ORIGINAL RESEARCH PAPER

Oral Medicine

TONGUE DISORDERS-A REVIEW

KEY WORDS:

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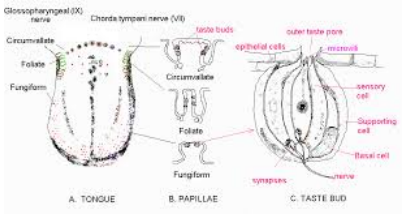
ABSTRACT

Taste alterations or disorder might not be pleasing for patients which thereby pose a serious threat to the health of older and more vulnerable patients, who can become malnourished through a loss of taste or changes in taste perception. Correct diagnosis of taste loss is the first step in the treatment of taste abnormalities and the avoidance of unnecessary dental treatment. It is therefore helpful for both patient and health care provider to be aware of the causes of taste changes.

INTRODUCTION

Taste results from the sensation born in the taste buds, i.e. in the cruceiform, foliate and fungiform gustative papillae found on the tongue.

The sensation is then transmitted, depending on the stimulation site, either via the chorda tympani to the facial nerve or to the glossopharyngial nerve, and finally to the upper branch of the vagus nerve[1].



CLASSIFICATION OF TASTE DISORDERS

Physiological	Atrophy of oral epithelium Increase degrees of atrophy and fibrosis of acini of the salivary gland Reduction of taste buds
Diseases	Local: Dental Caries, periodontal diseases, candidosis, erythema migrans, fissure tongue, glossitis, dermatoses, denture faults Systemic diseases: Stroke, Alzheimer's disease, Parkinson's disease, Depression, Diabetes mellitus types 1 and 2, Hypo/hyperthyroidism,Chronic renal failure, Acute and chronic liver disease, cirrhosis, Sjögren's syndrome, irritable bowel syndrome, Gastroesophageal reflux disease, Hiatus hernia, Inflammatory bowel diseases, HIV
Iatrogenic	Drugs Chemotherapy/radiotherapy
Nutrition deficiencies	Iron deficiency, Folate/Vitamin B12deficiency, Zinc deficiency
Allergy	Food and additives allergy

Gustatory-Olfactory Confusions in Patients with Hyposmia:

Many individuals experience smell and taste loss or report that particular foods no longer taste the same. Most of these problems are transient and associated with decreased transport of volatile substances to the olfactory receptors as a result of heavy nasal mucus or swelling of the sinus mucosa. Such symptoms may last longer if the episode is associated with viral damage to the olfactory receptors or a more permanent lesion of the ethmoidal sinus. In either case, due to the common use of "taste of food" as a synonym for food flavor (i.e a combination of gustatory, olfactory, touch and other sensations'F), loss of olfactory function alone may be reported as taste loss or altered taste. In many cases, patients experiencing this phenomenon are found on testing to have decreased olfactory function (hyposmia, anosmia) and normal taste responses.[2]

Surveys of chemosensory loss in individuals exposed to industrial dusts and air contaminants, such as chromium, lead, mercury, nickel, silver, and zinc, in some cases describe symptoms of taste dysfunction and olfactory loss

SECONDARY DYSGEUSIAS AND PARAGEUSIAS:

The sensation of taste in humans is largely concerned with substances introduced into the mouth (rather than those contacted by the extruded tongue), and an unusual or unpleasant taste that is not directly 'associated with ingestion or introduction of some object into the mouth is usually perceived as abnormal and if it persists, as evidence of disease. Substances with unpleasant or unusual tastes are produced in the mouth as the result of bacterial ferrmentation in dental plaque and the gingival crevice but are certainly not restricted to this source.[3]

Many medications and natural and artificial ingredients in processed foods, such as garlic, saccharin, and cyclamate, and possibly other flavor enhancers can be the source of after tastes and an often unsuspected source of some stimulated dysgeusias.[4]

TRANSPORT DISORDERS

SALIVARY HYPOFUNCTION AND XEROSTOMIA

Taste bud cell function is dependent on specific soluble molecules gaining access to the cell membrane, followed by transport of the molecule across the membrane. In patients with greatly diminished salivary flow, the concentration of taste stimuli and salivary electrolytes reaching the cell membrane may be greatly reduced unless the stimulus is provided in liquid form. This phenomenon probably underlies the loss of taste reported by many patients with xerostomia, regardless of the cause (Sjogren's syndrome.Y" 220 nonspecific sialadenitis, therapeutic radiation, although the taste bud cells themselves are damaged by radiation and also may undergo regressive changes with extended drying of the tongue dorsum). These changes may not be apparent with short-term drug induced salivary hypofunction.' which by contrast, has minimal effect on taste thresholds.

BLOCKING OF TASTE BUD PORES BY MICROO RGANISMS OR DENTURES

Salivary hypofunction and radiotherapy to the head and neck also are often accompanied by overgrowth of oral bacteria and yeasts, and electron micrographic illustrations of taste bud pores blocked by microbial elements and other debri suggest an additional mechanism for taste loss in these patients. A proportion of the palatal taste receptors also may be occluded by an upper denture, although the gag reflex usually prevents extension of the palatal surface of the denture over the entire palatal taste bud field, and it is unlikely

that blocking of a few taste receptors would significantly reduce taste sensitivity. More than 10% of edentulous patients may complain of some degree of taste loss when they initially wear an upper denture but this is more likely due to the reduced oral food-handling time documented in denture wearers, diminished touch and pressure sensations, and retention of food particles and bacterial plaque on the surfaces of the denture.[5]

DAMAGE TO TASTE NERVES (LINGUAL V, CHORDA TYMPANI, AND GLOSSOPHARYNGEAL) LESIONS OF THE LINGUAL NERVE

Lesions of the lingual nerve usually result from surgical trauma to the nerve as it passes from the retromolar space along the lingual aspect of the mandible below the mylohyoid ridge and across to the ventral aspect of the tongue. Such trauma may be a complication of regional blocking of the nerve, extraction of mandibular molar teeth, jaw fracture, a stone in the submaxillary gland duct; ' probing or removing such stones, or accidental laceration of the ventral surface: of the tongue during dental restorative treatment. Suggestions that the relative numbers of fungiform papillae on the contralateral versus ipsilateral sides of the tongue can be used as a measure of the severity of or prognosis for recovery from lingual nerve damage are probably unfounded and difficult to interpret given the variation in the distribution of fungiform papillae on apparently normal tongues. Like the unilateral numbness and burning sensations that follow lingual nerve damage, the altered gustatory function that accompanies neuropathic changes to fibers of the chorda tympani contained within the damaged lingual nerve, should be unilateral, restricted to one half of the anterior dorsal surface and temporally related to the nerve injury. In the majority of cases, unilateral loss of general somatic sensation on the tongue secondary to lingual trauma of this type resolves within 6 months to 1 year.

LESIONS OF THE CHORDA TYMPANI NERVE

Lesions of the chorda tympani often follow middle 'ear surgery because the nerve passes immediately behind the eardrum in the middle ear and : must be tympani and produce unilateral anterior third dysgeusia. In patients with Bell's palsy and other diseases affecting the seventh nerve, the lesion may be sufficiently proximal to affect the chorda tyrnpani. Dysfunction of salivation (and lacrimation) on the affected side also may accompany damage to the seventh nerve.

Administration of systemic corticosteroids is an important component of treatment of Bell's palsy and probably hastens recovery if given within 5 days of onset of symptoms. Anastomosis of the damaged portions of the facial nerve may be performed when spontaneous recovery of an extra cranial neuropathy of the seventh nerve docs not occur. Such operations are frequently successful in reestablishing gustatory, motor. and autonomic function in the nerve.[6]

LESIONS OF THE GLOSSOPHARYNGEAL NERVE

Lesions of the extracranial portion of the glossopharyngeal nerve are unusual, but damage to this nerve may result from removal of pharyngeal tumors and repeated tonsillectomy or cauterization of the tonsillar bed.

Transient dysgeusia, in fact,' often follows tonsillectomy. Dysgeusia restricted to one side of the posterior two thirds of the tongue is an unusual but not impossible presenting complaint for a neoplasm arising in or invading the lateral pharyngeal wall. Loss of gag reflex on the same side usually accompanies these symptoms. Section of the nerve for intractable glossopharyngeal neuralgia abolishes taste sensation on the posterior lingual surface, palate, and pharynx on the affected side.

Extracranial lesions of the vagus nerve do not cause dysgeusia, although motor abnormality (paralysis of the soft palate with deviation of the uvula to the opposite side on speaking) may be detected on oral examination, and dysphagia may be present. Intracranial lesions affecting the roots of the vagus nerve' may cause some dysgeusia because it is likely that some of the special sensory fibers of the ninth nerve enter the medulla by means of anastomosis with the roots of the 10th nerve. Cranial nerves VII and IX also may be affected by diphtheritic neuropathy caused by spread of toxin from the pharyngeal infection in nonimmunized individuals.

INTRACRANIAL LESIONS OF NERVES VII, IX, AND X :

Cranial nerves VII, IX, and X may be affected by neoplasms (metastatic carcinoma, meningioma, glioma, neurofibroma) and by pressure from aberrantarteries , aneurysms, granulomas, and infectious processes. Symptoms of dysgeusia appear together with various motor and sensory abnormalities characteristic of the nerve 'affected. 'Because of the close anatomic association of these three nerves and the roots of cranial nerves V, VI, and VIII, multiple cranial neuropathy is the usual feature of many of the syndromes that .result from intracranial lesions in the region of the pons and brain stem. The roots of these nerves also may be damaged during surgical treatment of other intracranial lesions or head trauma (q. v.). The facial nerve in its intracranial course is most commonly affected by metastatic carcinoma, meningitis, Paget's disease of the temporal bone, or aneurysms.

METABOLIC DISORDERS

Taste dysfunction has been documented in a variety of metabolic disorders, such as adrenal insufficiency, diabetes mellitus. Hypothyroidism and hyperthyroidism, pseudohypoparathyroidism and obstructive jaundice and hepatitis. Symptoms of taste abnormality are possibly fairly common in these various diseases, although they are rarely the cause of complaints of taste dysfunction among patients referred for evaluation in clinical taste and smell research centers. Routine evaluation of a patient with a taste complaint by an internist will usually detect such underlying problems, even if they are not already suggested by abnormalities in a screening blood chemistry examination.

Exceptions to this are the specific reduction in taste sensitivity for glucose in individuals with impaired glucose tolerance, which has been interpreted as a manifestation of a hereditary defect in systemic glucose recognition the variability of gustatory symptoms and taste thresholds in hypothyroidism and hyperthyroidism; a complaint of taste loss following treatment with antithyroid drugs such as propylthouracil and mcthylrhiouracil the increased intake of salt and lowered taste thresholds associated with adrenal insufficiency'" and specific food aversions and preferences in patients with liver disease, who as a group have been shown to have increased taste thresholds.

PREGNANCY

Many pregnant women experience changes in the way certain foods taste and smell and traditionally develop a variety of food preferences and aversions. Both these phenomena characteristically disappear following delivery. In addition, a hormonally driven physiologic mechanism for increasing salt intake in pregnant women is suggested by their diminished ability to identify correctly different concentrations of salt solution and their increased preference for stronger salt solutions.[7]

CONCLUSION

Sense of taste affords human being to have ability to evaluate what it eats or drinks. At the most basic level, the evaluation is to promote ingestion of nutritious substances and prevent consumption of potential poisons or toxins. Taste perception fades with ageing and there are many disorders which affect

the sensation of taste. Thus, sensation of taste plays vital role in maintaining general wellbeing of an individual.

REFERENCES :

1. Fauci, Anthony S.,et al. Harrison's Principles of Internal Medicine. 17th ed. United States: McGraw-Hill Professional, 2008.
2. Chaudhari, Nirupa& Roper, Stephen D(2010)," The cell biology of taste", Journal of Cell Biology 190(3):285-296.
3. Guyton, Arthur C. (1991) Textbook of Medical Physiology (8thed). Philadelphia: W.B.Saunders.
4. Cowart B J, Young IM, Feldman R.S et al Clinical disorders of smell and taste. Occup Med 1997. 12465- 483.
5. Mistretta CM. Ageing effect on anatomy and neurophysiology of taste and smell. Gerodontology 1984,3243-248.
6. Mavi A, Ceyhan O. Bitter taste thresholds, numbers and diameters of circumvallate papillae and their relation with age in a Turkish population. Gerodontology 1999. 16119-122
7. Greenburg Glick ship. burkitt oral medicine, 9th edition, B.C Decker Inc2008.