<table>
<thead>
<tr>
<th>Disorders of Taste and Smell</th>
<th>Image</th>
</tr>
</thead>
<tbody>
<tr>
<td>4.3.1 <strong>CC</strong></td>
<td>Describe the basic physiology of olfaction.</td>
</tr>
<tr>
<td>4.3.2 <strong>CC</strong></td>
<td>Describe the basic mechanisms of gustatory physiology.</td>
</tr>
<tr>
<td>4.3.3 <strong>CC</strong></td>
<td>What is the mechanism by which individual odorants chemicals are encoded as distinct?</td>
</tr>
<tr>
<td>4.3.4 <strong>CC</strong></td>
<td>Discuss olfactory and taste testing. Discuss the UPSIT and interpret the scoring.</td>
</tr>
<tr>
<td>4.3.5 <strong>SW</strong></td>
<td>What is the alcohol sniff test?</td>
</tr>
<tr>
<td>4.3.6 <strong>SW</strong></td>
<td>What is the differential diagnosis of anosmia?</td>
</tr>
<tr>
<td>4.3.7 <strong>SW</strong></td>
<td>What are the different types of taste receptors, their location, and which kinds of tastes are each responsible for?</td>
</tr>
<tr>
<td>4.3.8 <strong>SW</strong></td>
<td>Define and describe the terms Aparosmia and phantosmia. What are the underlying conditions and how are they managed?</td>
</tr>
</tbody>
</table>
What is the differential diagnosis for dysgeusia and complete loss of taste?

**Gustatory dysfunction**

Much of what is perceived as a taste defect is truly a primary defect in olfaction, which alters flavor. The components that comprise the sensation of flavor include the food's smell, taste, texture, and temperature. Each of these sensory modalities is stimulated independently to produce a distinct flavor when food enters the mouth. Taste may be enhanced by tongue movements, which increase the distribution of the substance over a greater number of taste buds. Adaptation in taste perception exerts a greater influence than in other sensory modalities.

Other than smell dysfunction, the most frequent causes of taste dysfunction are prior URI, head injury, and idiopathic causes, but many other causes can be responsible.

- Lesions at any site from the mucosa, taste buds, unmyelinated nerves, or cranial nerves to the brain stem may impair gustation.

- Oral cavity and mucosal disorders including oral infections, inflammation, and radiation-induced mucositis can impair taste sensation. The site of injury with radiotherapy is probably the microvilli of the taste buds, not the taste buds themselves, since taste buds are thought to be radioresistant.

- Poor oral hygiene is a leading cause of hypogeusia and cacogeusia. Viral, bacterial, fungal, and parasitic infections may lead to taste disturbances because of secondary taste bud involvement.

- Normal aging produces taste loss due to changes in taste cell membranes involving altered function of ion channels and receptors rather than taste bud loss.

- Malignancies of the head and neck, as well as of other sites, are associated with decreased appetite and inability to appreciate flavors.

- Use of dentures or other palatal prostheses may impair sour and bitter perception, and tongue brushing has been shown to decrease taste acuity.

- Surgical manipulation may alter taste permanently or temporarily.
  - Resection of the tongue and/or portions of the oral cavity most commonly for reasons of malignancy decreases number of taste buds.
  - Radiation and chemotherapy damages taste receptors and decreases salivary flow altering taste perception.
  - In otologic surgery, stretching or transection of the chorda tympani nerve may result in temporary dysgeusia. Bilateral injury still may not result in permanent taste dysfunction because of the alternate innervation through the otic ganglion to the geniculate ganglion via the greater superficial petrosal nerve.
Nutritional deficiencies are involved in taste aberrations. Decreased zinc, copper, and nickel levels can correlate with taste alterations. Nutritional deficiencies may be caused by anorexia, malabsorption, and/or increased urinary losses.

Endocrine disorders also are involved in taste and olfactory disorders. Diabetes mellitus, hypogonadism, and pseudohypoparathyroidism may decrease taste sensation, while hypothyroidism and adrenal cortical insufficiency may increase taste sensitivity. Hormonal fluctuations in menstruation and pregnancy also influence taste.

Heredity is involved in some aspects of gustation. The ability to taste phenylthiourea (bitter) and other compounds with an –N-C= group is an autosomal dominant trait. Studies have shown that phenylthiourea tasters detect saccharin, potassium chloride (KCl), and caffeine as more bitter. Type I familial dysautonomia (ie, Riley-Day syndrome) causes severe hypogeusia or ageusia because of the absence of taste bud development.

Direct nerve or CNS damage, as in multiple sclerosis, facial paralysis, and thalamic or uncal lesions, can decrease taste perception.

Many other diseases can affect gustation (eg, lichen planus, aglycogeusia, Sjögren syndrome, renal failure with uremia and dialysis, erythema multiforme, geographic tongue, cirrhosis).

There are two kinds of taste disorders: losses (hypogeusia refers to a partial loss that can be differential with regard to quality or location and ageusia refers to a total loss) and chronic taste sensations that occur in the absence of obvious stimulation (dysgeusia). Some dysgeusias arise within the nervous system and are akin to phantoms in other sensory systems (e.g., phantom limb, tinnitus); we call these taste phantoms. Others reflect the presence of some abnormal substance in the mouth that is perceived by a completely normal taste system.

**Taste Phantoms or Dysgeusia**

The first step in the evaluation of any chronic taste is to ask the patient to describe the quality of the taste. A chronic taste sensation will be described by a taste quality name, that is, the patient will state that the taste is bitter, sweet, and so on. Patients often find chronic olfactory sensations particularly difficult to describe qualitatively and may simply use a hedonic description, such as "unpleasant." Thus, if the patient cannot describe the sensation, it may actually be an olfactory sensation.

**Dysgeusia Resulting from a Genuine Stimulus**

If the patient is tasting a real stimulus, then the clinical task is to determine what the stimulus is, how it is gaining entrance to the mouth, and whether the underlying cause reflects a medical problem. If the stimulus is a tastant, then it should be possible to rinse it out of the mouth with water. Even if the taste reappears quickly, the fact that it could be rinsed away is significant. If an evaluation of the patient's taste system with a spatial test designed to test each cranial nerve (22) shows no losses, this is further evidence for a normal taste system perceiving a genuine tastant. Finally, topical anesthesia of the mouth should abolish a chronic taste due to the presence of a genuine tastant. Several possible sources for a genuine tastant are saliva (many medications make their way into saliva), gingival fluid, reflux, postnasal drip, and microorganisms that are present in the mouth. When the taste is gaining entrance to the mouth via saliva, it can sometimes be exhausted temporarily by chewing gum (chewing increases the rate of salivary flow).

The quality associated with the tastant can be a clue as to how that tastant gained
entrance to the mouth. For example, salty or metallic dysgeusia sensations suggest the possibility that blood is the source of the taste. Blood tastes salty because its NaCl concentration (approximately 0.15 M) is much higher than that of saliva (approximately 0.015 M NaCl). A bitter dysgeusia suggests the presence of small amounts of medication in saliva or gingival fluid.

**Taste Phantoms**

Taste phantoms are associated with damage to the taste system. Thus, a patient complaining of a chronic taste should be evaluated to see if any localized areas of the mouth show taste loss. In some cases, a taste is perceived to arise from an area that is actually devoid of taste. Obviously, such a taste phantom cannot be due to the presence of a genuine tastant.

Topical anesthetics can be used to verify that such a taste phantom originates within the nervous system. The patient is asked to rate the intensity of the phantom, and then the mouth is anesthetized by swishing a topical anesthetic in the mouth for 60 seconds (e.g., 0.5% Dyclone). We recommend that the patient not gargle with the anesthetic because anesthesia of the gag reflex is uncomfortable and unnecessary. After expectoration of the anesthetic, we ask patients to wait for an additional 60 seconds and then rinse thoroughly. We then test with a tastant to ensure that anesthesia is complete and ask the patient to rate the taste phantom at time intervals (1- to 2-minute intervals are adequate to reveal changes). If the patient's phantom fails to be abolished or actually increases, then the dysgeusia obviously must originate from a location central to the taste receptors. In fact, we typically see intensification of nerve-stimulation phantoms when the mouth is topically anesthetized. We interpret this as a release-of-inhibition phenomenon.

We have been able to create taste phantoms by anesthetizing the chorda tympani nerve (8). These phantoms were localized to a normal area that was not anesthetized. There may be an analogue to this in patients. Localized damage to the taste system might produce phantoms that appear to arise from normal areas.

In some cases, patients do not complain of a chronic taste but rather complain that the taste of certain foods has been altered. If the complaint genuinely involves taste, consider the possibility that differential losses among qualities may be involved. For example, some foods and beverages that are sweetened taste unpleasantly bitter when the sweet taste is removed. Thus, it is not surprising that a patient with a specific loss of the ability to taste sweet describes these as bitter.

**Taste Loss or Alteration Produced at Vulnerable Loci in the Taste System**

In the following section, we relate vulnerable loci in the taste system to some of the known pathologies of taste. When the taste system is damaged, the result may be either a taste alteration (usually a loss) or a taste phantom.

**Tongue**

Substances that alter taste are thought to do so because they have effects on the taste membrane. These substances include foods, beverages, toothpaste, mouthwashes, and medications. A variety of taste alterations of this sort have been observed. For example, the detergent in toothpaste alters taste (Fig. 41.2) (18). Compounds in the globe artichoke cause water to taste sweet in some individuals; *Gymnema sylvestre* (found in an Indian plant) can inhibit sweet. One of the most dramatic of these effects is the sweet taste induced by acidic substances after exposure of the tongue to berries of the *Synsepalum dulcificum* plant (called "miracle fruit"). Adaptation effects also alter taste. For example, adaptation to sweet taste can make water taste bitter.

**Venous Taste**

Venous taste is a phenomenon that occurs as the result of stimulation of receptor sites that are on the bottom of the taste cell (i.e., below the microvilli). A sweet taste is perceived in about 13.5 seconds after saccharin is injected into a vein. Similarly, dehydrocholic acid will produce a bitter taste. This may be the origin of taste sensations reported during chemotherapy.

**Peripheral Nerves**

**Chorda Tympani**

The chorda tympani nerve is most commonly involved in taste disorders and is identified
by taste loss on the anterior two thirds of the tongue. The chorda tympani leaves the
tongue with the lingual nerve (CN V), and the two travel through the pterygomandibular
space. The inferior alveolar nerve, which conveys pain from the lower teeth, passes
through the same space; thus, dental anesthesia directed into this space can injure the
chorda tympani. In addition, the chorda tympani can be damaged by third molar
extractions.
Acute or chronic otitis media is the most common pathology of the middle ear. Although
this source of taste damage was well known in the 19th century, it has only recently been
recognized as a modern source of damage to the taste system. The viral involvement of
the facial nerve that can occur in such disorders as Bell palsy or Ramsey Hunt syndrome
can also be associated with taste dysfunction and unilateral facial paralysis.
Middle ear or mastoid surgery, especially with facial recess exposure, can produce either
temporary or permanent taste loss. Bull (23) evaluated 126 cases in which the chorda
tympani nerve was cut in the course of stapedectomy. Cutting the nerve rendered the
ipsilateral anterior tongue completely devoid of taste.

When the chorda tympani is severed peripheral to the cell bodies in the geniculate
ganglion, Wallerian degeneration would be expected to move from the site of damage
toward the periphery. However, the effects on taste buds vary by species. In humans, the
lingual nerve appears to sustain fungiform papillae so the tongues of individuals in whom
the chorda tympani has been severed do not look abnormal (Janjua and Schwartz,
unpublished observations).

Infectious processes in the oral cavity rarely produce taste loss, but neoplastic
involvement of the floor of the mouth, submandibular space, or an infratemporal fossa
lesion may produce such symptoms. A more noticeable symptom of disease in these
regions would be tongue
numbness because the lingual nerve would also be involved. Isolated unilateral tongue
numbness in the absence of taste loss indicates a trigeminal nerve lesion. In the absence
of oral cavity pathology and tongue numbness, involvement of the chorda tympani in the
temporal bone or the nervus intermedius in the cerebellopontine angle must be
considered.

**Nervus Intermedius**
The nervus intermedius is central to the cell bodies in the geniculate ganglion. Thus, if
this nerve is cut, Wallerian degeneration would be expected to move from the cut toward
the central nervous system, not transganglionically toward the periphery. Neoplastic
processes such as acoustic neuroma, meningioma, or facial nerve neuroma are the most
common causes of nervus intermedius dysfunction. Lack of transganglionic degeneration
is supported by examining patients after unilateral acoustic tumor surgery in which there
were no differences in number of fungiform papillae or number of taste pores per
fungiform papillae between the operated and unoperated sides.

**Glossopharyngeal Nerve**
Trauma to the lingual or pharyngeal branches of CN IX can occur after tonsillectomy,
uvulopalatopharyngoplasty, or any insertion of a deep mouth gag in which the base of
masses in the high jugular space (carotid body tumors, squamous cell carcinoma, deep
neck abscess) may alter CN IX function. Following CN IX more cephalad, lesions directly
involving the skull base at the jugular foramen (e.g., glomus jugulare, schwannoma,
squamous cell carcinoma) will compromise taste function and will be associated with other
cranial nerve neuropathies. Vernet syndrome occurs when jugular foramen lesions
paralyze all nerves that traverse the jugular foramen (CN IX, CN X, and CN XI). Along
with taste disturbance, there is the loss of sensation in the distribution of CN IX and X,
paralyzed ipsilateral pharyngeal wall and vocal cord, and ipsilateral shoulder drop. When
a tumor extends below the skull base or deeper into the foramen magnum, CN XII may
also be involved, causing ipsilateral tongue paralysis and occasional fasciculations. This is
termed Collet syndrome. Villaret syndrome involves a lesion extending out of the jugular
foramen, which causes sympathetic trunk compromise and CN IX to CN XII paralysis.
Such patients exhibit ptosis, miosis, and occasionally enophthalmos. Schwannomas
especially can begin intracranially in the cerebellopontine angle to produce similar
symptoms and nerve dysfunction.

**Central Nervous System**
Unilateral pontine hemorrhage and unilateral damage to the rostral insular cortex give
rise to unilateral taste loss on the same side as the injury. These observations played an important role in the conclusion noted previously that taste projects ipsilaterally. Taste loss from head trauma is often said to be rare (0.4% to 0.5%). However, the incidence may be much higher than this because head trauma can produce spatially discrete taste losses that go unnoticed for the reasons discussed previously. These taste losses are probably the result of a variety of types of damage, but little is known at this time.

**Cumulative Taste Loss**

It is important to note that damage to a localized part of the taste system may occasionally lead to taste loss that is much more severe than the damage would seem to warrant. This would be expected as a result of the fact that a considerable amount of damage can occur without any changes in the everyday taste experience of the patient (see “Interactions Within the Nervous System”). For example, on rare occasions, tonsillectomy produces a virtually total taste loss even though only CN IX is especially vulnerable during surgery. In such cases, the patient might have had severe CN VII damage (e.g., as a result of chronic otitis media) before surgery but not have noticed it because of the release-of-inhibition phenomenon discussed previously. Damage to CN IX during surgery would then leave the patient with no reserves, and the taste loss experienced would be much more extensive than would seem to be accounted for by surgery alone.

**Phantoms from Damage to Peripheral Structures**

The mechanisms by which damage to a structure produces phantoms are not fully understood. We present here an overview about taste phantoms that we have developed as a result of experiments with anesthesia and observations of patients. We note that phantoms associated with damage to sensory nerves provide a paradox. Why would nerve injury that blocks conduction increase rather than decrease sensation? If we substitute taste for pain, we find ourselves in complete agreement. We cannot explain this paradox, but we will document its consequences for patients.

**Peripheral Nerves**

**Chorda Tympani**

As we noted previously, despite the fact that Bull’s (23) patients lost taste in the area innervated by the cut chorda tympani, their most common complaint was a metallic taste, less frequently described as a bitter or salty taste. Over half of these phantoms were not localized to the area innervated by the severed chorda tympani but rather were felt all over the mouth or just on the tip of the tongue. The remaining phantoms were localized to the side of the mouth on which the chorda tympani was cut. Bull (23) noted that in 100 patients in whom the nerve had been stretched rather than cut, the symptoms were less noteworthy, but metallic phantoms still occurred. Bitter and metallic phantom tastes have been reported when the chorda tympani nerve was stretched as a result of dislodgement of ossicular replacement prostheses. Washing the ear canal has been reported to produce a metallic taste phantom and touch sensations in some subjects.

**Nervus Intermedius**

The nervus intermedius often must be sacrificed to permit the removal of an acoustic neuroma. Phantoms (usually salty) have been reported by 13 of 26 patients we interviewed after acoustic neuroma surgery. In patients available for tests, the phantoms could not be rinsed away with water and intensified after anesthesia of the mouth. One of the most interesting features of the taste phantoms associated with surgery for acoustic neuromas is their duration. Patients reported that the phantoms lasted about 6 months and then faded. This is in contrast to the persistence of phantoms noted by some patients whose chorda tympani nerves were cut.

**Glossopharyngeal Nerve**

Loss of function of the circumvallate papillae (CN IX) is rare but can occur after tonsillectomy or surgery to the hypopharynx. Some patients have reported a bitter taste phantom that increases with topical anesthesia. This finding supports the idea that CN VII normally inhibits CN IX.

**Other Effects on the Sense of Taste**
Age
The belief that taste dims with age is common but not supported by a careful review of the psychophysical literature. One of the reasons that this belief survives is because some of the suprathreshold scaling studies have been conducted with methodologies that are open to criticism. In our view, the most conservative summary of this work is as follows. Taste is much less affected by age than is olfaction. Across the standard stimuli, sucrose is the most robust, and citric acid and quinine are the most likely to show loss, but that loss is not great.

Cancer and Cancer Therapies
Early belief that some cancer patients had abnormally low taste thresholds for bitter was invoked to explain meat aversion on the grounds that meat contains bitter compounds that would cause aversions in the supersensitive patient. This result was widely cited but is incorrect. More recent work focused on the effects of chemotherapy and radiation therapy on taste showed that some patients lost taste (often temporarily) and other patients experienced taste phantoms as the result of these therapies.

Medications
Many medications have been associated anecdotally with loss of taste or with taste phantoms (25); however, more work in this area would be valuable because these effects may have important implications for taste physiology and for patient care. When medications are associated with phantom tastes, the clinician should first evaluate the possibility that the medication itself is the source of the taste. Medications taken orally enter the blood and from there may enter crevicular fluid and saliva. In addition, they may be tasted from blood directly (the venous taste phenomenon). Medications used in chemotherapy provide excellent examples. When drugs are tasted in this way, the taste sensitivities of the patient may play a role in the detection of the drug. For example, those most genetically sensitive to bitter taste would be expected to taste more bitterness in medications.

Taste Changes with Disease
Some diseases are believed to affect the sense of taste. The clearest cases for effects on taste occur with renal disease, diabetes, and depression (see reference 26 for reviews). Renal disease has been associated with both taste loss and taste phantoms (metallic, bitter). Improvement occurs with dialysis, which suggests a role for the uremic toxins that accumulate with kidney dysfunction. The metallic/bitter phantoms may reflect the presence of these toxins in the mouth and/or blood. Diabetes causes neuropathies that involve the taste nerves and other nerves. However, work with subjects with a family history of diabetes suggests a deficit specific to glucose in addition to the general taste neuropathy. This work may provide a genetic marker for a predisposition to diabetes. However, it is also of considerable significance for our understanding of sweet receptors. A specific deficit for glucose suggests that the receptor mechanism for glucose must be different from those of other sugars. There is some evidence of reduced taste function in some depressed patients. In addition, there is also some evidence of an association between depression and PROP tasting. The literature on taste disorders and disease is complicated by the use of psychophysical procedures that have come to be viewed with some concern. The older literature used threshold measures almost exclusively, which limits the value of those studies in the modern era.

Additional Effects of Taste Damage
Inhibition in the taste system may serve a more general function than previously suspected. Taste input may inhibit activities incompatible with eating (gagging, nausea, cough, hiccups, etc.). The clinical insights of Berger et al. (27) have played an important role in the development of these ideas. They noted that nausea, cough, and hiccups are among the clinical problems afflicting cancer patients at the end of life. Because cancer patients typically undergo therapies that damage taste, this raises the possibility that these clinical problems have been exacerbated by taste damage. Given that taste also plays a role in preparing the gastrointestinal tract for the arrival of food (e.g., cephalic phase responses), damage to taste may have gastrointestinal consequences as well (28).
What therapeutic interventions exist in the management of persistent dysgeusia?

**Capsaicin: A Novel Way to Treat Oral Pain**

Capsaicin stimulates oral pain, as all chili pepper aficionados know. However, capsaicin also has the unique property of desensitizing pain receptors and has been used as an analgesic for a variety of sources of dermal pain, albeit with mixed success, presumably because skin provides a barrier to capsaicin. Capsaicin desensitization acts as an analgesic on oral mucosal tissue far better than on nonmucosal tissue. Thus, capsaicin desensitization is a useful clinical analgesic for oral pain (24).

**Coping with Taste Disorders**

Burning mouth syndrome is the only taste disorder in which medical management has been effective. Grushka (29) has used clonazepam to reduce oral pain in 70% of patients with burning mouth syndrome. Taste phantoms associated with the disorder were controlled as well. Being a gamma-aminobutyric acid (GABA) agonist, clonazepam increases the concentration of this inhibitory neurotransmitter, therefore simulating taste inhibition.

Unfortunately, in most cases, even if the cause for the taste disorder is known, there are few effective therapies. Thus, the primary aim of the clinician may often be to help patients cope with these disorders (30). Patients with taste disorders cope via cognitive adaptations just as those experiencing other chronic disorders. They find meaning in the disorder, find benefits in it, and make comparisons with others who are less fortunate. Patients' attempts to find the cause of the disorder play a role in coping. Blaming oneself for the disorder is associated with better adjustment.

Some types of support for patients are clearly not helpful. The disorder should not be trivialized nor should it be attributed to psychological causes. Psychological support, however, may be of value to help the patient deal with the stress produced by the symptoms.

As with olfactory problems, direct initial treatment of gustatory dysfunction toward the causative abnormality, if possible.

- Address any nasal pathology causing decreased olfaction and thus affecting taste.
- Treat mucosal disorders (eg, infections, inflammations).
- Treat oral candidiasis and other local factors, and replete any vitamin deficiency that may cause glossitis.
- Aid patients in eliminating local irritants (eg, mouthwashes, ill-fitting dentures)
- In mucositis or dry mouth as a result of radiation therapy, artificial saliva or salivary stimulants and local anti-inflammatory medications may improve some taste dysfunction.
- Correcting endocrine disorders with the appropriate hormone replacement may improve the taste disorder.
- Consider eliminating a medication suspected of causing dysgeusia unless the medication is crucial in treating another medical problem and cannot be substituted.
- In the case of familial dysautonomia, in which patients have a complete lack of lingual taste buds, subcutaneous administration of methacholine has been reported.
to normalize previously elevated taste thresholds for all taste qualities. The cholinergic mechanism is probably related to taste transduction via free nerve endings because these patients have no taste receptors.

- Some gustatory deficits are untreatable (e.g., some cases of nerve or CNS damage, end-stage diabetic neuropathy, multiple sclerosis). Certain mechanical aids exist to enable the patient to make use of whatever taste function is left.

- Advise patients that chewing food well increases the release of the tastant and increases saliva production to further distribute the chemicals. Switching foods during the meal decreases the phenomenon of adaptation and can improve detection of the tastes.

- Finally, for patients who are anosmic or hyposmic (including many elderly people), simulated odors are available to use while cooking to augment the sensation of flavor. A drawback of these simulated odors is that, to normosmic people, the smell is quite pungent. Thus, these odors cannot be used in mixed groups of anosmic and normosmic individuals.

<table>
<thead>
<tr>
<th>4.3.12 CB</th>
<th>How does septoplasty and turbinate reduction influence olfaction?</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>4.3.13 AL</th>
<th>How does chronic renal failure affect olfactory function? Any other medical conditions that interfere with chemosensory function?</th>
</tr>
</thead>
</table>

**Abstract**

**BACKGROUND:**

Patients with chronic renal failure (CRF) show a high prevalence of poor nutritional state so that dietary treatment becomes a significant part of the therapeutic regimen. Because smell plays an important role in nutrition, this study aimed to investigate olfactory function in CRF patients.

**METHODS:**

A total of 64 CRF patients were investigated. Forty-nine of them were treated with hemodialysis, 15 CRF patients were not dialysis dependent. For comparison we examined 15 healthy subjects.
Olfactory function was assessed for odor discrimination, odor identification, and butanol odor thresholds.

RESULTS:

Olfactory loss was found in 56% of the patients, with 3 functional anosmics and 33 hyposmics. CRF had specific effects on individual tests of olfactory function. Elevated odor thresholds were found in 11% of patients, 38% of patients had reduced odor discrimination, and 48% of patients exhibited deficits in odor identification. Results of psychological tests (Mini-Mental State Examination and Trail-Making Test) correlated with results from odor identification ($p < 0.01$) and discrimination ($p < 0.01$) but not with odor thresholds.

CONCLUSIONS:

The ability to discriminate and identify odors was found severely impaired whereas odor thresholds were similar to what is seen in the general population. Consequently, CRF patients should be counseled with regard to the possibility of reduced chemosensory functions.

---

4.3.14 CB

What happens to taste and smell senses after total laryngectomy?