Orofacial Disease: Update for the Dental Clinical Team: 10. Halitosis and Disturbances of Taste, Orofacial Movement or Sensation

CRISPIAN SCULLY AND STEPHEN PORTER

Abstract: This article discusses halitosis, and disorders of taste, orofacial movement or sensation.

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Clinical Relevance: Halitosis (malodour) is a far more common complaint than at first sight appears to be the case. Bell's palsy, though uncommon, can be distressing and requires immediate care.

Halitosis (oral malodour) and cacogeusia (an unpleasant taste in the mouth) are both usually a consequence of poor oral hygiene. Oral or nasal infections, starvation, xerostomia, foods or drugs or psychogenic disorders are other causes, as are various systemic disorders such as respiratory, hepatic or renal disease, diabetic and gastrointestinal disease.

The common causes of facial paralysis are strokes (upper motor neurone lesions) and Bell's palsy (lower motor neurone lesion). Most patients with the latter recover within a few weeks, but the results in others can be so severe and distressing that all patients should immediately be treated with systemic corticosteroids. The eye should be protected with a pad, because the corneal reflex is impaired and corneal damage may occur.

Facial sensory loss is usually caused by extracranial lesions of the trigeminal nerve. Trigeminal nerve branches may be affected by trauma from local analgesic injections, fractures, surgery (particularly osteotomies, resections or surgical extraction of lower third molars), rarely by osteomyelitis or tumour deposits.

The first article in this series presented several general observations on diagnosis and treatment which should be borne in mind in relation to this article.

***Crispian Scully**, PhD, MD, MDS, FDS RCPS, FFD RCSI, FDS RCS, FDS RCSE, FRCPath, FMedSci, Professor, and **Stephen Porter**, PhD, MD, FDS RCSE, FDS RCS, Professor, Eastman Dental Institute for Oral Health Care Sciences and International Centre for Excellence in Dentistry, University of London.*

HALITOSIS

Halitosis, or oral malodour is common and seen mainly in adults.

Aetiology

Oral sepsis is the most usual cause of halitosis (Figure 1). Potent causes of halitosis and bad taste include smoking, and eating various foods (see Table 1). Sepsis in the sinuses or other parts of the respiratory tract may occasionally be responsible. A few systemic diseases such as diabetic ketosis can also give a characteristic odour to the breath (Table 1). With halitosis from any cause, the patient may also complain of a bad taste.

Management

Assessment of halitosis is usually subjective, though a few centres do have the apparatus for objectively measuring the volatile sulphur compounds (methyl mercaptan, hydrogen sulphide, dimethyl sulphide) thought to be causal.

The management is:

- treatment of the underlying cause;
- eating regularly;
- avoiding smoking and eating foods such as onions;
- improving oral hygiene:prophylaxis, toothbrushing and flossing;
- mouthwashes of chlorhexidine, cetylpyridinium or others;
- chewing gum;

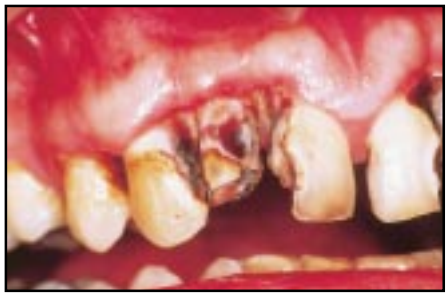

***Figure 1.** Oral sepsis;the main cause of halitosis*

- **Starvation**
- **Oral sepsis or poor hygiene**
- **Xerostomia**
- **Volatile foodstuffs:**
  - Garlic
  - Durivan
  - Curries
  - Onions
- **Drugs and tobacco:**
  - Smoking
  - Alcohol
  - Solvent abuse
  - Chloral hydrate
  - Nitrites and nitrates
  - Dimethyl sulphoxide
  - Cytotoxic drugs
- **Respiratory tract disease:**
  - Infection
  - Tumour
- **Metabolic disease:**
  - Gastrointestinal disease
  - Hepatic failure
  - Renal failure
  - Diabetic ketoacidosis
- **Psychogenic**

Table 1. *Causes of halitosis.*

- using proprietary 'fresh breath' preparations.

DISTURBANCES OF TASTE

Taste is susceptible to genetic and hormonal factors. For example, sensitivity to the bitter taste of phenylthiourea is genetically determined. There appear to be no significant differences in the taste sense between sexes, but it varies through the menstrual cycle and may be distorted during pregnancy, often with the appearance of cravings for unusual foods.

Taste is susceptible to the general sensory phenomenon known as adaptation: the progressive reduction in the appreciation of a stimulus during the course of continual exposure to that stimulus.

Cacogeusia (an unpleasant taste in the mouth) is usually a consequence of poor oral hygiene and oral or nasal infections, starvation, xerostomia, foods or drugs, or psychogenic disorders, but may appear in various systemic disorders such as respiratory, hepatic or renal disease, diabetes and gastrointestinal disease (see Table 2).

The terminology of the various taste dysfunctions is shown in Table 3. Disorders that can result in taste dysfunction include:

- lingual, facial or chorda tympani nerve damage;
- xerostomia;
- smoking;
- drugs such as penicillamine;
- irradiation;
- psychotic disorders;
- neurological disease, including brain tumours;
- ageing;
- nutritional defects;
- anosmia.

Disorders of taste, especially loss of taste, can be distressing and sometimes incapacitating, and can cause anorexia and depression. Probably the most common cause of loss of the sense of taste is anosmia from a viral upper respiratory tract infection; anosmia commonly produces an *apparent* loss of sense of taste. Anosmia may also follow head injuries, due to tearing of olfactory fibres, and may arise in ageing. Other causes of anosmia include some endocrine disorders (especially hypothyroidism), Parkinson's disease, and some other cerebral disorders. Patients need specialist care.

Salivary gland disorders:
- Sjögren's syndrome
- Irradiation damage
- Mumps

Psychogenic causes:
- Depression
- Anxiety states
- Psychoses
- Hypochondriasis

Drugs:
- Drugs causing dry mouth
- Metronidazole, lithium, gold, etc.

Foods:
- Garlic
- Durivan
- Curries
- Onions

Drugs:
- Smoking
- Alcohol
- Solvent abuse
- Chloral hydrate
- Nitrites and nitrates
- Dimethyl sulphoxide
- Cytotoxic drugs

Oral or respiratory tract infections

Liver failure and cirrhosis

Renal failure

Diabetic ketosis

Gastrointestinal disease

Table 2. *Causes of unpleasant taste.*

Dysfunction	Sense of taste
Absence	Ageusia
Diminished	Hypogeusia
Distorted	Dysgeusia
Heightened	Hypergeusia

Table 3. *Terminology of disorders of taste.*

DISORDERS OF FACIAL MOVEMENT AND SENSATION

Facial Palsy

Aetiology

The common causes of facial paralysis are:

- strokes (upper motor neurone lesions), seen mainly in older men; and
- Bell's palsy (lower motor neurone lesion), seen mainly in younger patients.

Other common causes are listed in Table 4. Occasionally, a temporary facial palsy follows the administration of an inferior alveolar local analgesic if the solution tracks through the parotid gland to reach the facial nerve.

Clinical Features

The neurones to the upper face receive bilateral upper motor neurone innervation. Upper motor neurone facial palsy is usually associated with damage in the middle capsule of the

Upper motor neurone lesion:
- Cerebrovascular accident
- Trauma
- Tumour
- Infection
- Multiple sclerosis

Lower motor neurone lesion:
- Infection
  - Bell's palsy
  - Varicella-zoster virus infection
  - Lyme disease
  - HIV infection
- Middle ear disease
- Lesion of skull base
- Parotid lesion:
  - Trauma to branch of facial nerve
  - Tumours
- Trauma

Table 4. *Causes of facial palsy.*

brain. Damage thus extends to other areas, including motor neurones, but extrapyramidal influences can still act on the face, for example, on laughing because of the bilateral cortical representation. An upper motor neurone lesion therefore is characterized by unilateral facial palsy, with some sparing of the frontalis and orbicularis oculi muscles; the face may still move with emotional responses (because of extrapyramidal influences) and there may also be a paresis of the ipsilateral arm or arm and leg or some aphasia.

In contrast, lower motor neurone facial palsy is characterized by total unilateral paralysis of all muscles of facial expression, both for voluntary and emotional responses. However, there is no hemiparesis, because the facial nerve neurones supplying the lower face receive upper motor neurones only for the contralateral motor cortex (the two conditions are compared in Table 5).

In facial palsy (Figure 2):

- the forehead is unfurrowed;
- the patient is unable to close the eye on the affected side;
- the eye rolls upward on attempted closure (Bell's sign);
- tears tend to overflow onto the cheek (epiphora);
- the nasolabial fold is obliterated;
- the corner of the mouth droops;
- saliva may dribble from the commissure.

Depending on the site of the neurological lesion, other features such as loss of taste may be seen. Food collects in the vestibule on the affected side and plaque readily accumulates on the teeth.

Diagnosis and Management

Facial weakness is demonstrated by asking the patient to close their eyes against resistance, to raise their eyebrows, or to raise their lips to show the teeth or to try to whistle (Figure 2). The following are also necessary:

- a full neurological examination, looking particularly for V, VI and VII nerve signs, hemiparesis etc.;
- tests for hearing loss, examination for discharge from the ear and other signs of middle ear disease;
- measurement of blood pressure (to exclude hypertension) and fasting blood sugar levels (to exclude diabetes).

In some areas, Lyme disease (tick-borne infection with *Borrelia burgdorferi*) should be excluded. Thus a specialist referral may be warranted.

Bell's Palsy

This is a rare acute lower motor neurone facial palsy without neurological deficit in any other nerve.

Aetiology

The common lower motor neurone lesion in Bell's palsy is caused by inflammation in the stylomastoid canal. It may be immunologically mediated and associated with herpes simplex virus or, rarely, another micro-organism such as varicella-zoster, Lyme disease or HIV.

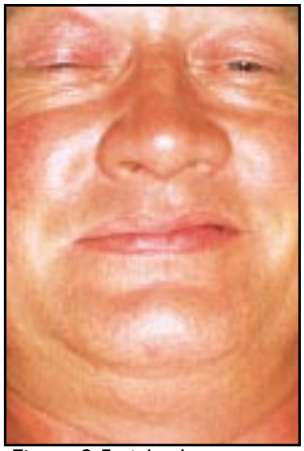

Figure 2. *Facial palsy.*

Clinical Features

There is acute onset of unilateral complete facial paralysis over a few hours. This is occasionally preceded by pain or followed by partial loss of taste or hyperacusis (accentuated hearing).

Management

Most patients recover within a few weeks, but the results in others can be so severe and distressing that all patients should immediately be treated with systemic corticosteroids. Aciclovir may also be indicated. The eye should be protected with a pad, because the corneal reflex is impaired and damage may occur.

In chronic cases, where there is no recovery after months or years, it may be necessary to use Faradic stimulation, a splint or fascial graft to prevent

	UMN lesions	LMN lesions
Emotional movements of face	Retained	Lost
Blink reflex	Lost	Retained
Ability to wrinkle forehead	Lost	Retained
Drooling from commissure	Common	Uncommon
Lacrimation, taste or hearing	May be affected	Unaffected

Table 5. *Differentiation of upper (UMN) from lower (LMN) motor neurone lesions of the facial nerve.*

drooping at the commissure, or other manoeuvres such as facial-hypoglossal nerve anastomosis in an attempt to overcome the cosmetic deformity. Surgical referral is then necessary.

Facial Sensory Loss

Apart from the loss of sensation from lingual nerve damage after third molar surgery, sensory loss in the face is rare. It may denote serious disease.

Aetiology

Facial sensory loss is caused mainly by *extracranial lesions* of the trigeminal nerve. The mandibular division or its branches may be affected by:

- trauma from inferior alveolar or other local analgesic injections, fractures, surgery (particularly osteotomies, resections or surgical extraction of lower third molars). The lingual nerve may be damaged, especially during removal of lower third molars, particularly when the lingual split technique is used;
- pressure (occasionally the mental foramen is close beneath a lower denture and there is anaesthesia of the lower lip on the affected side, as a result of pressure from the denture);
- osteomyelitis or tumour deposits.

Damage to branches of the maxillary division of the trigeminal nerve may be caused by:

- trauma (especially zygomatic or middle-third facial fractures);
- tumours such as carcinoma of the maxillary antrum.

Intracranial causes of facial sensory loss (Table 6) are less common but more important and include:

- multiple sclerosis;
- brain tumours;
- syringobulbia.

Since other cranial nerves are anatomically close, there may be associated neurological deficits. For example, in posterior or middle cranial fossa lesions, there may be features such as ataxia.

Benign trigeminal neuropathy is a transient sensory loss in one or more divisions of the trigeminal nerve though the corneal reflex is not affected. The aetiology is unknown, but some patients prove to have a connective tissue disorder.

Psychogenic causes of facial sensory loss include hysteria, and particularly hyperventilation syndrome.

Local
- Trauma to nerves during removal of wisdom or other teeth, jaw fractures, or orthognathic surgery.
- Osteomyelitis, leukaemic deposits and metastases
- Nasopharyngeal carcinoma or other neoplasm invading the pterygomandibular space
- Antral carcinoma

Systemic
- Multiple sclerosis
- Tumours
- Syringomyelia
- Connective tissue disease
- Surgical treatment of trigeminal neuralgia
- Psychogenic, hyperventilation syndrome, hysteria
- Drugs
- Benign trigeminal sensory neuropathy

Table 6. *Causes of loss of orofacial sensation.*

Clinical Features

Lesions of the sensory part of the trigeminal nerve initially result in a diminishing response to pin-prick of the skin and, later, complete anaesthesia. Later there may be corneal, facial or oral ulceration.

Management

In view of the potential seriousness of facial sensory loss, full neurological assessment should be undertaken unless there is a very obvious local cause.

It is important in patients complaining of facial anaesthesia to test all areas but particularly the corneal reflex. Lesions involving the ophthalmic division of the trigeminal nerve cause corneal anaesthesia, which is tested by gently touching the cornea with a wisp of cotton wool twisted to a point. Normally, this causes a blink but, if the cornea is anaesthetic (or if there is facial palsy) there is no blink, as long as the patient does not actually see the cotton wool.

If the patient complains of complete facial or hemifacial anaesthesia, but the corneal reflex is retained or there is apparent anaesthesia over the angle of the mandible (an area *not* innervated by the trigeminal nerve), then the symptoms are probably functional (non-organic).

If the cornea is anaesthetic, a protective eye pad should be worn and tarsorrhaphy (an operation to unite the upper and lower eyelids) may be indicated because the protective corneal reflex is lost and the cornea may be damaged.

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