Smell and taste disorders; recent management approaches

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Abstract: Olfactory or gustatory loss should be taken seriously, as it makes a person more liable to risk for toxic exposures, such as gas leaks, smoke, and rotting food, and it also decreases the enjoyment of some of life's pleasures, such as the fragrance of flowers or the taste of good food or fine wine. In many patients, the loss follows a viral upper respiratory tract infection, and the only real treatment is psychological reassurance of the patients that the problem may resolve if the damaged sensory cells regenerate. Olfactory or gustatory loss has many other causes that require a careful investigation and appropriate treatment. This article reviews the proper steps to take when investigating and treating chemosensory difficulties.

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Introduction

The chemical senses of smell and taste contribute to quality of life and environmental appreciation. Not only do these senses guard against toxic and dangerous stimuli, but the ability to smell and taste contributes to the finer qualities of life ***(Allis and Leopold, 2012).***

Significant advances have been made in the development and application of easy-to-use and reliable clinical tests of olfactory function. It is clearly is no longer tenable to simply ask a patient whether a few odorants placed under the nose can be identified, since this approach can result in misleading conclusions, as it is not quantifiable, lacks reliability, has no normative referent, and is easily faked by malingerers. ***(Doty, 2015).*** By contrast with the evaluation of olfactory function, which has been standardized for almost two decades, the clinical assessment of gustatory function with psychophysical and objective testing is still in its infancy ***(Schuster et al., 2009).***

Olfactory disorders

Anatomy of the olfactory nervous system:

The olfactory nervous system consists of three main stations: the olfactory epithelium, olfactory bulb and olfactory cortex ***(Fukushima et al., 2002).*** The human olfactory epithelium is a pseudostratifiedcolumnar epithelium that rests on a highly cellular lamina propria that contains the Bowman's glands and extends about 150 um down to the underlying bone or cartilage ***(Lucero 2013).*** The oval olfactory bulbs are comprised of six concentric layers: namely, the glomerular nerve cell layer, the external plexiform layer, the mitral and tufted cell layer, the internal plexiform layer, and the granule cell layer ***(Shipley and Reyes, 1991).*** The LOT is a major central olfactory pathway connecting the olfactory bulb to the olfactory cortex and is made up of myelinated fiber bundles of mitral cells ***(Fukushima et al., 2002).***

Physiology of the olfactory nervous system:

Olfactory transduction mechanisms:

The olfactory sensory neurons are bipolar neurons with a single dendrite that terminates in a knob from which 10-20 fine cilia originate. These cilia are immersed in the nasal mucus and are the sites where the entire transduction mechanism occurs. The binding of odorants to odorant receptors in the cilia causes, via G protein activation of adenylyl cyclase, the production of a cyclic nucleotide, cAMP, which directly opens ionic channels in the plasma membrane. An inward transduction current is carried by Na^+ and Ca^{++} ions. Olfactory sensory neurons maintain an unusually high intracellular concentration of Cl^- ions, and the increase in the internal concentration of Ca^{++} causes the opening of Ca^{++} activated Cl^- channels that produce an efflux of Cl^- from the cilia, contributing to the olfactory neuron depolarization. The depolarization spreads passively to the dendrite and soma of the olfactory neuron, triggering action potentials that are conducted along the axon to the olfactory bulb ***(Firestein, 2001).***

Classification of olfactory disorders:

Olfactory disorders may be quantitative or qualitative disorders. Quantitative disorders include anosmia (no smell sensation), hyposmia (decreased

sensitivity to smell), partial anosmia (ability to receive some but not all odorants), hyperosmia (increased sensation of smell).

Qualitative disorders (dysosmias) are divided in parosmia and phantosmia. Parosmia or troposmia describes distortion of the perceived odorant. Phantosmia (lasting longer than a few minutes) and olfactory hallucination (lasting only a few seconds) describes the perception of an odor (usually unpleasant) when there is no odorant stimulus present. ***(Rombaux et al., 2005).***

Etiology of smell disorders:

❖ ***Conductive defects*:**

1-Inflammatory processes: including allergic, acute, or toxic (eg, cocaine use). Chronic rhinosinusitis**.**

2-Masses: nasal polyps (most common), inverting papilloma, or any nasal tumor.

3- Developmental: (eg, encephaloceles) also may cause obstruction.

4- Patients with laryngectomies or tracheotomies: experience hyposmia because of a reduced or absent nasal airflow. ***(Leopold et al., 2016)***.

❖ ***Central/ sensorineural defects***

1- Infectious and inflammatory processes: viral infections, sarcoidosis, Wegener granulomatosis, multiple sclerosis and chronic ***(Leopold et al., 2016)***.

2- Head trauma, brain surgery, or subarachnoid hemorrhage *(Martin et al., 2009).*

3- Aging: Sense of smell decreases with age ***(Bhatnagaret al., 1987).***

4- Congenital syndromes: Kallmann syndrome is one type of congenital smell loss and is due to failed olfactory structure ontogenesis.

5- Endocrine disturbances: (eg, hypothyroidism) may affect olfactory function ***(Leopold et al., 2016)***.

6- Toxicity of systemic or inhaled drugs: (eg, aminoglycosides, formaldehyde) ***(Tuccori et al., 2011).***

7- Smoking *(Schriever et al., 2013).*

8- Various neuropsychiatric disorders: (eg depression, schizophrenia, seasonal affective disorder) ***(Negoias et al., 2010).***

9- Anterior skull base occupying lesions *(Mishra and Doty, 2002).*

Diagnosis of olfactory disorders

Assessment of a patient with smell dysfunction should include detailed history, physical examination including otorhinolaryngological and neurological assessment and investigations ***(Mishra and Doty, 2002).***

❖ ***Physical examination:***

Complete otolaryngological and neurological examination is very important including evaluation of cranial nerves and orbital contents (to direct attention to lesions of the skull base), as well as general evaluation of the upper respiratory tract and ears ***(Mishra and Doty, 2002).***

❖ **Investigations:**

(1) Subjective measures (psychphysical tests):

I- Odor identification tests in the USA:

1-The University of Pennsylvania Smell Identification Test (UPSIT)*:* is a multiple-forced-choice odor identification test. Normative data for the UPSIT include a score on a scale of 0–40 to evaluate olfactory dysfunction. This test has several unique features, including amenability to self-administration and a means for detecting malingering ***(Doty and Latin, 2015).***

2- Other odor identification tests of utility in USA: are the Connecticut Chemosensory Clinical Research Center (CCCRC) Test ***(Cain WS et al., 1988)*** and the Odorant Confusion Matrix ***(Kurtz DB et al., 2001).***

II- Odour identification tests in Europe:

1-The Sniffin' Sticks (SS) test: The SS test comprises a first level (**Sniffin' Sticks Screening Test**) and a second level (**Sniffin' Sticks Extended Test**). The Extended test is composed by 112 sticks to evaluate odour threshold, discrimination and identification ***(Eibenstein et al., 2005)***.

2- **The Scandinavian odour-identification test (SOIT)**: The advantages of this test are the low cost and the easy and fast administration (10–15 min) ***(Nordin et al., 1998).***

3- Other tests in Europe: the Biolfa olfactory test ***(Frank et al., 2003)*** and the Sniff Magnitude test (SMT) ***(Bensafi et al., 2003)***.

III- Olfactory tests in other countries:

The odour stick identification test (OSIT) OSIT is a new type of smell identification test recently developed by Japanese. ***(Kobayashi, 2005).*** **The 12-item Modular Smell Identification Test (MODSIT)** is a smell screening test also used in China ***(Liu et al., 1995)***.

IV- Olfactory screening tests:

1-The Cross-Cultural Smell Identification Test (CCSIT) and the Pocket Smell Test (PST): ***(Doty et al., 1996).***

2- The San Diego Odor Identification test ***(Murphy et al., 1994).***

3- The Sniffin' Sticks Screening (SSS) test ***(Hummel et al., 2001).***

4- The Smell Diskettes test ***(Simmen et al 1999).***

(2) Objective measures:

(A) Psychophysiologic measures:

i- The electro-olfactogram (EOG) or the evoked potential of the olfactory epithelium: A more objective way to analyze olfactory disturbances is to use chemosensory event-related potentials ***(Murphy et al., 2000).***

ii- Chemosensory event-related potential (CSERP) or the olfactory event-related potential (OERP): The OERP reflects the activity of both the peripheral and the central elements of the olfactory system ***(Wang et al., 2003).***

(B) Imaging techniques:

1- Computed tomography (CT) ***(Li et al., 1995).***

2- Magnetic reasonance imaging (MRI) ***(Yousem et al., 1993).***

3- Radionuclide imaging (Functional imaging) (Jagust and Eberling, 1991).

(C) Volumetric evaluation of the bulbusolfactorius (BO) ***(Frasnelli et al., 2010).***

(D) Biopsies from the olfactory regions ***(Witt et al., 2009).***

Treatment of smell disorders:

1- Non-surgical treatment of smell disorders:

Corticoids are used as topical and systemic treatments of sinunasal disease ***(Jafek BW et al., 1987).*** Other therapeutic lines may include the use of antihistaminics ***(Settipene, 1994)***, antileukotrienes ***(Parnes and Chuma, 2000)*** antifungal therapy ***(Ponikau et al., 2002),*** saline lavage as a treatment of olfactory loss secondary to SND. Antibiotic therapy should only be started after the bacteria have been identified and tested for resistance to antibiotics ***(Wolfensberger and Hummel, 2002).*** Zinc sulphate may be useful in post-traumatic olfactory loss as it is believed to play an important role in regeneration of olfactory neurons ***(Aiba et al., 1998).*** The use of theophylline has been proposed ***(Levy et al., 1998).*** In a further study, **Hummel et al** showed that alpha lipoic acid also had a positive effect for patients with post-infection smell disorders ***(Hummel et al., 2002).*** **Quint et al**, reported significant improvements in smell after a 4 week course of caroverine ***(Quint et al., 2002).*** Vitamin A at a dose of 10,000 I.E. oral over 4 weeks ***(Vent J et al., 2010)***. There are promising approaches that include the use of acupuncture ***(Vent et al., 2010)*** and the transcranial magnetic stimulation for parosmia and phantosmia ***(Henkin et al., 2011).*** In a more recent study the effectiveness of smell training showed promising results ***(Hummel et al., 2009).***

- **Additional non-surgical treatment for Patients with olfactory distortion (parosmia and phantosmia):**

Patients with olfactory distortion (parosmia and phantosmia), need to be reassured. Most patients will note a gradual decrease in the symptom with time, and this can occur over several years ***(Duncan and seiden, 1995).***

Several medications, including sedatives, antidepressants, anticonvulsants and antiepileptic drugs, have been suggested to treat olfactory distortions ***(Zilstroff, 1966).*** Currently gabapentin is being used by several olfactory centres ***(Leopold, 2002).*** Also a neuroleptic, "Haloperidol" can control hallucinations, thus inhibiting the phantom smells ***(Henkin et al., 2000).*** The topical use of cocaine hydrochloride can temporarily block most distortions by anesthetizing the neurons and is useful in the diagnosis of these individuals ***(Leopold, 2002).***

2- Surgical treatment of olfactory disorders:

(A) Surgical treatment of olfactory loss (anosmia nadhyposmia): Surgical therapy aims at either elimination or reduction of nasal obstruction and removal of inflamed mucosa or polyps ***(Jafk et al., 2002).***

(B) Surgical treatment of olfactory distortions (parosmia and phantosmia):

Neurosurgical approaches using a bifrontal craniotomy to remove the olfactory bulbs or nerves have been reported. ***(Market et al., 1993).*** Streostacticamygdalectomy was tried for the treatment of olfactory hallucinations ***(Chitannondh, 1966).*** To treat a patient's phantosmia and avoid craniotomy, an excision of the olfactory epithelium followed by temporalis fascia grafting as an endoscopic intranasal procedure was tried ***(Leopold et al., 1991).*** A new approach for repairing damage throughout the central nervous system has been the transplantation of olfactory cells harvested from the olfactory mucosa ***(Goldstein et al., 1998).***

Gustatory disorders

Anatomy of the tongue:

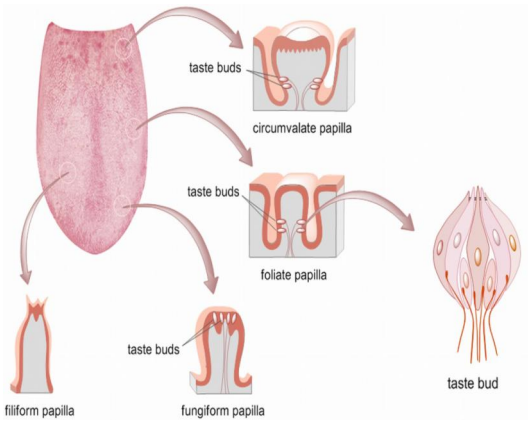

Figure 26: The distribution and types of human lingual papillae. The drawings present the distribution of taste buds in the epithelium of the papillae ***(Eelamand Arlen, 2016).***

From anterior to posterior, the tongue has 3 surfaces: tip, body, and base. The terminal sulcus, or groove, is a V-shaped furrow that separates the body from the base of the tongue. ***(Eelamand Arlen, 2016).*** The surface of the body of the tongue derives its characteristic appearance from the presence of lingual papillae, which are projections of lamina propria

covered with epithelium ***(Smith, 1991).*** The 4 types of lingual papillae are as follows: vallate (circumvallate), foliate, filiform, and fungiform ***(Eelamand Arlen, 2016).***

Physiology of the gustatory system:

Taste transduction mechanisms:

Sweet, umami, and bitter compounds each activate different taste G-protein coupled receptors (GPCRs) that are expressed in discrete sets of receptor cells. Receptor cells that express members of the T2R family of GPCRs sense bitter compounds ***(Chandrashekar et al., 2000).*** Receptor cells expressing the heterodimer taste 1 receptor 2 +taste 1 receptor 3 (T1R2+T1R3) respond to sugars, synthetic sweeteners, and sweet-tasting proteins such as monellin and brazzein ***(Jiang et al., 2004).*** A third class of receptor cells expresses the heterodimeric GPCR, T1R1+T1R3, which responds to umami stimuli, particularly the combination of l-glutamate and guanine monophosphate/inositol monophosphate (GMP/IMP), compounds that accumulate in many foods after hydrolysis of proteins and NTPs ***(Nelson et al., 2002)***.

Classification of taste disorders:

❖ **quantitative measures**, taste disorders can be described as

Ageusia (complete loss of taste), **hypogeusia** (diminished sense of taste), or **hypergeusia** (enhanced gustatory sensitivity).

❖ **Qualitative taste disorders:** taste disorders can be described as

Dysgeusia is a qualitative gustatory disturbance relating to a distorted taste perception or to a persistent taste sensation in the absence of stimulation ***(Brand JG, 2000).*** **Taste phantoms (phantogeusia)** have been reported in patients with epilepsy and schizophrenia ***(Hausser and Bancaud, 1987).***

Etiology of taste disorders:

1- Head trauma: The mechanisms for head-trauma-related taste loss have been even less clearly defined ***(Hummel T et al., 2011).***

2- **Post upper respiratory tract infection:** Destruction or damage of both periphcral olfactory receptors and central olfactory pathways has been proposed as a pathophysiologic mechanism ***(April et al., 1991).***

3- Olfactory loss (perceived taste loss): Most patients who complain of a taste loss suffer from an olfactory deficit ***(Soter et al., 2008).***

4- Medications: Many medications can alter or decrease both taste and smell perception ***(Terah et al., 2012).***

5- Neoplasm: It is well established that a neoplasm, especially in the floor of the mouth or tongue can alter taste. ***(Finelli et al., 2008).***

6- Poor oral hygiene: Odontogenic infections and dental caries also contribute to altered taste sensations ***(Terah et al., 2012).***

7- Chorda tympani injury: If there is a temporal bone tumor, infection, or history of otologic surgery, the chorda may be injured, altering a patient's taste ***(Terah et al., 2012).***

8- Age-related alterations (decrease in bitterness or sour): In the elderly, the taste threshold can be more than twice as high as that of the general population, and drugs and disease states may further decrease taste ***(Schiffman, 1997).***

9- Toxins (smoking): Multiple toxins exist in our environment that may be inhaled, ingested, or absorbed systemically, and are associated with dysgeusia ***(Terah et al., 2012).***

10- Systemic diseases (burning mouth or Sjogrensyndrome): Systemic diseases affect taste more readily than olfaction ***(Suarez and Clark, 2006).***

Diagnosis of taste disorders:

A thorough history and physical examination provide critical information toward the cause of gustatory loss ***(Cullen et al., 1999).***

❖ ***Physical Examination:***

The physical examination should be centered on the head and neck and neurologic systems. A complete physical examination may also be necessary ***(Cullen et al., 1999)***.

❖ ***Modern psychophysics and the assessment of human oral sensation:***

I- Threshold procedures:

(A) Chemical testing of taste disorders:

i- Three-drop method: One of the first standardised tests was described by ***Henkin and Powell GF, (1962)***. It consisted of three consecutive drops applied to the patient's mouth: one with a taste solution and the two others being blanks. The patient's task was to indicate which droplet contained the taste solution.

ii- Taste Tablets and Wafers: In order to create tests for prefabrication and storage, taste tablets and wafers containing basic tastants were used with good test-retest reliability but with the same drawback of whole-mouth stimulation only ***(Ahne et al., 2000).***

iii- The 8-cup technique: The 8-cup technique is a sorting task in which four cups of tastant and four cups of water are presented; threshold is defined as the lowest concentration at which the participant separates the cups into tastant and water groups without error ***(Snyder et al., 2015).***

(B) Electrical testing (Electrogustometry):

Proponents of electrogustometry emphasize its convenience ***(Frank ME and Smith 1991);*** it is portable, avoids the use of chemical solutions, permits regional stimulation of taste bud fields, and provides values that can be compared across individuals, time

points, locations within the mouth, or treatment conditions. Electric taste thresholds show high test-retest reliability and bilateral correspondence ***(Murphy et al., 1994).***

II- Direct Scaling of Suprathreshold Intensity:

1- Magnitude Estimation: subjects provide a number reflecting the perceived intensity of a stimulus; they then give a number twice as large to a stimulus that is twice as intense, a number half as large to a stimulus half as intense, and so on. The size of the numbers is irrelevant; only the ratios among numbers carry meaning ***(Marks, 1974).***

2- Magnitude matching: Magnitude matching addresses the problem of group comparisons by changing the task: Oral sensations cannot be compared directly across PROP taster groups, so subjects rate stimuli of interest relative to a non-oral sensory standard ***(Snyder et al., 2015).***

3- Measuring Oral Sensory Differences: Labeled Scales. (general Labeled magnitude Scales (gLMS)): The Labeled Magnitude Scale (LMS) had been developed specifically to measure oral sensations ***(Green et al., 1993).***

4- Spatial Taste Testing:

The integrity of specific taste nerves is assessed clinically via spatial testing ***(Magendie, 1822).***

❖ ***Objective measurements for assessment of taste function:***

a) Gustatory event-related potentials:

These techniques give consistent results for sour, but show certain weaknesses in investigating the other basic tastes ***(Schuster et al., 2009)***.

b) Imaging:

With the introduction of functional imaging such as **PET** and functional **MRI,** it was possible to establish an insight into gustatory processing in healthy and living humans. These techniques could confirm or reject observations and conclusions drawn from patients with circumscribed anatomical or neurological lesions and taste disorders ***(Ogawa et al., 2005).***

c) Confocal microscopy:

This technique does notdirectly measure gustatory function. However, it does generate a direct vision of the taste organ, which allows for the correlation of morphology with measured taste function ***(Just et al., 2005).***

d) Oral Sensory Anatomy: Videomicroscopy of the Tongue

Multiple reports indicate that differences in taste bud density for human oral sensory variation ***(Prutkin et al., 2000)***

e) More extensive testing with haematological and biochemical investigations are frequently required to discover the various nutritional and endocrine causes of smell and taste disorders ***(Cowart et al., 1997).*** Radiological investigation such as computed tomography is necessary to detect neurological causes for the disorder ***(Boyce and Shone, 2006).***

❖ **Retronasal Olfaction:**

Verification of the specific taste complaint is necessary, since many patients complain of taste loss and yet testing reveals the presence of normal taste. In most of these cases, it is flavor that has been impaired ***(Mann, 2002).***

Treatment of taste disorders

1- Treatment of idiopathic taste disorders:

A treatment course using zinc (140 mg/day for 4 months) may be promising ***(Ross et al., 2008).*** In addition, systemic corticosteroids and vitamin A have been used to treat taste disorders, despite a lack of clinical studies ***(Henkin et al., 1967).*** The same applies for studies involving acupuncture ***(Brandt et al., 2008).***

2- Treatment of trauma or URTI causing smell disorders:

If trauma is the cause, no specific therapy is available, but the condition may improve in time with the regeneration of nerve cells ***(Mann NN, 2002).*** As is the case with smell dysfunctions secondary to URI or head trauma, some proportion of taste dysfunctions with these etiologies ***(Sumner, 1967).***

3- Treatment of smell dysfunction produced by burning mouth syndrome (BMS):

Tricyclic antidepressants (which have analgesic properties) and clonazepam can be helpful ***(Mann, 2002).*** Transcranial magnetic stimulation has also been suggested as treatment ***(Henkin et al., 2011).***

4- Flavour enhancement:

For patients with unpleasant phantom tastes or a lowered taste threshold, reducing the intake of metallic or bitter foods such as meat, coffee or tea and eating more mild flavoured foods such as chicken, dairy and eggs may help with the enjoyment of food; cooling foods before eating may reduce unpleasant flavours and odours ***(Hong et al., 2009).***

5- Cancer and radiotherapy:

In selected cases, modification of cancer therapy may be possible. In head and neck cancer patients, radiation fields that avoid exposure to critical sites for taste can be chosen when not detrimental to tumor management ***(Lin et al., 2003).*** Radioprotectors such as amifostine can also contribute to taste maintenance ***(Wasserman et al., 2005).*** Use of topical antiseptics (e.g. chlorhexidinegluconate), or systemic antimicrobials (e.g. metronidazole) may be considered in necrotic tumors ***(Epstein and Barasch, 2008).***

6- Specific conditions:

Specific conditions related to taste dysfunction such as hyposalivation, poor oral hygiene, use of tobacco products and/or alcohol has relatively simple solutions. Discontinuing the etiologic habit, chewing

sugarless gum or candy for taste and salivary stimulation or prescribing sialogogue can be used for individuals with residual salivary gland function. Chewing gum or candy may also cover unpleasant taste and provide symptomatic relief ***(Peregrin, 2006).***

7- Alpha lipoic acid:

It has also been suggested for treating idiopathic dysgeusia ***(Femiano et al., 2002).***

8- Ice cube:

Fujiyama and colleagues reported improvement in salt, sweet, sour and bitter tastes in an 80 years old woman who started holding an ice cube in her mouth before meals ***(Fujiyama et al., 2010).***

9- Mouth wash:

There are studies showing that mouth wash containing chlorhexidine would be effective in some cases of dysgeusia for salty or sour ***(Mattes and Kare, 1986).***

10- Hypothyroidism:

In patients with hypothyroidism, it has been observed that administration of thyroxine substitutes led to normal reappearance of taste sensation ***(Grushka et al., 1998).***

11- Gastric reflux:

Acid pump inhibitors are effective ***(Mann, 2002).***

12- Chorda tympani nerve section

For patients with dysgeusia that is severe and significantly interferes with quality of life ***(Cullen and Leopold, 1999).***

13- Repeat taste testing:

It gives the patients a rough estimate of prognosis for recovery. If the patient's score is significantly improved, there is a chance for further recovery. If the score is unchanged or significantly worse, the likelihood of recovery may be less ***(Cullen and Leopold, 1999).***

4. Discussion

In recent years, both psychophysical and electrophysiological tests have been developed to quantify olfactory function. Additionally, modern structural and functional imaging procedures have been applied to better define the underpinnings of functional losses, such as damage to or the lack of olfactory bulbs and tracts ***(Gottfried, 2015).*** However, olfactory tests vary in terms of sensitivity and practicality, ranging from brief tests of odor identification to sophisticated olfactometers capable of quantifying odor-induced changes in electrical activity at the level of the olfactory epithelium (the electro-olfactogram; EOG) and cortex (odor event-related potentials; OERPs). Psychophysical tests are more practical and less costly than electrophysiological tests, making them much more popular, particularly in light of technical issues with electrophysiological testing. For example, the EOG cannot be reliably measured in all patients, given the intolerance of some subjects to electrodes that are placed within their non-anesthetized noses. Since the EOG is present in some anosmics and can be recorded even after death, it cannot be used, by itself, as a reliable indicator of general olfactory function. Unlike the auditory brainstem evoked potential, the OERP is presently incapable of localizing anomalies within the olfactory pathways. OERP recording sessions can be quite long since relatively long inter-stimulus intervals are needed to prevent adaptation ***(Osman and Silas, 2015).***

Summary

To assess an olfactory disorder, we should start with a full detailed history, then proceed to complete otolaryngological and neurological examination including evaluation of cranial nerves and orbital contents (to direct attention to lesions of the skull base), as well as general evaluation of the upper respiratory tract and ears. A hearing problem reflecting viral or bacterial otitis media may alter taste function in the anterior tongue via chorda tympani nerve (CN VII) damage or inflammation.

Many subjective (psychophysical tests) have been developed to assess olfactory function. These tests assess the odor detection threshold, the odor recognition threshold, the odor identification, the odor discrimination and the odor memory.

Many objective measures have been developed also to assess olfaction including the electro-olfactogram (EOG) which can be used to examine olfactory function, detect malingerers and in forensic medicine. Also chemosensory event related potentials (CSERP) can be used. Imaging using CT, MRI and functional MRI may be used to detect the cause of olfactory function. Functional imaging is more sensitive in detecting brain anomalies than anatomical imaging in case of Alzheimer's disease and Parkinson's disease which are associated with loss of olfactory function. Volumetric evaluation of the bulbusolfactorius (BO), which in turn appears to reflect the degree of afferent neuronal activity as with congenital smell disorders. Biopsies from the olfactory regions may have a role. Also some laboratory tests may be required.

The treatment of olfactory disorders may be non-surgical including the use of systemic and local steroids, antihistaminics, antileukotrienes, antifungal therapy, saline lavage, antibiotic therapy especially macrolides and minocycline, zinc sulphate, theophylline, alpha lipoic acid, caroverine, acupuncture, transcranial magnetic stimulation for parosmia and phantosmia and smell training. Additional non-surgical treatment for Patients with

olfactory distortion (parosmia and phantosmia) include the use of gabapentin, haloperidol, sodium valproate, phenytoin and the topical use of cocaine hydrochloride.

Surgical treatment of olfactory loss (anosmia and hyposmia) aims at either elimination or reduction of nasal obstruction and removal of inflamed mucosa or polyps. These surgeries are not surprising.

Surgical treatment of olfactory distortion (parosmia and phantosmia) includes a bifrontal craniotomy to remove the olfactory bulbs or nerves, Streostacticamygdalectomy, excision of the olfactory epithelium followed by temporalis fascia grafting endoscopically and the transplantation of olfactory cells harvested from the olfactory mucosa.

To assess a gustatory disorder, start with a full detailed history, then proceed to physical examination that is centered on the head and neck (oral cavity, ears, nose and neck) and neurologic systems. A complete physical examination may also be necessary.

Psychophysical evaluation of threshold of taste may be chemical using the three-drop method, taste tablets and wafers and the 8-cup technique, or may be electrical using the electrogustometry.

Psychophysical evaluation of suprathreshold of taste include magnitude estimation, magnitude matching, measuring oral sensory differences (general Labeled magnitude Scales (gLMS) and spatial taste testing.

Objective measurements for assessment of taste function include gustatory event-related potentials, imaging (CT, MRI and FMRI), conofocal microscopy, videomicroscopy of the tongue and other hematological and biochemical investigations to detect the cause. It is important to differentiate between taste loss and flavor loss which is due to interaction between taste, smell, texture, and temperature.

Treatment of taste disorders includes the treatment of the cause despite most of taste disorders are untreatable especially neurological causes. Treatment of idiopathic taste disorders involves zinc, systemic steroids, vitamin A, acupuncture and psychological treatment. If the cause of smell loss is trauma or URTI spontaneous recovery is expected and there is no specific treatment. Treatment of taste dysfunction produced by burning mouth syndrome (BMS) may include tricyclic antidepressants, clonazepam and transcranial magnetic stimulation. Flavour enhancement may be used for patients with unpleasant phantom tastes or a lowered taste threshold. Taste loss due to cancer and radiotherapy can be avoided by modification of cancer therapy if possible, avoid exposure to critical sites of taste, using intensity modulated radiotherapy (IMRT) and image guided radiotherapy (IGRT-tomotherapy) and radioprotectors. Necrotic tumors present with smell and taste changes that may be secondary to Gram-negative bacterial overgrowth, topical and systemic antibiotics may have a role. In xerostomiapilocarpine may have a role. α- lipoic acid may be beneficial in case of idiopathic dysgausia. Mouth wash using chlorhexidine would be effective in some cases of disgeusia for salty or sour. Administration of thyroxine substitutes to hypothyroid patients led to normal reappearance of taste sensation. Tratment of gastric reflux may have beneficial role. For patients with dysgeusia that is severe and significantly interferes with quality of life, Chorda tympani nerve section can be done. Repeated taste testing is always required.

Conclusion:

Smell and taste disorders can markedly affect the quality of life. In recent years validated and reliable methodshave been developed for testing the ability to smell and taste, and these methods allow detailed investigation of these senses. Although some of the methods are very simple, they would, for example, allow determination of the ability to smell and taste for quality control purposes, both before and after surgical procedures. Even though a few treatments currently exist, blinded studies are absolutely necessary in order to separate the effect of a treatment from the spontaneous remission of smell and taste disorders. Of special importance for the treatment is the ability of the olfactory epithelium to regenerate.

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