

# Sensorial abnormalities: Smell and taste

## Anormalidades sensoriais: Olfato e paladar

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### SUMMARY

**Introduction:** Taste and smell abnormalities have proven to be an extremely more complex subject than previously regarded. Wide-ranging nosologic entities arise along with smell and taste alterations, and they can be congenital or acquired.

**Objective:** Analyze the main features of smell and taste dysfunctions.

**Method:** Automated databases were used to collect data, by searching keywords like 'alteration', 'smell', and 'taste'. A non-systematic search was also made in scientific printings and medical books.

**Literature Review:** Smell and taste dysfunctions have a vast etiology, the most significant of which are obstructive nasal and sinus disease, infections of the upper respiratory tract, cranioencephalic trauma, aging, exposure to toxics and some drugs, nasal or intracranial neoplasias, psychiatric and neurological pathologies, iatrogenic disease, idiopathic and congenital causes. A detailed anamnesis, a careful physical examination and supplementary evaluations are important for the diagnosis of these alterations.

**Conclusion:** As a rule, smell and taste dysfunctions occur in a combined way. The early discovery of such dysfunctions can lead to a more efficient treatment, making the progress of diseases causing them retard and the symptoms less severe. In many cases, treating these alterations is not easy and there needs to be a multidisciplinary cooperation among the otorhinolaryngologist, endocrinologist, neurologist, psychiatrist, among others.

**Keywords:** smell, smell disorders, taste, taste disorders, sensory modalities, otorhinolaryngology.

### RESUMO

**Introdução:** Anormalidades do paladar e do olfato comprovaram ser um tema bem mais complexo do que se reconhecia anteriormente. Diversas entidades nosológicas cursam com alterações olfatórias e gustatórias, podendo ser congênicas ou adquiridas.

**Objetivo:** Analisar os principais aspectos das disfunções olfatórias e gustatórias.

**Método:** Foram utilizadas as bases de dados informatizadas para a coleta de dados, tendo como palavras-chave "alteração", "olfato" e "paladar". Realizou-se também busca não-sistemática em publicações científicas e livros médicos.

**Revisão da Literatura:** Disfunções olfatórias e gustatórias possuem etiologia variada, destacando-se as doenças nasais e sinusais obstrutivas, infecções do trato respiratório superior, traumatismo cranioencefálico, envelhecimento, exposição a tóxicos e algumas medicações, neoplasias nasais ou intracranianas, patologias psiquiátricas e neurológicas, iatrogenia, causas idiopáticas e congênicas. Anamnese detalhada, exame físico atencioso e exames complementares adequados são importantes para o diagnóstico dessas alterações.

**Conclusão:** Disfunções olfatórias e gustatórias frequentemente ocorrem juntas. A detecção precoce de tais disfunções pode levar a um tratamento mais efetivo, retardando a progressão das doenças que as ocasionam e atenuando a severidade dos sintomas. Em muitos casos o tratamento dessas alterações não é fácil e é necessária uma cooperação interdisciplinar entre o otorrinolaringologista, endocrinologista, neurologista, psiquiatra entre outros.

**Palavras-chave:** olfato, transtornos do olfato, paladar, distúrbios do paladar, modalidades sensoriais, otolaringologia.

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## INTRODUCTION

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The smell and taste are chemical senses. The neural symptoms intermediating these sensations, the gustatory and olfactory systems are among those phylogenetically older in the encephalon and when they notice chemical substances in the oral and nasal cavities, they work together (1,2).

The sensations arise by the interaction of molecules with the taste and smell receivers. As the impulses are propagated to the limbic system (as well as to upper cortical areas), some smells and tastes can unleash intense emotional responses of memory flows (1).

The importance of taste lies in the fact that it allows an individual to select specific substances according to his/her desires and, frequently, according to the metabolic requirements of the corporeal tissues (3).

More than taste, smell has an emotional quality of being pleasant or unpleasant. That is why smell is probably more important than taste to select food (3).

It is known that taste is particularly a function of the gustatory corpuscles of the mouth, but it is commonsense that the olfactory sense strongly contributes to notice taste (2). It is fundamental to emphasize its relation with taste, since without smell we cannot properly feel the taste of food, hence losing appetite and pleasure while eating.

The gustatory buttons reduce with age and the gustatory papillae, reaching their development climax at puberty, start degenerating at the age of 40-45 in females and at the age of 50 in males (5).

As to smell, the reduction in the olfactory sensitivity with age can result from the degeneration of central cells and independently from peripheral modifications of the olfactory system. However, the regenerating ability of the olfactory epithelium is reduced with age (5). The quality and intensity of the olfactory sense depend on the anatomic and functional of the nasal epithelium of central and peripheral nervous systems. Long-term rhinitis and colds can cause hyposmia (moderated loss of olfactory sensitivity) (4, 6).

The alterations in smell and taste can be associated with the nasal septum abnormalities, nasal polyposis and chronic nasal congestion derived from allergic and non-allergic rhinitis (7).

Damage to the olfactory system, as a result of cranial trauma or even an ordinary cold preventing the molecules transported through the air to be taken to the nasal cavities,

can reduce the sense of taste, even though the basic sensations of sweet, acid, salty and bitter tastes are preserved.

The smell can also help achieve an early diagnosis of some neurodegenerative diseases, such as Parkinson's disease. The result indicates that the olfactory impairment and, subsequently, the taste one, is a relevant indication for an early diagnosis of the disease in a stage in which the typical motor symptoms (such as trembles, rigidity and slowness to perform movements) are not manifested yet (8).

Accordingly, it is important to perform studies about the alterations in smell and taste for an early detection, hence manage a more effective treatment with a view to delaying the progression of diseases and likely complications that may lead to smell and taste losses, thus reducing the severity of the symptoms.

The objective is to perform a literature review about the several aspects associated with the alterations in smell and taste.

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## METHOD

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Automated databases were used to collect data by searching keywords like 'alteration', 'smell', and 'taste'. A non-systematic search was also made in scientific printings and medical books.

### *Morphophysiology*

Olfactory and gustatory senses provide a way to evaluate volatile molecules in the environment and the volatile and non-volatile components of the food. Thus, humans and other mammals are able to distinguish a wide range of smells and tastes. Despite the human olfactory ability is limited, when compared with the ability of other mammals, man is able to notice a huge series of different smelling molecules. Taste sensation results from the association between gustatory, olfactory and somatosensory information (9, 10).

### *Smell*

In men, smell is probably the least understood sense, because it can greatly be a subjective phenomenon. The receiving surface for smell agents is located in the upper part of the nasal cavity and it typically has a surface area of 2.4 cm<sup>2</sup>. The olfactory cells are bipolar neurons derived from the central nervous system. From its apical pole, each neuron stretches only one dendrite onto the epithelial surface, where the dendrite is expanded to a big button, of

which 5-20 thin lashes are protruded in the mucus layer covering the epithelium. From the basal pole, each neuron protrudes only one axon through the cribriform osseous plate above the nasal cavity for the olfactory bulb (10).

There is a specialization of the olfactory neurons to detect smells, because they have specific receivers for smell agents, as well as the transduction machines required to amplify the sensory signals and the generation of action potentials in the neuron axon. The mucus around the lashes is emitted by the support cells of the olfactory epithelium and the Bowman's glands located under the epithelium and have ducts that are opened in its surface. It is believed that the mucus provides the proper smell-detecting molecular and ionic environment (9-11).

The normal neuron response to a smell agent lies in the depolarization and production of action potentials. The number of responding neurons ranges with the agent concentration (9, 10).

The sensorial information of nose is transmitted to the brain through olfactory bulbs. The olfactory nerves pass through the cribriform plate drillings and enter the olfactory bulbs, which are walled structures located above and behind the nasal cavities and consist in a confused node of dendrites of the mitral and clustered cells and olfactory nervous fibers. The mitral axons and the axons of cluster cells leave the bulb through the olfactory tract and enter specialized regions of cortex without passing through the thalamus initially (9, 10).

## **Taste**

Gustatory cells grouped in gustatory buttons in tongue, palate, pharynx, epiglottis and upper third of the esophagus can detect some types of molecules. In tongue, the gustatory buttons are mainly located in the papillae, which are in the epithelium (9-11).

Three morphological types of papillae are found in different areas of the tongue. Several hundreds of fungiform papillae, which have a structured similar to a bolt, are located in the anterior two-thirds of the tongue. In the posterior third, the great circumvallate papillae are located, and each of them is surrounded by a sulcus. The foliate papillae, situated in the posterior border of the tongue, are structures similar to leaves, and each of them is also surrounded by a sulcus. Each fungiform papilla contains between one and five gustatory buttons, whereas each surrounded or foliate papilla contains hundreds of gustatory buttons (9, 11).

There are four morphologically distinguishable

forms of cells found in each gustatory button: basal cells, dark cells, clear cells and intermediate cells. Basal cells are believed to be the germinating cells, which the other cells are derived from.

The gustatory button has a small opening in the epithelium surface named gustatory pore. A hundred or more gustatory cells in each button extend microvillousities, where the sensory transduction occurs. The gustatory cell is invaded by sensory neurons (primary gustatory afferent fibers) in its basal pole. Besides, gustatory cells, such as neurons, are electrically excitable cells, with voltage-dependent channels of sodium, potassium and calcium capable of generating action potentials (9).

For practical purposes, the gustatory system is considered to distinguish among four qualities of basic stimuli: bitter, salty, acid and sweet. The monosodium glutamate can represent a fifth category called 'umami'. Each type of gustatory stimulus is detected by a different mechanism. These interactions typically depolarize the cell, directly or through the action of second messengers. The resulting receiver potential generates action potentials in the gustatory cell, which, in turn, leads to an influx of calcium dependent on the voltage and the release of neurotransmitter in the synapses formed with sensory fibers. Another alternative mechanism can involve the release of calcium from the intracellular stocks (9).

The acid and salty tastes include permeation, or blocking, of ionic channels by sodium ions (salty) or hydrogen ions (acid), whereas sweet and bitter tastes seem to be mediated in some cases by specific receivers (but in some cases they may derive from direct effects in ionic channels) (9).

The gustatory fibers of the anterior two-thirds of the tongue firstly transit by stems of the trigeminal nerve and then by the tympanum cord, a stem of the facial nerve. The gustatory sensation of the posterior third of the tongue is driven by fibers of the glossopharyngeal nerve, whereas other fibers of the epiglottis and other areas are driven through stems of the vagum nerve. From its entrance into the brainstem, all the gustatory fibers are narrowed to the solitary tract and eventually make synapses in the rostral part of the nucleus of the solitary tract. After this point, the axons rostrally pass by barely defined ways towards the ventromedial nucleus of the thalamus and then towards the cerebral cortex in the central area of the post-central gyrus, which is rolled into the lateral fissure (10).

## **Etiology**

A range of nosologic entities occur along with smell

and taste alterations, which can be congenital or acquired, and the ones mostly mentioned in literature are: nasal and obstructive sinus disease, infections of upper airways, cranioencephalic trauma, aging, congenital cause, exposure to toxics, some drugs, nasal or intracranial neoplasias, psychiatric alterations, neurological diseases, iatrogenic and idiopathic. The taste and smell abnormalities are proven to be an extremely more complex subject than previously considered and they are also present in situations such as deficits of vitamins (B6, B12, A) and zinc or copper, tabagism, pregnancy, general anesthesia, dental traumas, arhinencephaly and nasal septum deviations (12-18).

The obstruction is the most common cause of olfactory disorder. If the obstruction is complete, the individual shows an anosmia (smell molecules do not reach the olfactory epithelium), releasing the obstruction and the olfactory ability is returned. The anteromedial part of the inferior part of the medium turbinate bone works as a regulator of the air flow to the olfactory area. An obstruction in this critical area by mucosa edema, polyps, tumors, bone abnormalities, surgeries between the medium turbinate bone and the nasal septum or trauma can reduce or eliminate the olfactory ability. This can also happen when the inferior cavity looks normal. It can occur in any age group, prevalently in women. The patients usually mention a progressive and gradual loss of floating smell, and acute losses can occur with acute infections and exposure to allergens (12, 13).

The upper airway infections are also one of the main causes of olfactory loss. Most of them happen in individuals aged between 40-60, out of whom 70-80% are women, usually by a flow obstruction and it is solved in a period between one and three days. In some cases, smell does not return to normal. At the biopsy, a metaplasia can happen with a reduction or absence of olfactory receivers and a replacement for a respiratory epithelium in some cases. The olfactory loss is proportional to the neuronal loss and the prognosis is poor. One third spontaneously recovers with or without a treatment, and hyposmia occurs more frequently than anosmia. Phantosmia rarely occurs (sense of a smell which is not real) (16, 17, 19).

Cranioencephalic trauma can damage the olfactory nerves in the cribriform lamina because of the coup or countercoup powers. In adults, the olfactory loss is 5-10%; in children, it is 1.3-3.2%. It is more prevalent in the male gender, around 60% of cases. Generally speaking, the loss degree is associated with the trauma severity, what does not mean that a minimum trauma cannot be associated with anosmia. The start of loss is usually immediate, but some patients only notice it after some months. Parosmias

are common. Amnesia in the first 24 hours is associated with a permanent anosmia in more than 90% of cases. When smell is partially preserved, a reduction in the distinction of smells has been noticed (17).

The exact cause has not been determined. The most popular theory assumes a lesion of the nerves when they leave the top of the cribriform lamina. The lesion can happen in the frontal cortex, because, in addition to post-TCE anosmia, some patients show psychosocial alterations. The computed tomography is usually normal, and in some cases it can show a fracture in the cribriform lamina. Hyposmia occurs mostly in frontal lesions; anosmia in occipital lesions, five times more frequent. Around 8-39% of patients recover the function, 75% out of whom do in the three first months (17).

The olfactory threshold reduces with age (1% per year), and this effect is lower in women than in men. The elderly have a higher ratio of smelling reduction for some smells than others, showing a reduction in the ability to distinguish the taste of everyday food. This olfactory reduction is due to either the physiological process of aging (presbyosmia), occurring in the sixth or seventh decade, or to Alzheimer and Parkinson diseases (18).

The olfactory dysfunction is one of the most prevalent signs in the Parkinson disease. Alterations in olfactory discrimination, identification and threshold are observed. Hyposmia is one of the signals that can precede the motor signals of the pathology. In a recent research, it was found that 80% of patients with this pathology showed an abnormal olfactory identification, in comparison with the controls (20- 23).

In congenital anosmia, the likely physiopathology would be the degeneration or atrophy of the epithelium and/or olfactory bulb in the developing process. It is usually an isolated finding, but there is a familiar anosmia associated with a premature baldness and vascular headache, and it is hereditary, dominant with a variable penetration. Kallmann's Syndrome is the most common cause of congenital olfactory dysfunction, 1/10000-50000), with anosmia (agenesis of the olfactory bulb) and hypogonadotropic hypogonadism, as well as kidney abnormalities, undescended testicle, deafness, medium-facial abnormalities and diabetes. It is caused by a defect in the migration of the neurons producing the gonadotropin-releasing hormones (GnRH) and the neurons composing the olfactory nerves. Anosmia is related to the deficit of GnRH because the migration and the differentiation of the secretory neurons of GnRH depend on the formation of the olfactory bulb. The impaired individuals do not understand the idea of smell; therefore, they do not miss it. Due to the fact some chemical

receivers still remain intact, bitter, irritating smells and tastes can be normally detected (24, 25).

When there is an exposure of the olfactory system to toxic substances, the olfactory loss can occur in days or years, and it can be reversible or permanent. The lesion degree seems to be related to the time of exposure and the concentration and toxicity of the agent, commonly associated with tobacco. Examples of olfactory-impairing drugs are: Amphetamine, antibiotics (aminoglycosides, tetracycline), cocaine, petroleum-derived, sulfur dioxide, ethanol, formaldehyde, heavy metals, methanol, carbon monoxide, nicotine, organic solvents, zinc sulfate (topic) and carbon tetrachloride (12, 13).

The drugs usually attack taste more than smell. In most cases, smell is returned when drugs are suspended, but there are reports about a permanent lesion. Drugs attacking the composition of the mucus can change olfaction, such as beta-adrenergic, cholinergic and peptidergic agents.

The neoplastic processes also deserve an attention, emphasizing those of intranasal location, such as nasal polyps, papilloma, epidermoid carcinoma, adenoma, esthesioneuroblastoma (rare neuro-olfactory tumor), because they block the air flow for the olfactory cleft or due to a local destruction of the olfactory system (26).

The intracranial neuroplasias involving the orbital surface of the brain can cause unilateral anosmia. Meningiomas of either the sphenoid ridge or the olfactory sulci, as well as gliomas of the frontal lobe, can cause lesion on the bulbs or olfactory tracts. Anosmia can also occur in association with other tumors of the frontal lobe and parasellar and hypophyseal lesions. In meningiomas of the olfactory sulcus or of the area of the cribriform lamina, unilateral anosmia occurs early and evolves into bilateral anosmia together with the frequency of optical neuropathy. Foster Kennedy's syndrome consists in anosmia together with unilateral ipsilateral optic atrophy and contralateral papilledema derived from a big tumor involving the orbitofrontal area (26).

Some psychiatric pathologies occur simultaneously with smell disorders. Schizophrenics can occur simultaneously with smell hallucinations in 15-30% of cases. Patients showing a bigger depression can show the same symptom, but they usually have a preserved olfactory ability. Phantosmia can be shown as an aura in patients with epilepsy of the temporal lobe (15, 18).

Iatrogenic cannot be unmentioned as a relevant etiologic factor. In surgical procedures, a neural damage

and a narrowing of the nasal flow can occur due to anatomic alterations or cicatricial tissue. Alterations in the smell and taste occur after total laryngectomy, because the patients start breathing directly through the trachea and the air does not pass through the nose to the terminal olfactory organs. As smell and taste are closely linked, sensations of taste are changed. Yet, through time, the patient usually gets used to this problem, what can justify the fact that not all patients mention an olfactory alteration (27-29).

In surgeries of the anterior fossa and post-transsphenoidal neurosurgery, cribriform lamina lesion can occur. Radiotherapy is also included in the group of conditions leading to smell and taste dysfunctions, as well as those with idiopathic causes, usually in young adults, in middle age and healthy individuals (12, 26).

In leprosy, the olfactory alterations can be found in any form of the disease. Besides, it is a common complaint in this pathology and it can be mostly found in the lepromatous form, and it is reported that this impairment is associated with the severity of the clinical alterations in the nasal mucosa. In a study performed in 2005, the findings of olfactory alterations were obtained in four different forms of leprosy; however such findings were obtained in patients at an advanced stage or in reaction. Hyposmia was found in 7.5% of patients; cacosmia in 2.3%, and anosmia in 0.6% (16, 30, 31).

A few cases of olfactory disorders have a neurological background. Multiple sclerosis can cause olfactory alterations due to involving the olfactory areas. Several neurological conditions causing anosmia include hydrocephaly, impairment of the anterior cerebral artery near its origin, basilar meningitis, abscesses of the frontal lobe and Refsum's disease. Temporal lobectomies including the piriform cortex can cause deficits in smell identifications (26).

Hyperosmia is usually functional, but it can occur in certain types of drug abuse and migraine. Olfactory hallucinations are mostly due to psychosis, but they can as well derive from a lesion in the central olfactory system, usually neoplastic or vascular, or as a manifestation of convulsive crisis. The so-called uncinate crises are partial complex crises of the temporal lobe preceded by an usually unpleasant olfactory or gustatory atmosphere, and they are often accompanied, whereas the patient loses the conscience of lip-cracking and chewing movements. These attacks are usually derived from a convulsive focus involving structures of the medial temporal lobe (26).

The taste can be attacked in cases of lesions of the

facial nerve near the exit of the tympanic cord. However, in the case of permanent gustatory disorders, these can survive after Bell's facial palsy. Dysfunctions in the taste and smell usually occur together, because the abnormalities of the taste usually result from the olfactory dysfunction. Dysgeusia can be a direct or indirect effect of malign conditions. Hypergeusia and parageusia can occur in psychoses and in the conversion disorder (32).

Gustatory hallucinations can occur in partial complex crises and in tumors involving the uncus and the parietal operculum and they often occur together with olfactory hallucinations. Elderly patients sometimes develop obscurely originated dysgeusia that can cause anorexia and loss of weight. The increased gustatory sensitivity occurs in patients having Addison's disease, pituitary deficits and cystic fibrosis. Lesions in the lingual nerve can cause a loss of palate together with the loss of exteroceptive sensation of the affected side of the tongue (32).

### Diagnostic Methods

The evaluation of the olfactory alterations can be made by introducing smells (cinnamon, turpentine, lemon, smoke, chocolate, rose, paint solvent, banana, pineapple, gasoline, soap, onion). Each nostril must be separately explored, and the patient is inquired about the type of smell (21, 33).

A detailed anamnesis must be performed, clarifying out family diseases, previous surgeries, cranial traumas, exposure and/or use of drugs. Microscopy or endoscopy can define alterations in the air flow as a cause of anosmia (33).

The nasal endoscopy is useful to access the olfactory ridge, and it is in association with the computed tomography, the most sensitive ways to diagnose pathologies derived from the nasal cavity, paranasal sinus and encephalon. Rhinometry shows a little diagnostic value, and it is valid only to demonstrate the reductions in the respiratory flow. The nuclear magnetic resonance is useful to evaluate the olfactory bulb, olfactory tracts and intracranial causes of olfactory disorders (34).

TSUKATANI et al in 2005 demonstrated that these tests agree with each other when evaluating whether an olfactory dysfunction is absent or present; however, the tests disagree when evaluating the levels of hyposmia (34).

In the detection tests, a lower smelling concentration capable of being detected is pursued. They are performed to provide the patient with two or more stimuli, only one

of which has a smelling substance. This type of investigation proved to be more effective than simply asking whether a smell can or cannot be felt (35).

In the recognition tests, a lower concentration of the smell capable of being recognized is pursued, and the most used method is the ascending threshold one. In this test, smells are sequentially presented from the lowest to the highest concentration and it is estimated the point of smell recognition (35).

It is emphasized that both the detection test, such as the recognition test, are subjective methods and depend on factors like age, cooperation of the examined patient and degrees of his/her understanding, lasting around 20-30 minutes for its correct performance (35).

BRINER & SIMMEN in 1999 described a screening olfactory test by using eight diskettes containing different smells (5), which are open to release the smell and closed after the test, and one (01) point is assigned for each right answer the patient gives. The values between  $6,2 \pm 1,0$  are considered normal for the age group between 18 and 50 and  $6,0 \pm 0,9$  for the group between 51 and 80. It is a simple and rapidly performed method, and it eliminates the risk of contaminating the examiner's hand and the patient by the smell. Nevertheless, there is an intense release of the smell when the diskette is open, and it is therefore a suprathreshold test, functioning as a screening (36).

The "University of Pennsylvania Smell Identification Test" (UPSIT) is a widespread psychophysical test (subjective). 50 different smells are provided to the patient by a card evaporating a smell when it is risked. The following items are analyzed: intensity, irritation, cold, familiarity and pleasantability (37).

The "Modular Smell Identification Test" (MODSIT) is a variation of the UPSIT method, yet, with a lower cost and time to perform the exam, because 12 smells are provided to the patient that needs to be taught how to read the card (38).

Apparatus for the subjective olfactory measurement are described in literature, such as T&T olfactometer, consisting in small bottles containing dilutions of 5 different smells, and it is used to determine the threshold of detect and recognition of each stimulus, hence, achieving a medium threshold value and the Jet Stream olfactometer (jet current), consisting in three parts: one adaptor for the nasal fossa, one device to collect and dilute the smelling in a tube and a mini air compressor (39,40).

Other exams can help diagnose olfactory dysfunction, such as: Single-Photon Emission Computed

Tomography (SPECT), olfactory-pupillary reflex olfactory-tensional or cardiovascular reflex, cutaneous or psychogalvanic reflex, olfactory-respiratory reflex, electro-olfactogram and the evoked potential of the olfactory nerve (4).

A detailed anamnesis is also important to diagnose the taste alterations. In some situations, help from other specialists is necessary, such as endocrinologist and geneticist, to better clarify the diagnosis. Solution-solved papers with different concentrations of glucose, salt, acid, etc., can be used, or electrogustometry can be used, and it comprises the stimulation of the tongue with electrical currents, causing a sensation of acid and/or metallic taste on the patient (33).

### Treatment

Many simple ideas can be suggested to patients with taste alterations. Chewing gum or ice can work as a temporary help in hypogeusia. The patients must be encouraged to chew their food very well, changing the sides of the mouth or their foods (41).

Disorders in chemical sensitivity, smell and taste are symptoms of diseases; hence the treatment depends on their cause. The olfactory alterations caused by viral infections are treated with oral hydration, relaxation and analgesia, if necessary. In nasal obstructive causes, a surgical correction together with topic corticosteroids has been effective. In trauma-derived losses, the gustatory and olfactory alterations, if they do not recede after the improvement of the edema, they are usually irreversible (3).

In many cases, treating the smell and taste alterations is not easy and there needs to be a multidisciplinary cooperation containing the otorhinolaryngologist, endocrinologist, neurologist, psychiatrist, among others (41).

### CONCLUSION

Smell and taste show a complex anatomy and physiology, however not entirely known. Its relevance to animals and human beings is vital and its loss causes severe consequences in the quality of life, as well as it can be a risk to the individual's health. Damage to the olfactory system can reduce the sense of taste, since these systems are closely connected. Knowing the tests presently available is essential to perform an accurate diagnosis and answer the patient's doubts about the partial or total loss of smell and taste. Individuals must bear in mind that the early detection of these alterations can lead to a more effective treatment,

with a view to delaying the progression of the diseases causing olfactory and gustatory losses, hence reducing the severity of the symptoms.

### REFERENCES

1. Tortora GJ, Grabowski SR. *Corpo Humano: Fundamentos de Anatomia e Fisiologia*. 6ª ed. Porto Alegre (RS): Artmed; 2005.
2. Pellegrini G, Veleiro RVB, Gomes ICD. A percepção do gosto salgado em indivíduos com e sem obstrução nasal. *Rev. CEFAC* 2005, 7(3):311-7.
3. Hungria, H. *Otorrinolaringologia*. 8ª ed. Rio de Janeiro (RJ): Guanabara Koogan; 2000.
4. Rocha FMN, Ximenes Filho JA, Alvarenga EHL, Mello Jr JF. *Olfação: revisão de literatura*. *Arq Int Otorrinolaringol*. 2002, 6(2):123-8.
5. D'Ottaviano EJ. *Sistema nervoso e 3ª idade: 2ª parte*. *Rev. das Faculdades de Educação, Ciências e Letras e Psicologia Padre Anchieta*. 2001, 3(5):19.
6. Almeida MM, Freire GL, Morais LCSL. *Implantação e avaliação da prática: "cansando o olfato"*. In: *Encontro de iniciação científica à docência*, 11; 2008 outubro; Paraíba. Anais. p. 33 [resumo 002].
7. Weckx LLM. *Consenso sobre rinites*. *Rev Bras de Otorrinolaringol*. 2000, 66(3):1-34.
8. Toneloto C. *Mal de Parkinson: teste do olfato auxilia no diagnóstico precoce da síndrome*. *Ciencia e Cultura*. 2007, 59(3):11-3.
9. Buck LB. *Olfação e gustação: os sentidos químicos*. In: *Kandel ER, Schwartz JH. Princípios da neurociência*. 4ª ed. Barueri: Manole; 2002. p.625-47.
10. Guyton AC, Hall, JE. *Os sentidos químicos: gustação e olfação*. In: *Guyton AC, Hall, JE. Tratado de fisiologia médica*. 10ª ed. Rio de Janeiro: Guanabara Koogan SA; 2002. p.570-7.
11. Junqueira LC, Carneiro J. *Histologia básica*. 10ª ed. Rio de Janeiro (RJ): Guanabara Koogan S.A.; 2004.
12. Andreas FP, Temmel MD, Christian QMD, Bettina SFMD, Ludger KMD, Hummel EST. *Characteristics of olfactory disorders in relation to major causes of olfactory loss*. *Arch Otolaryngol Head Neck Surg*. 2002, 128(8):635-41.

13. Daniel A, Kimmelman CP, Mester AF, Brightman VJ, Settle GR, Snow JB et al. Smell and taste disorders, a study of 750 patients from the University of Pennsylvania. *Otolaryngol Head Neck Surg.* 1991, 117(5):519-28.
14. Hummel T, Nordin S. Olfactory disorders and their consequences for quality of life. *Acta Oto-Laryngologica.* 2005, 125(2):116-21.
15. Kopala LC, Good KP. Olfactory deficits in patients with schizophrenia and severe polydipsia. *Biol Psychiatry.* 1998, 43(7):497-502.
16. Kern RC. Chronic sinusitis and anosmia: pathologic changes in the olfactory mucosa. *Laryngoscope.* 2000, 110(7):1071-7.
17. Mueller A, Rodewald A, Reden J, Gerber J, von Kummer R, Hummel T. Reduced olfactory bulb volume in post-traumatic and post-infectious olfactory dysfunction. *Neuroreport.* 2005, 16(5):475-8.
18. Solomon GS, Petrie WM, Hart JR, Brackin HB. Olfactory dysfunction Discriminates Alzheimers dementia from major depression. *J Neuropsychiatry Clin Neurosci.* 1998, 10(1):64-7.
19. Weckx UM, Sakano E, Araújo E, Castro F, Aun W. Consenso sobre Rinites. *Rev Bras Otorrinolaringol.* 2000, 66 (supl. 10):1-34.
20. Katzenschlager R, Lees AJ. Olfaction and Parkinson's syndromes: its role in differential diagnosis. *Curr Opin Neurol.* 2004, 17(4):417-23.
21. Quagliato LB, Viana MA, Quagliato EMAB, Simis S. Alterações do olfato na doença de Parkinson. *Arq Neuropsiquiatr.* 2007, 65(3):647-52.
22. Doty RJ, Bromley SM, Stern MB. Olfactory testing as an aid in the diagnosis of Parkinson's disease: development of optimal discrimination criteria. *Neurodegeneration.* 1995, 4:93-7.
23. Katzenschlager R, Lees AJ. Olfaction and Parkinson's syndromes: its role in differential diagnosis. *Curr Opin Neurol.* 2004, 17:417-23.
24. Ribeiro RS, Abucham J. Síndrome de Kallmann: uma revisão histórica, clínica e molecular. *Arq Bras Endocrinol Metab.* 2008, 52(1):8-17.
25. Schmidt VB, Roithmann R, Corleta HE, Capp E. Hipogonadismo hipogonadotrófico e anosmia: síndrome de Kallmann. *Rev Bras Otorrinolaringol.* 2001, 67(6):880-4.
26. Campbell, WW. O nervo olfativo. In: Campbell, WW. DeJong, o exame neurológico. 6ª ed. Rio de Janeiro: Guanabara Koogan; 2007. p.97.
27. Smeltzer SC, Bare BG. Brunner & Suddarth: tratado de enfermagem médico-cirúrgica. 8ª ed. Rio de Janeiro (RJ): Guanabara Koogan S.A; 1998.
28. Silva LSL, Pinto MH, Zago MMF. Assistência de enfermagem ao laringectomizado no período pós-operatório. *Rev Bras de Cancerol.* 2002, 48(2):213-21.
29. Ghirardi, ACAM. Laringectomizados usuários de prótese traqueoesofágica: princípios e métodos da prática fonoaudiológica [Tese - Mestrado]. São Paulo (SP): Pontifícia Universidade Católica de São Paulo; 2007.
30. Martins ACC, Castro JC, Moreira JS. Estudo retrospectivo de dez anos em endoscopia das cavidades nasais de pacientes com hanseníase. *Rev Bras Otorrinolaringol.* 2005, 71(5):609-16.
31. Barton RPE, Davey TF. Early leprosy of the nose and throat. *J Laryngol Otol.* 1976, 90(10):953-61.
32. Campbell, WW. O nervo facial. In: Campbell, WW. DeJong, o exame neurológico. 6ª ed. Rio de Janeiro: Guanabara Koogan; 2007. p.190.
33. Maffeis ER, Netto SCR. Fatores que alteram a percepção gustativa. *Rev Fac Odontol Lins.* 1990, 3(2):28-32.
34. Tsukatani T, Reiter, ER, Miwa T, Costanzo RM. Comparison of Diagnostic Findings using Different Olfactory Test Methods. *Laryngoscope.* 2005, 115(6):1114-1117.
35. Doty RL, Kobal G. Current trends in the measurement of olfactory function. *Handbook of olfaction and gestation.* 1995; 8: 191-225.
36. Briner HR, Simmen D. Smell diskettes as screening test of olfaction. *Rhinology.* 1999, 37:145-148.
37. Doty RL, Shaman P, Dann M. Development of the University of Pennsylvania Smell Identification Test: A Standardized Microencapsulated Test of Olfactory Function. *Physiology & Behavior.* 1984, 32:489-502.
38. Liu HC, Wang SJ, Lin KP, Lin KN, Fuh JL, Teng EL. Performance on a smell screening test (the MODSIT): A study of 510 predominantly illiterate Chinese subjects. *Physiology & Behavior.* 1995, 58(6):1251-5.
39. Kondo H, Matsuda T, Hashiba M, Baba S. A study of the



relationship between the T&T Olfactometer and the University of Pennsylvania Smell Identification Test in a Japanese population. *Am J Rhinology*. 1998, 12(5):353-8.

40. Ikeda K, Tabata K, Oshim T, Nishikawa H, Hidaka H, Takasaka T. Unilateral examination of olfactory threshold using the Jet Stream Olfactometer. *Auris Nasus Larynx*. 1999, 26:435-9.

41. Burkert S, Haberland EJ, Gudziol H. Olfactory and gustatory disorders: causes, diagnosis and treatment. *MMW Fortschr Med*. 2005, 147(11):51-3.