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Clinical assessment of patients with smell and taste disorders

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Disorders of sense of smell and taste can present diagnostic and therapeutic challenges for the otolaryngologist. A systematic approach to evaluating the patient, detailed knowledge about the etiology of olfactory and gustatory disorders, and proper use of clinical tests helps to establish an appropriate diagnosis and assess the degree of the sensory impairments. Knowledge of the etiology of olfactory and gustatory dysfunction is fundamental for establishing the diagnosis and directing the work-up. In the last 20 years, significant progress has been made in the development of widely available, reliable, and reproducible methods of evaluating olfactory function. The assessment of the taste remains less standardized.

Although the patient who has olfactory dysfunction typically complains of loss of taste, true gustatory disorders are rare [1–3]. Because up to 80% of a meal's flavor is a result of olfactory input, patients frequently interpret a loss of smell as a loss of taste. For most patients who complain of chemosensory loss, however, the sense of taste—biologically the sensation of salt, bitter, sweet, sour, and umami (monosodium glutamate)—is intact.

This article outlines a systematic approach to the clinical evaluation of patients who have smell and taste disorders and discusses the etiology of smell and taste dysfunction.

Olfactory system

Types of olfactory dysfunction

The normal ability of smell is termed *normosmia*. The terms for the olfactory disorders are as follows: *anosmia,* inability to smell; *hyposmia,*

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decreased ability to smell; and *dysosmia,* altered perception of smell, which includes *phantosmia*, perception of odor without stimulus present, and *parosmia* or *troposmia*, altered perception of an odor in the presence of an odorant stimulus. Flavor is the combined perception of taste and smell.

Etiology of olfactory disorders

Olfactory dysfunction can be caused by various etiologic factors. The two main types of olfactory dysfunction are as follows:

1. Conductive losses: Losses secondary to obstruction of the nasal airflow to the olfactory cleft. Examples include chronic rhinosinusitis (CRS) (although a neural loss in CRS also has been documented), allergic rhinitis, polyps, and tumors.
2. Sensory/neural losses: Losses secondary to damage to or dysfunction of the olfactory nerves anywhere from the olfactory receptors through the olfactory bulb to the processing centers in the brain. Examples include loss of smell after upper respiratory infection (URI), head trauma, toxins, congenital disorders, dementia, Alzheimer's disease, and multiple sclerosis.

Obstructive nasal disease and sinus disease

Olfactory loss can be secondary to complete obstruction of the airflow from intranasal tumors such as inverting papilloma, hemangioma, squamous cell carcinoma, esthesioneuroblastoma, or sinonasal-undifferentiated carcinoma. Unilateral testing of the sense of smell sometimes will show a loss on one side. CRS was viewed as a solely conductive cause of olfactory dysfunction, but more studies suggest that olfactory dysfunction secondary to CRS is caused by conductive and neural factors. Kern [4] looked at the histopathologic changes in the olfactory mucosa of patients who had CRS undergoing endoscopic sinus surgery. Neuroepithelial inflammation was found with olfactory deficits, suggesting that inflammation of the olfactory epithelium, in addition to altered airflow, may contribute to olfactory losses.

Aging and effect on olfaction

Decreased olfactory function often is associated with aging. Olfactory identification ability drops sharply in the sixth and seventh decades of life. More than half of people 65 to 80 years of age show major olfactory declines [5] that may be related to extensive degeneration in the olfactory bulb associated with aging. Whether this group is more susceptible to losses after surgery is unclear.

Alzheimer's disease and Parkinson's disease are associated with decreased olfactory function, likely as a result of damage to the olfactory bulb or central olfactory cortex [6]. Decreased sense of smell is often the first sign of these dementias. Neuronal loss has been identified in the olfactory bulb and

neural tracts of individuals who died of Parkinson's disease with a strong correlation to the duration of the disease process.

Congenital olfactory dysfunction

Patients who have congenital olfactory dysfunction usually have no memory associations of flavor or smell perception throughout their life [7]. Usually, the anosmia is not noticed until approximately 10 years of age. The differential diagnosis is difficult because other causes such as head trauma and post-URI must be excluded. Few syndromes have been associated with anosmia; the best known is Kallmann syndrome, or hypogonadotropic hypogonadism. Failure to develop secondary sexual characteristics in an anosmic male should cause one to suspect the syndrome and prompt referral to an endocrinologist. MRI may demonstrate the absence of olfactory bulbs in these patients [8]. Another congenital olfactory dysfunction, familial anosmia, has been associated with baldness, vascular headaches, and other features.

Head trauma

Few patients with head trauma (5%–10%) lose olfactory function [9,10]. In the pediatric population, the reported incidence of olfactory loss from head trauma is 3.2% (transient) and 1.2% (permanent) [11]. The degree of loss usually correlates with the severity of trauma. Blunt impact to the frontal region is associated most commonly with olfactory losses, followed by occipital trauma.

Posttraumatic olfactory dysfunction can be the result of: (1) shearing injury of the olfactory nerve axons, (2) brain contusion and hemorrhage in the olfactory regions, and (3) sinonasal tract alternation. The usual course of injury to olfactory function is an immediate onset, but it also can be delayed months after the initial injury [12,13]. In 10% of cases, olfactory function returned but usually was diminished. Human olfactory biopsy studies after head trauma suggest that regrowing olfactory neurons cannot find the olfactory bulbs or the holes in the cribriform plate and therefore olfaction does not return [14].

Upper respiratory infection

URI is the most frequent (25%–33%) cause of neural olfactory loss. Such loss more commonly affects women (70%–80% of cases), is more frequent in people 65 years or more of age [15], and follows the viral-like URI. One third of patients will improve after 6 months, but the longer the dysfunction is present, the poorer the prognosis for recovery. Among patients who have URI-related olfactory loss, hyposmia is more common than anosmia. Viruses can cause severe damage to the olfactory neuroepithelium, with replacement by respiratory epithelium, but this has not been proved in humans. Biopsy specimens of the olfactory cleft in such patients show either fewer or no olfactory receptors [16,17]. Coronavirus was shown in mice to

cause degeneration and destruction of the olfactory bulb and little effect on the neuroepithelium [18]. Although this condition is thought to be caused by viruses, this has not been proved, nor has the exact viral type been identified.

Exposure to toxin

Some patients have olfactory dysfunction after exposure to toxins, various gases, and aerosols. Common chemicals known to cause olfactory loss include formaldehyde, cyanoacrylates, herbicides, pesticides, and cigarette smoke. The concentration of the toxins and the length of exposure correlate directly with potential damage to the olfactory system [19]. These losses are usually permanent.

Nasal surgery and risk to olfaction

Olfactory impairment can result from surgical intervention [20–22]. Postsurgical scarring between the middle turbinate and the septum can alter the pathway for the airflow to the olfactory cleft. Olfactory impairment also may result from overresection of the superior/middle turbinate or avulsion of the olfactory neuroepithelium, especially in patients whose only remaining olfactory neuroepithelium is in an "exposed" location.

Miscellaneous

Psychiatric disorders such as depression and schizophrenia have been associated with olfactory losses. Patients who have dementia, epilepsy, Down syndrome, or various endocrine abnormalities such as hypothyroidism, Addison's disease, Cushing's disease, and diabetes mellitus may have decreased or absent olfactory ability. Nutritional deficiencies (eg, vitamin A, zinc, or thiamine) affect olfaction rarely. Progressive hyposmia leading to anosmia can result from intracranial tumors and anterior cranial fossa tumors such as Foster-Kennedy syndrome–olfactory-groove meningioma, frontal-lobe gliomas, and pituitary adenomas. These patients usually present with olfactory complaints and cranial nerve abnormalities. Anosmia can be an initial symptom of neurosarcoidosis. One quarter of temporal lobe tumors cause olfactory dysfunction.

Dysosmia also can be part of the presentation of psychiatric disorders (psychosis); psychosomatic anosmia usually is accompanied by ataxia and hysteria. Dysosmia as auralike hallucinations can be the only symptom of temporal lobe seizures. The metabolic disorder trimethylaminuria (fish-odor syndrome) and the pediatric neurologic disorder cat-odor syndrome, associated with b-methyl-crotonyl-CoA carboxylase deficiency, can present as dysosmia. Finally, 30% to 40% of patients with olfactory dysfunction have an undetermined etiology.

Etiology of parosmia (troposmia) and phantosmia

A distortion of the sense of smell sometimes follows damage to the olfactory neuroepithelium, such as after URI or head trauma, or can be

associated with aging. It also can be secondary to temporal lobe tumors or seizures. These patients usually have decreased olfactory ability in at least one nostril on testing [23]. Often, however, there is not a good reason for the olfactory loss or perceived distortion.

Clinical assessment of smell

Clinical history

The first and most important step in evaluating a patient who has a smell disorder is to obtain a detailed history and clearly define the symptoms (Fig. 1). Most patients with chemosensory deficits complain of taste alteration, but upon closer inquiry, patients state that they can taste sweet, bitter, sour, and salt. These patients could say more accurately that food has decreased flavor. Perception of flavor, therefore, is lost mostly because of olfactory damage, not a gustatory disorder. Among patients with complaints of smell and taste loss, less than 5% suffer true gustatory loss.

The examiner should document with examples the status of the patient's chemosensory ability before present loss. It is important to estimate the degree of olfactory loss (eg, complete or partial) and the duration (weeks, months, years). The examiner should inquire about events that occurred around the time of the initial loss such as head trauma, exposure to toxins or fumes, URI, or nasal surgery. Unilateral epistaxis or obstruction gives rise to suspicion for sinonasal tumor. A fluctuating sense of smell is common in patients who have rhinosinusistis and polyposis. Some of these patients will report temporary improvement of olfactory ability when the nasal patency increased, such as after using topical or systemic corticosteroids. Attention also should be paid to a history of sinus problems, allergies, nasal obstruction, visual changes, central nervous system complaints, delayed puberty (Kallmann syndrome), or endocrine (hypothyroidism) or metabolic (uremia) abnormalities. Decreased olfactory function has been observed in some patients who have HIV. Past medical history is important to review for occupational exposure and cigarette smoke. Olfactory ability decreases as a function of cumulative smoking dose [24].

The patient's current medications and the ones used at the time of the onset of olfactory dysfunction need to be reviewed because many medications can affect olfaction and gustation. Some examples are adrenal corticosteroids, amino acids in excess (eg, cysteine or histidine), antimicrobials (eg, neomycin, penicillins, macrolids), antithyroids (eg, methimazole, thiouracil), opiates (eg, codeine, morphine), and cardiovascular or hypertensive medications [25]. Usually the olfactory function will return after the medication is discontinued, but the change can be permanent. Attention must be paid to signs of early dementia, Parkinson's disease, depression, or schizophrenia.

Physical examination

A complete ears, nose, and throat examination with emphasis on the nasal airways is necessary. Nasal endoscopy is especially helpful in

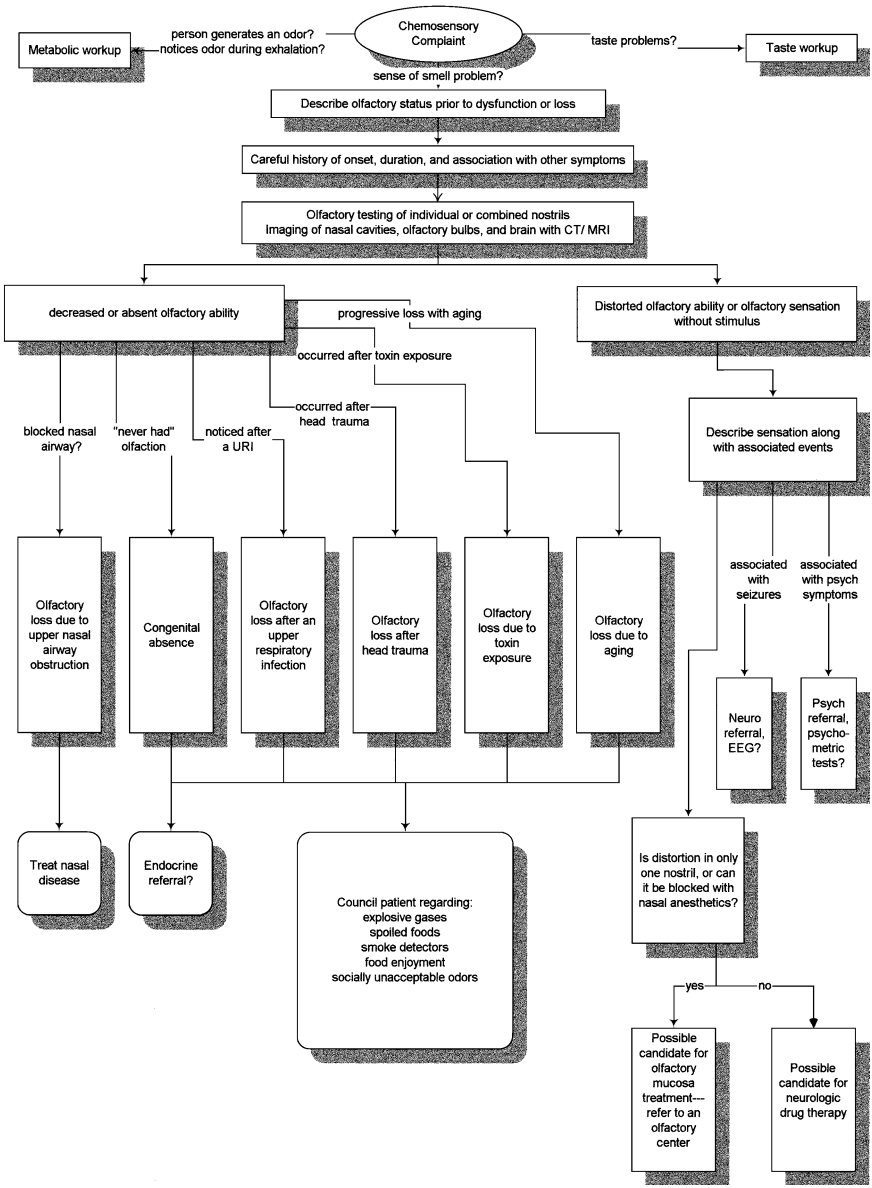

Fig. 1. Algorithm of evaluation of the patient who has olfactory loss. *From* Leopold DA. Physiology of olfaction. In: Cummings CW, Fredrikson JM, Harker LA, et al, editors. Otolaryngology Head and Neck Surgery, Third edition, Philadelphia: Elsevier/Mosby-Year Book; 1998; Vol. 2, Chapter 41, p. 70; with permission.

identifying obstructive causes of olfactory losses. Anterior rhinoscopy alone can miss 51% of obstruction of the olfactory cleft versus 9% for nasal endoscopy [26]. During the nasal endoscopy, attention should be paid to the patency of the olfactory cleft and middle meatus; the presence of polyps or tumors; mucosal edema of the sinonasal mucosa; the presence of airspace between the middle turbinate and the septum, especially superiorly toward the olfactory cleft; and presence of adhesions, postoperative changes, or crusting.

If a patient presents with distortion of smell (parosmia or phantosmia) localized to one nostril, blocking the affected nostril with local anesthetic (cocaine, 4%) can help to identify the candidate for endoscopic excision of olfactory neuroepithelium [27]. A neurologic examination should be performed with emphasis on assessment of cranial-nerve function. Optic-disk examination can determine the presence of papilledema, a sign of increased intracranial pressure seen in patients who have unilateral olfactory dysfunction secondary to tumors of the olfactory groove. Tumors of the olfactory groove and sphenoidal ridge can cause the combination of symptoms known as Foster Kennedy syndrome: ipsilateral hyposmia or anosmia, ipsilateral optic nerve atrophy, and central papilledema. Because olfactory dysfunction can be a first sign of Alzheimer's disease, Parkinson's disease, or multi-infarct dementia, the otolaryngologist can perform the Mini-Mental State Examination if dementia is suspected. If further assessment of dementia is required, proper referral to a neurophysiologist should be made.

Imaging

If nasal obstruction is suspected secondary to anatomic deformity, tumor, or polyps, then CT scan of the sinuses is indicated. Presence of an intranasal mass warrants further evaluation with MRI to assess possible intracranial extension. In many patients presenting with a loss of smell, imaging is not indicated. Brain tumors that present with isolated anosmia are extremely rare, which is why imaging is not required [28]. MRI can be used to confirm agenesis of olfactory bulbs in Kallmann syndrome. Imaging is indicated in individuals who present with unusual, ominous symptoms or a pattern that does not fit a standard diagnosis. For instance, a 30-year-old woman with olfactory loss that occurred 3 days after a URI would not need imaging if her nasal endoscopy was healthy.

Olfactory testing

Mandatory tests for the work-up of the patient with chemosensory complaints include testing of the gustatory and olfactory ability [29]. Assessment of olfactory function is essential to establish the degree of chemosensory loss and confirm the patient's complaint of olfactory loss. Also, it enables monitoring of the changes in olfactory function over time (especially in patients with anosmia/hyposmia after nasal surgery or head

trauma) and helps to detect malingerers and to establish compensation for permanent disability. (See later discussion for information about gustatory testing.)

Whether to use unilateral or bilateral olfactory testing can be predetermined from the history and physical examination. If unilateral testing is performed, some recommend occlusion of the nontested nostril (eg, with a piece of Microfoam tape) to prevent crossing of inhaled or exhaled air at the nasopharynx to the opposite side.

There are two general types of olfactory testing: psychophysical and electrophysiologic testing. The information derived from these tests does not allow differentiation between central and peripheral deficits. Psychophysical tests are used for clinical evaluation of olfactory loss, whereas electrophysiologic tests are used primarily for research. Most olfactory testing relies on measuring detection thresholds of a specific odorant or the ability to identify multiple odorants [30].

Psychophysical tests

Olfactory threshold tests. The absolute threshold of detection is defined as the lowest concentration of an odorant that can be detected reliably. The forced-choice procedure is used, in which an odorant such as phenyl ethyl alcohol (PEA) or butyl alcohol, 4%, in one sniff bottle and water in another bottle are presented. The patient is asked to identify the bottle containing the odorant. There are two common ways to present the stimulus: the ascending method of limits procedure (AML) and the single staircase procedure (SS). In the AML, the odorant and the water are presented sequentially from low to high concentration and the point of transition between no detection and detection is estimated [5]. In the SS method, the concentration of the stimulus is increased following trials in which a subject fails to detect the stimulus and decreased following trials in which correct detection occurred. In both methods, the stimulus is presented from weak to strong. The SS procedure is more reliable and used more often in threshold testing.

Commercially available smell threshold test kits use squeeze bottles or odorant-impregnated felt-tipped pens. These kits can be used to determine a patient's threshold for rose-like PEA or sweet alcoholic n-butanol [31]. Results are compared with norms based on hundreds of subjects in different age groups.

Odor identification tests. Odor identification tests are quantitative tests. Patients are asked to identify the odorants at the suprathreshold level.

The Connecticut odor identification test presents 10 stimuli in an opaque jar to each nostril. Seven of the odorants stimulate primarily cranial nerve I: baby powder, chocolate, cinnamon, coffee, mothballs, peanut butter, and soap. The other three odorants are trigeminal stimulants. Patients are given a list of 20 possible answers, of which 10 are the actual stimuli and the other

10 are names used as distracters, and asked to choose the answer from the list. The score is calculated from the number of correctly identified stimuli and compared with a normal control-group performance.

In clinical practice, the instrument most widely used for olfactory testing is the University of Pennsylvania Identification Test (UPSIT). The test consists of four "scratch and sniff" booklets, each containing 10 microencapsulated odorants (Fig. 2). Above the "scratch and sniff" strip on the bottom of the page is a question with four answers. The patient is required to choose an answer even if he or she does not recognize the odorant. The test is self-administered and usually takes 15 to 20 minutes to complete. Chance performance is 10 out of 40, so a score less than 5 or 6 raises the suspicion of malingering. The UPSIT has been studied extensively and has norms based on testing 4000 people. Scores are compared with various patient groups and against sex- and age-related norms [32]. According to the test results, a patient's olfactory function is classified as normosmia, mild microsmia, moderate microsmia, severe microsmia, anosmia, or probable malingering. A chart is available to compare the patient's score to those of

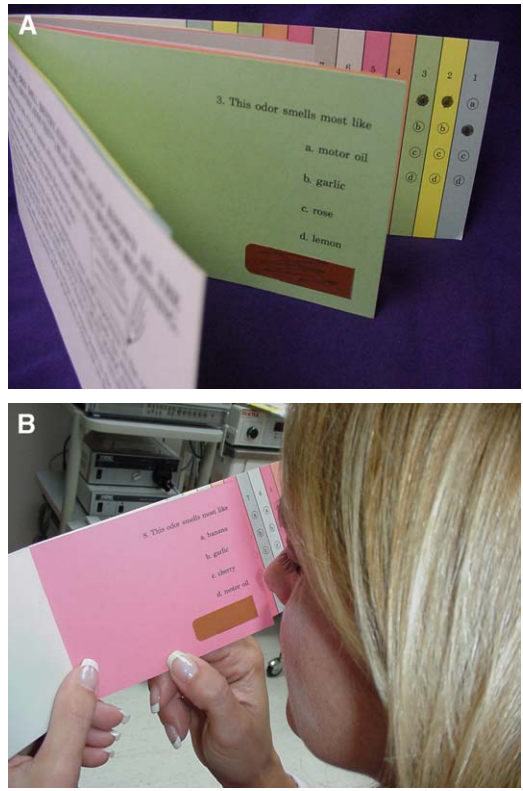

Fig. 2. (*A*,*B*) University of Pennsylvania Identification Test.

various patient populations, including patients who have Korsakoff syndrome and multiple sclerosis. The reliability of the test is high (test-retest Pearson r = 0.94).

Cross-cultural smell identification test. The Cross-Cultural Smell Identification Test (CC-SIT) is a variant of UPSIT and contains 12 items [33]. The odorants include banana, chocolate, cinnamon, gasoline, lemon, onion, paint thinner, pineapple, soap, smoke, and turpentine. These stimuli are the most consistently identified odorants by subjects representing various countries (China, France, Germany, Italy, Japan, Russia, and Sweden). It takes approximately 5 minutes for the patient to complete this test.

In Europe, a commonly used test is a threshold- and odorant-identification forced-choice test that uses odorant-impregnated felt-tipped pens (Sniffin' Sticks). This test developed by Kobal and Hummel [31,34,35] contains one test in the threshold range (determination of the olfactory perception threshold for n-butanol) and two suprathreshold tests (discrimination and identification). The test can be reused and is easy to administer, but unlike the UPSIT, it requires the help of a medical assistant for the presentation of sticks [36].

Recently, a simple olfactory screening test using a 70% isopropyl alcohol pad as a stimulant has been described. The alcohol pad is brought gradually toward the patient's nose. The patient with eyes closed reports the moment when he or she can detect the odor, and then the distance is measured from the nares to the pad. The distance correlates with the degree of olfactory loss [37] and gives ability to differentiate between unilateral and bilateral losses and hyposmia or anosmia.

All of these psychophysical olfactory tests can be used to assess a patient's olfactory ability before and after treatment and to follow the progression of the disease.

Electrophysiologic tests

Electrophysiologic tests such as electro-olfactogram (EOG) or odor event–related potentials (OERPs) are used for research purposes only.

The EOG is measured from an electrode placed on the surface of the human olfactory epithelium [38]. Because no local anesthetic can be used, the placement of the electrode with endoscopic visualization is uncomfortable for a patient and can cause sneezing and mucous discharge. Sometimes, despite correct placement of the electrode, the EOG cannot be recorded, which may be related to topographic distribution of the specific olfactory receptors and age-related metaplasia of the olfactory neuroepithelium [39,40]. These limitations make recording of an EOG difficult.

OERPs are recorded from the surface of the scalp and derived from overall electroencephalographic activity after presentation of odorants. The changes in amplitude and latency of the OERPs can reflect airway obstruction or dysfunction anywhere along the afferent pathway. The

quality of the recording depends on the alertness of the subject. OERPs can be useful in detecting malingering.

Functional MRI also is used by researchers to detect areas of brain activity in response to odorant presentation [41]. In rare circumstances, biopsy of the olfactory neuroepithelium is performed in a research setting. This typically requires multiple biopsies to obtain true olfactory neuroepithelium because of age-related metaplasia of the respiratory-like epithelium within the region of olfactory neuroepithelium [42,43].

Gustatory system

Disorders of taste: etiology

True loss of taste is extremely rare. Most patients who complain of a loss of taste actually suffer from olfactory dysfunction, with inability to perceive the flavors of food. Patients with a true taste loss, however, often complain bitterly of their problems. Pathology along each segment of the gustatory pathway might lead to the alteration of taste: partial loss (hypogeusia), complete loss (ageusia), or a sensation of altered taste (dysgeusia). Taste distortion with stimulus (parageusia) or without stimulus (phantgeusia) also can occur. Viral, bacterial, fungal, or parasitic infections of the oral and hypopharyngeal mucosa may disturb taste. Radiation-induced mucositis also can impair taste sensation, and poor oral hygiene can cause loss or distortion of taste. The aging process adversely affects the sense of taste, but to a lesser degree than the sense of smell. Bitter and sour tastes are most likely to diminish with age, but the loss is generally only partial [44]. Neoplastic involvement of the floor of the mouth, submandibular space, or infratemporal lesion may produce a loss of taste. Smoking may have an adverse affect on taste sensitivity and discrimination.

Injury to the chorda tympani also can occur secondary to ear surgery or infections such as chronic otitis media, Bell's palsy, Ramsay Hunt syndrome, or Lyme disease. Patients with chorda tympani injuries complain of phantom taste (metallic, sometimes salty or bitter) more frequently than loss of taste [45]. Trauma to the lingual or pharyngeal branches of cranial nerve IX during tonsillectomy or uvulopalatopharyngoplasty also can result in the taste dysfunction. Pathologic involvement of cranial nerves IX and X in glomus tumors, squamous cell carcinoma, or schwannoma with other neurologic deficits will produce a loss of taste. Head trauma rarely causes a loss of taste, with a reported incidence of 0.4% to 0.5% [46].

Some systemic diseases have been associated with a loss of smell or phantom smell. Renal disease may produce a phantom taste (metallic or bitter), likely secondary to accumulation of uremic toxins because improvement occurs after dialysis [47]. Patients who have diabetes mellitus may experience taste disturbances as a consequence of diabetes-related

neuropathies. Many medications have been associated with distortion or loss of taste, but few have been evaluated with clinical studies [25,48].

Evaluation of taste

Patients who complain of decreased ability to perceive food flavor or who experience ''unpleasant taste'' likely are suffering from olfactory dysfunction. Evaluation of chemosensory ability should assess the sense of smell and the quality and intensity of taste. The taste qualities can be affected in different degrees and each needs to be tested. Typically, the following stimuli are used to test four taste qualities: sodium chloride (salty), sucrose (sweet), citric acid (sour), and quinine hydrochloride or coffee (bitter). To assess taste intensities, patients are used as their own control and the differences between the right and the left sides of the mouth are evaluated [48].

The evaluation of taste disorders measures detection or recognition thresholds. The tests are extremely variable and changes in threshold detection do not indicate necessarily correlation to changes in suprathreshold taste intensity. Generally two testing methods are used: magnitude matching and spatial testing.

Magnitude matching

Magnitude matching involves suprathreshold testing using one sensory modality that is normal (eg, hearing) compared with deficiency in another sensory modality (eg, taste) [49]. Several concentrations of sodium chloride, sucrose, citric acid, and quinine hydrochloride acid are presented along with several loudness levels at 1000 Hz. The patient tastes each solution and at the same time the tones are presented through headphones. The patient estimates the perceived magnitude for each stimulus. The results are scored in relation to the loudness function. Patients with hypogeusia associate stronger taste concentration with weaker tones than normal patients. This test depends on patients having normal hearing.

Spatial test

Taste function in various areas of the tongue and oral cavity can be measured using a spatial test. Four standardized sizes of filter paper soaked with a strong concentration of four basic tastes are placed randomly on the four quadrants of the tongue and both sides of the soft palate. Cotton-tipped swabs soaked with basic tastes also can be used as an applicator (Fig. 3). Patients then identify the quality of the taste and rate the intensity with a number from 1 to 10, with 10 being the strongest. The variation of this test is the three-drop technique, which involves placing three drops of a given chemical on a random portion of the tongue. The concentration is titrated to obtain the threshold of sensitivity. Recently, several tests have been developed in Germany that resemble this technique but are more standardized in the form of ''taste strips,'' wafers, or tablets with specific

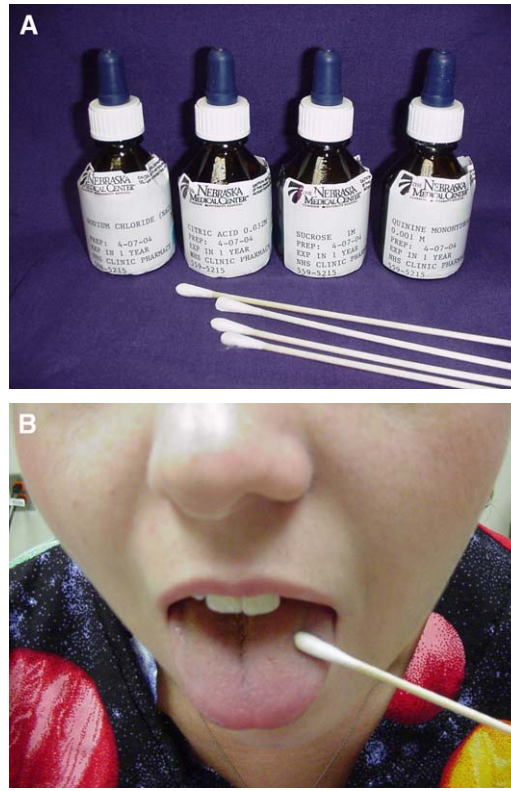

Fig. 3. (*A*,*B*) Spatial gustatory testing.

concentration of the stimuli [50–52]. Some allow testing of the whole mouth, whereas others facilitate testing of each side of the tongue separately.

Another important factor to determine in gustatory testing is the differentiation between the genuine stimulus and the phantom taste. Patients are asked to rinse their mouth with water. Elimination of the unpleasant taste suggests that the stimulus is real. Then, if the spatial testing reveals a normal taste system, this additionally suggests the presence of the genuine tastant. Also, topical anesthesia applied to the tongue or mouth can eliminate the taste caused by a genuine tastant or peripheral taste dysfunction. The source of the genuine tastant could be saliva, laryngopharyngeal reflux, or sinonasal drainage. Taste phantoms can intensify after the topical anesthesia has been applied to the mouth, such as in a patient complaining of the metallic taste after ear surgery with injury to the chorda tympani.

Electrogustometry

Electrogustometry is based on a weak electrical stimulus producing a sour taste when applied to the taste receptors. This method has several strengths [53], such as the ability to provide quantitative control of the

intensity of stimulation, the short time required for testing, and ease of administration, but electrogustometry is inappropriate for evaluation of taste qualities other than sour.

Available spatial and magnitude matching tests of gustatory function are subjective and unable to detect psychogenic disease or malingering. From the objective modalities that have been investigated, gustatory-evoked potentials (GEPs) have been used most widely, but the techniques developed to date for measuring GEPs are not yet clinically useful. Problems remain in difficulty of interpreting the test and distinguishing between normal and abnormal GEPs patterns, and lack of agreement among investigators regarding the waveform [54,55].

Summary

Clinical assessment of the patient who has smell and taste disorders requires understanding of the etiology of the olfactory and gustatory disorders. Available clinical tests of olfactory and gustatory systems allow detecting and measuring the degree of the sensory loss, but are unable to determine the cause and give neither prognostic information nor therapeutic guidance. With physical examination, however, clinical history can help to establish the diagnosis and guide the treatment if available. A clinician evaluating a patient who has smell and taste loss must understand that "taste" complaints usually are symptoms of an olfactory dysfunction. The distinction between true gustatory loss (bitter, sweet, salty, sour, or umami) and olfactory loss, the inability to perceive complex flavors of food, will help clarify the patient's diagnosis. Easy-to-administer tests are available for olfactory (eg, UPSIT) and gustatory testing (eg, spatial tests, taste sticks, tasting tablets). In rare circumstances, imaging studies (eg, MRI, CT) are indicated.

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