limb, and should therefore be interpreted with caution. Finally, Quint *et al.* evaluated the efficacy of caroverine in the management of non-conductive olfactory disorders.[57] Caroverine is thought to act by preventing glutamatergic neurotoxicity and was compared to the effects of oral zinc sulphate as a control. Quint *et al.* found a significant improvement in odour thresholds and identification in the treatment group, with no improvement in the zinc sulphate group. However, the study does not appear to have been blinded or randomised.

The evidence therefore suggests that, other than corticosteroids, there are no other successful pharmacological treatments for anosmia.

Conclusion

Formal olfactory testing should include both threshold testing and odour identification. 'Forced choice' procedures can detect malingering. Modern tests are robust and quick to perform. Olfactory testing has a role in the diagnosis and monitoring of olfactory disorders, but the same test should be used consistently in each individual.

Computed tomography imaging is most appropriate for planning surgery for sinonasal disease, while magnetic resonance imaging evaluates the olfactory apparatus and parenchyma more accurately. It is

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suggested that all patients with unilateral dysosmia should undergo radiological evaluation. There are no established guidelines regarding indications for imaging, or sensitivity or specificity evidence.

Following head injury, poor prognostic factors for the recovery of olfaction include injury severity, reduced consciousness, post-traumatic amnesia and radiological abnormalities. The likelihood of recovery is proportional to the severity of injury, with anosmic patients being unlikely to recover a normal sense of smell. Recovery in olfactory scoring has been reported up to five years after injury.

Olfaction may remain permanently distorted following upper respiratory tract infection, due to destruction of olfactory receptors. This is more likely in women and older patients. Recovery has been reported up to three years following such an olfactory insult.

Recovery in olfaction following chronic rhinosinusitis appears to be time-dependent. Permanent changes in the olfactory mucosa can result from prolonged disease. Hyposmic patients may show improved olfactory scoring with topical and oral corticosteroids but are unlikely to return to normosmia.

There does not appear to be any other successful form of pharmacological treatment for olfactory disorders.

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Appendix 1. Management of olfactory disorders: results of survey

266/590 questionnaires returned

259/266 clinicians evaluated patients with olfactory disorders

104 clinicians evaluated medicolegal cases of olfactory dysfunction

Investigations

Investigation	*n* (%)
Rigid nasendoscopy	246/259 (94)
CT scan	188/259 (72.5)
MRI scan	95/259 (36.6)
Other investigations*	14 (5.4)

*Allergy testing, zinc levels, provocation tests, blood tests (not otherwise specified).

Chemosensory smell test used?*	*n* (%)
No	142/259 (54.8)
Yes	117/259 (45.2)
– Rarely	47/117 (40.1)
– Usually	56/117 (47.9)
– Always	14/117 (12.0)

*Chemosensory smell tests used in 77/104 (74%) of medicolegal cases.

Olfactory test used	*n* (%)
UPSIT	56/117 (47.8)
Zurich smell test	12/117 (10.3)
Sniffin' Sticks	11/117 (9.4)
Other*	38/117 (32.5)

*CCSIT, alcohol sniff test, Combined Olfactory Score, Nez du Vin, or locally produced smell bottles. UPSIT = University of Pennsylvania Smell Identification Test

Clinical advice given

Olfaction prognosis after head injury	*n* (%)
Sense of smell will not return	116/259 (44.7)
Recovery unlikely	115/259 (44.3)
Time limit given*, beyond which recovery unlikely	28/259 (10)

*Range 3–18 months.

Olfaction prognosis after URTI	*n* (%)
Sense of smell will not return	21/259 (8.1)
Recovery unlikely	222/259 (85.7)
Time limit given*, beyond which recovery unlikely	16/259 (6.2)

*Range 3–36 months. URTI = upper respiratory tract infection

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