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內文：

### Introduction

Taste (or gustation) has long been regarded as a minor sense, less important even than its chemosensory cousin, smell (or olfaction). It has received much less medical and clinical research attention than smell. However, when disruptions do occur, they can have a substantial impact on nurture and quality of life.

Dental practitioners are often the first clinicians to be presented with complaints about changes in taste. This raises a problem in terms of appropriate evaluative response. Taste complaints generally take one of two forms. Either the patient complains of diminished or lost taste perception (hypogeusia or ageusia) or of the presence of a persistent, unpleasant taste sensation (phantogeusia), frequently in conjunction with distortions in taste quality (dysgeusia) and/or burning mouth symptoms (BMS). The first thing that must be determined in the case of diminished perception is whether the complaint reflects a true taste loss or a smell loss that impacts on food flavor perception.

### Taste vs smell: relative vulnerabilities

It was recognized well over a 100 years ago that true taste loss is rare, whereas loss of smell is more common. Studies from modern chemosensory clinics have confirmed this observation. For example, both the University of Pennsylvania Smell & Taste Clinic and the Monell-Jefferson Taste & Smell Clinic have reported that while close to 70% of patients presenting with a complaint of taste loss evidenced smell loss, fewer than 10% evidenced measurable taste loss.

- Olfaction depends on a single cranial nerve (I), while multiple branches of three cranial nerves (VII, IX, and X) carry gustatory information. Moreover, the olfactory nerve is located in a vulnerable position in that its axons must pass through the cribriform plate of the ethmoid bone prior to dissemination on the surface of the olfactory bulb. As a consequence, they are subject to the coup contra coup forces associated with head injury that can lead to tearing or severing of the axonal processes.
- Olfactory receptors are highly localized in a small patch of tissue high in the nasal cavity, rendering them vulnerable to changes in nasal patency or airflow patterns that might limit the access of stimulus molecules. In contrast, taste receptors are found on a large portion of the tongue dorsum, as well as on the soft palate, larynx, pharynx, and epiglottis.
- Both systems are subject to a barrage of potentially toxic chemical stimuli, although both have regenerative capacity. However, in the case of the olfactory system, in which the receptor cells are primary neurons, this requires reinnervation of the olfactory bulb. In contrast, receptor cells in the gustatory system are modified epithelial cells that, although they have some neuronal characteristics, can turn over more rapidly.

### Nature and assessment of taste dysfunction

As noted, taste loss is relatively rare, despite the frequency of patient complaint. A more common true taste disorder is a distortion in taste perception, most often taking

the form of a persistent unpleasant taste in the oral cavity (phantoguesia), sometimes accompanied by burning sensations. Primary distortions in the perceived qualities of taste stimuli (e.g., sweet stimuli eliciting a bitter taste) may also occur rarely.

Taste loss can be assessed via chemical (threshold or suprathreshold) or electrogustometric measures. Because of both the (largely) independent innervation of taste receptor fields in the oral cavity (tongue / palate / pharynx, left / right, and anterior / posterior tongue) and the unique receptors for the basic tastes as well as unique taste receptor cells that express them, taste loss can, in principle, be both regional and quality specific.

- Clinical centers in the United States have relied primarily on whole-mouth assessments of responses to the four traditional basic tastes (sweet, salty, sour, and bitter) supplemented with regional testing. However, testing is idiosyncratic, and widely accepted norms have not been developed.
- A test using taste identification of chemical stimuli presented via taste strips on the anterior tongue has recently been proposed as a diagnostic tool in taste dysfunction. However, the proposed measure does not distinguish either quality specific losses or spatial losses other than anterior tongue right / left. It is also unclear if the measure can identify anything other than ageusia, and there has been no attempt to relate diagnostic results to those obtained via whole-mouth testing.
- Electrogustometric measurement offers a seemingly simple solution for taste testing. However, it is limited in terms of quality specificity, and has not been widely used in the U.S. as a primary diagnostic tool, so again norms are lacking.
- There are no specific measurement techniques to objectively validate or quantify phantom taste complaints.

The clinician should bear in mind, however, that this is not the patient's fault, and does not invalidate his/her complaint.

### **Etiologies**

Detailed reports of the etiologic factors contributing to taste dysfunction in patients seen in chemosensory clinics are not available. More often than not, there appear to be no clear precipitating events or identifiable underlying pathology.

- Head trauma and upper respiratory viral infections may in some cases contribute to these (as well as to taste distortions and phantoms) but the underlying pathophysiology is still not completely understood.
- It can be argued that the single most common etiologic factor contributing to taste dysfunction is medication usage. This may be the result of the direct impact of medications on taste receptor function or of residual tastes associated with either the drug's presence in saliva or in the blood, since tastes can be perceived intravascularly.
- The principal nutrient deficiency that has been associated with taste loss is zinc. Some controlled studies of documented zinc deficiency in specific disease states do indicate it may be associated with taste loss that reverses with zinc supplementation, although the mechanisms by which zinc affects gustatory function are still uncertain.
- Poor oral hygiene, periodontal disease or changes in oral hygiene regimens are obvious potential sources of phantoguesias.
- The overgrowth of oral *Candida*, which may be associated with xerostomia, with the use of dentures, antibiotics or corticosteroids, or with immunological deficiencies or diabetes, may give rise to phantom taste and oral burning sensations even in the absence of objective manifestation, that is, without

- clinically evident thrush or angular cheilitis.
- Gastroesophageal reflux disease (GERD) can produce apparent phantom' taste sensations, which may be intermittent or persistent and are most often described as sour. This is also often associated with dental erosion, particularly of the posterior teeth.
  - Two common surgical procedures, one of particular relevance to the dental practitioner, may result in damage to the chorda tympani (CT) nerve, which mediates taste perception on the anterior tongue, leading to complaints of both loss and phantoms. Middle ear surgery may require stretching or severing it, resulting in the loss or diminution of taste sensation on one or both (if the surgery is bilateral) anterior quadrants of the tongue. The lingual branch of mandibular nerve is vulnerable to damage during third molar extraction, again resulting in localized taste dysfunction.
  - Much more rarely, CT-lingual damage may result from mandibular block analgesia, perhaps particularly inferior alveolar nerve block.
  - Phantogeusias may also be associated with depression, although the bases for and significance of this symptom in depressed patients are unclear. It should be borne in mind that psychological morbidity associated with persistent unpleasant tastes, and/or BMS, may be the result and not the cause of the symptoms.
  - Aging or factors associated with aging may render individuals more vulnerable to taste dysfunction. In a chemosensory clinic population, Cowart et al. (1997) found that elderly patients (>65 years) were significantly more likely than young or middle-aged patients to report phantogeusia and to evidence diminished taste.

#### **Practical guidelines for assessment and referral**

A patient complaining of diminished taste perception should first be assessed for olfactory function using one of the standardized tests that are now commercially available. If the patient is found to have an olfactory problem, he / she should be referred to an otorhinolaryngologist sub-specializing in diseases of the nose and sinuses.

- A thorough oral exam should be performed, including assessment of possible abnormalities in the microbial flora of the oral cavity. An empirical trial with oral antifungal agents, for example, clotrimazole troches, may be appropriate.
- A detailed consideration of changes in medications and oral health procedures (e.g., types of toothpaste and oral rinses used) should also be undertaken.
- Referral to a gastroenterologist should be considered to rule out the possible contribution of GERD to the persistent taste, particularly when there is evidence of dental erosion.
- In cases in which there is a suspicion of iatrogenic damage to the CT, microsurgical repair may be possible.
- The practitioner should also be sensitive to the patient's psychological state. Depression may be the result of a taste problem or contribute to a taste complaint. In either case, referral for psychological counseling should be considered, although not as a first step.

The patient should be reassured that although persistent taste symptoms are difficult to live with, taste is a resilient system. It appears that taste loss after traumatic head injury is more likely to recover than smell loss. In addition, two-thirds of patients with dysgeusias have been reported to experience spontaneous resolution of symptoms within an average of 10 months.

#### **Conclusion**

Taste complaints present a number of difficulties to the oral medicine practitioner;

clinicians should be attuned to these issues, and be prepared to make appropriate evaluations and referrals.

<b>題號</b>	<b>題目</b>																												
<b>1</b>	下列敘述何者錯誤？ <b>(A)</b> 牙周病或口腔內白色念珠菌感染可能造成味覺異常或是減弱。 <b>(B)</b> 偏頭痛、貝爾氏麻痺(Bell's palsy)或是帶狀皰疹(herpes zoster)可能引起味覺幻覺(taste hallucinations)。 <b>(C)</b> 味覺異常(dysgeusia)比嗅覺喪失(anosmia)常見。 <b>(D)</b> 腦幹缺血或是阻塞可能造成同側的半邊舌頭味覺喪失(hemiageusia)。																												
<b>答案</b> <b>(C)</b>	味覺異常(dysgeusia)比嗅覺減弱(hyposmia)及喪失(anosmia)少見。 <b>出處：</b> Oral and Maxillofacial Pathology 2 <sup>nd</sup> edition, P.753-754																												
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<b>2</b>	下列哪些藥物可能造成味覺改變？ ①抗癲癇藥物(anticonvulsant)②抗高血壓藥物(antihypertensive)③血管擴張劑(vasodilator)④抗組織胺藥物(antihistamine) <b>(A)</b> ①②③ <b>(B)</b> ①②④ <b>(C)</b> ①③④ <b>(D)</b> ①②③④																												
<b>答案</b> <b>(D)</b>	可能造成味覺改變的藥物有： <table border="1"> <tr> <td>Anticoagulant</td> <td>Phenindione</td> </tr> <tr> <td>Antihistamine</td> <td>Chlorpheniramine maleate</td> </tr> <tr> <td>Antihypertensive or diuretic</td> <td>Captopril, diazoxide, ethacrynic acid</td> </tr> <tr> <td>Antimicrobial</td> <td>Amphotericin B, ampicillin, griseofulvin, idoxuridine, lincomycin, metronidazole, streptomycin, tetracycline, tyrothricin</td> </tr> <tr> <td>Antineoplastic or immunosuppressant</td> <td>Doxorubicin, methotrexate, vincristine, azathioprine, carmustine</td> </tr> <tr> <td>Antiparkinsonian agent</td> <td>Baclofen, chlormezanone, levodopa</td> </tr> <tr> <td>Antipsychotic or anticonvulsant</td> <td>Carbamazepine, lithium, phenytoin</td> </tr> <tr> <td>Antirheumatic</td> <td>Allopurinol, colchicine, gold, levamisole, penicillamine, phenylbutane</td> </tr> <tr> <td>Antiseptic</td> <td>Hexatidine, and chlorhexidine</td> </tr> <tr> <td>Antithyroid agent</td> <td>Carbimazole, methimazole, thiouracil</td> </tr> <tr> <td>Hypoglycemic</td> <td>Glipizide, phenformin</td> </tr> <tr> <td>Opiate</td> <td>Codeine, morphine</td> </tr> <tr> <td>Sympathomimetic</td> <td>Amphetamines, phenmetrazine</td> </tr> <tr> <td>Vasodilator</td> <td>Oxyfedrine, bamifylline</td> </tr> </table> <b>出處：</b> Oral and Maxillofacial Pathology 2 <sup>nd</sup> edition, P.754	Anticoagulant	Phenindione	Antihistamine	Chlorpheniramine maleate	Antihypertensive or diuretic	Captopril, diazoxide, ethacrynic acid	Antimicrobial	Amphotericin B, ampicillin, griseofulvin, idoxuridine, lincomycin, metronidazole, streptomycin, tetracycline, tyrothricin	Antineoplastic or immunosuppressant	Doxorubicin, methotrexate, vincristine, azathioprine, carmustine	Antiparkinsonian agent	Baclofen, chlormezanone, levodopa	Antipsychotic or anticonvulsant	Carbamazepine, lithium, phenytoin	Antirheumatic	Allopurinol, colchicine, gold, levamisole, penicillamine, phenylbutane	Antiseptic	Hexatidine, and chlorhexidine	Antithyroid agent	Carbimazole, methimazole, thiouracil	Hypoglycemic	Glipizide, phenformin	Opiate	Codeine, morphine	Sympathomimetic	Amphetamines, phenmetrazine	Vasodilator	Oxyfedrine, bamifylline
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