# Orofacial Disease: Update for the Dental Clinical Team: 8. Salivary Complaints

CRISPIAN SCULLY AND STEPHEN PORTER

*Abstract:* Certain lesions exclusively or typically affect the salivary glands; these are discussed in this article.

*Dent Update 1999; 26: 357-365*

*Clinical Relevance:* An awareness of the causes of dry mouth is essential for the dental practitioner, who should also be able to provide advice on saliva substitutes and stimulation of salivation for patients so affected.

Saliva is essential to oral health, and patients who lack salivary flow suffer from lack of oral lubrication and defences, resulting in dysfunction and infections. Drugs are the most common cause of a reduction in salivary flow but there is a range of less common causes. Salivation may be stimulated by using chewing gums, diabetic sweets, or cholinergic drugs that stimulate salivation (sialogogues), such as pilocarpine. Salivary substitutes may help symptomatically. Care is needed to prevent caries, candidosis and sialadenitis. Causes of salivary gland swelling include inflammatory lesions (mumps, ascending sialadenitis, recurrent parotitis, HIV parotitis, Sjögren's syndrome, sarcoidosis), neoplasms, duct obstruction and sialosis.

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.*

## DRY MOUTH (XEROSTOMIA)

Saliva is essential to oral health; patients who lack salivary flow suffer from lack of oral lubrication, affecting many functions, and may develop oral and salivary gland infections as a consequence of the reduced defences.

### *Aetiology*

Dry mouth is a common salivary complaint, though objective evidence of xerostomia may be lacking. There is a range of causes of a reduction in salivary flow (see Table 1) but drugs are the most common cause. Extreme xerostomia may be seen in patients taking lithium for manic depression. The cause for which the drug is being taken may also be important. For example, patients with anxiety states or depressive conditions may complain of a dry mouth even in the absence of drug therapy or evidence of reduced salivary secretion.

Patients who have received head and neck irradiation for neoplastic conditions often develop a dry mouth, especially when both parotid glands fall within the field of irradiation.

Diseases affecting the salivary glands, such as Sjögren's syndrome, sarcoidosis, HIV disease and graft-versus-host disease may also cause a dry mouth.

### *Clinical Features*

A number of features are characteristic of xerostomia:

- difficulty eating dry foods such as biscuits (the cracker sign);
- soreness;
- difficulties in controlling dentures;
- difficulties in speech and swallowing;
- unpleasant taste or loss of sense of taste (this will be discussed further in a later article);
- clicking quality of the speech as the tongue tends to stick to the palate.

**IATROGENIC:**
**Drugs**
- Anticholinergic drugs such as tricyclics, phenothiazines and antihistamines
- Sympathomimetic drugs such as some antihypertensive agents
- Cytotoxic drugs

**Irradiation**
- External beam irradiation to neoplasms in the head and neck region
- Iodine 131 for treatment of thyroid disease

**Graft versus host disease**
**DISEASE AFFECTING SALIVARY GLANDS:**
- Sjögren's syndrome
- HIV salivary gland disease
- Sarcoidosis
- Cystic fibrosis
- Salivary gland aplasia
- Others

**DEHYDRATION:**
- Severe diabetes
- Others

**PSYCHOGENIC**

***Table 1.*** *Causes of dry mouth*

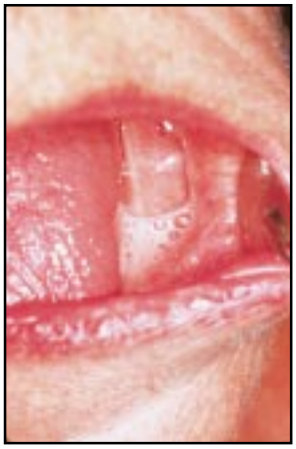

***Figure 1.*** *Xerostomia; frothy saliva.*

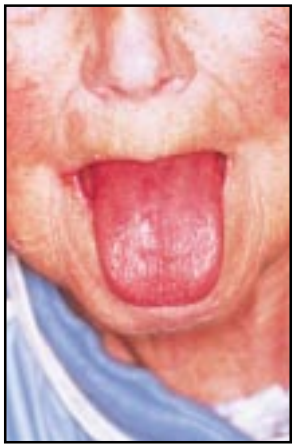

***Figure 2.*** *Xerostomia; lobulated tongue.*

The mouth may appear dry; on examination there may be a lack of the usual pooling of saliva in the floor of the mouth and thin lines of frothy saliva may form along lines of contact of the oral soft tissues (Figure 1). The mucosa also tends to stick to a dental mirror. In advanced cases the mucosa is obviously dry and glazed. The tongue typically also develops a characteristic lobulated, usually red, surface with partial or complete depapillation (Figure 2).

## Complications

Dental caries (Figure 3) tends to be severe and difficult to control; indeed, xerostomia is not infrequently diagnosed because the patient appears unduly predisposed to caries. Soreness and redness of the oral mucosa are usually the result of candidosis, which is common. Ascending (suppurative) bacterial sialadenitis is a hazard.

## Diagnosis

Salivary function studies may be indicated to establish true xerostomia and its underlying cause and patients may need referral to a specialist.

### *Salivary Flow Rates (Sialometry)*

Salivary flow rate estimation is a sensitive but non-specific indicator of salivary gland dysfunction. This is now carried out by allowing the patient to dribble into a measuring container over 15 minutes.

### *Salivary Biopsy*

Biopsy of a parotid gland could result in damage to the facial nerve, a salivary fistula, or scarring. For these reasons, minor salivary glands are usually biopsied. The glands selected are the glands in the lower labial mucosa because they are readily approached through a simple incision inside the labial mucosa with few adverse effects other than occasional mild anaesthesia.

### *Sialography*

Radio-opaque dye introduced into the salivary duct shows sialectasis, a snowstorm appearance as a result of leakage of contrast medium through the damaged ducts.

### *Salivary Scintiscanning*

Salivary scintiscanning with technetium pertechnetate correlates with both salivary flow rate and labial gland changes and offers the additional advantage that all major salivary glands are examined non-invasively, simultaneously and (if necessary) continuously. However, it is not always available, is expensive and carries a small radiation hazard.

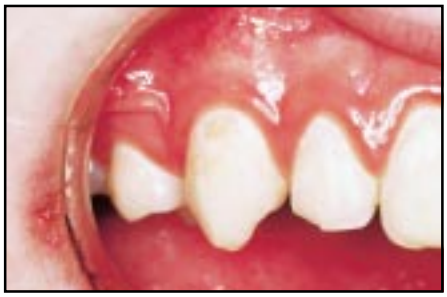

***Figure 3.*** *Xerostomia; decalcification and early caries.*

### *Ultrasound*

This is useful mainly where a neoplasm is suspected.

### *Sialochemistry*

In practical terms, sialochemistry (studies of constituents of saliva) is of limited clinical value.

## Treatment

Any underlying cause should, if possible, be rectified. It is also wise for the patient to avoid:

- drugs that may produce xerostomia (for example tricyclic antidepressants);
- alcohol;

| Dental surgeon | Ancillary, Hygienist, Nurse |
|---|---|
| Consult physician if a drug cause is likely. Refer patients with systemic disease, or where the diagnosis is in doubt. Understand disease and management in order to extend education of, and reassure, patient | Understand disease and management in order to extend education of, and reassure, patient |
| Alert Specialist to any possible adverse effects of treatment | Oral health education of patient |
| Oral health care; in particular to avoid complications of xerostomia (caries, candidosis, sialadenitis) | Dietary counselling and preventive dental care measures |
| Oral health education of patient | Alert dental surgeon to any changes to, or possible adverse effects of, treatment |

***Table 2.*** *Role of the dental clinical team in management of patients with dry mouth*

- Saliva usually helps swallowing and talking, and protects the mouth
- Where saliva is reduced there is a risk of dental decay (caries) and infections
- It is therefore important to keep your mouth clean and use fluoride rinses
- Dryness can be combated by chewing sugarless gum, and rinsing with water
- Always take water or non-alcoholic drinks with meals
- Avoid biscuits, sugar, sweets, alcohol and tobacco
- The dental surgeon or doctor may be able to help with an artificial saliva, or saliva stimulant

***Table 3.*** *Information sheet for the patient with dry mouth.*

- smoking; and
- dry foods such as biscuits.

Patients should be advised that salivation can be stimulated by using:

- chewing gums (containing sorbitol, not sucrose);
- diabetic sweets;
- cholinergic drugs that stimulate salivation (sialogogues), such as pilocarpine. These should be used only by the specialist because they unfortunately can cause other cholinergic effects such as bradycardia. Pyridostigmine is of greater benefit because it is longer acting and associated with fewer adverse effects.

Salivary substitutes may help symptomatically. Various are available including:

- water;
- methylcellulose;
- mucin.

Saliva Orthana is particularly useful because it contains fluoride. Glandosane is also effective but has a lowish pH and may thus not be so suitable.

The role a general dental practitioner plays in the management of xerostomia is summarized in Table 2, and a summary of the advice that should be given to the patient with dry mouth is shown in Table 3.

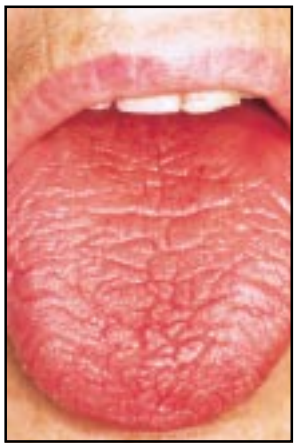

***Figure 4.*** *Xerostomia; dry tongue.*

## Dental Caries

Dietary control of sucrose intake, and the daily use of fluorides (1% sodium fluoride gels or 0.4% stannous fluoride gels) are essential to control dental caries.

## Candidosis

Dentures should be left out of the mouth at night and stored in sodium hypochlorite solution or chlorhexidine. An antifungal, such as miconazole gel or amphotericin or nystatin ointment, should be spread on the denture before reinsertion and a topical antifungal preparation such as nystatin or amphotericin suspension or lozenges used. Fluconazole is also effective.

## Bacterial Sialadenitis

Acute sialadenitis needs treatment with a penicillinase-resistant antibiotic such as flucloxacillin.

# SALIVARY DISORDERS

Certain lesions exclusively or typically affect the salivary glands but some of these conditions may affect other exocrine glands, particularly the lacrimal glands and pancreas. Sjögren's syndrome is a case in point.

## Sjögren's Syndrome

Sjögren's syndrome is the uncommon association of dry mouth and dry eyes, seen mainly in middle-aged and elderly women.

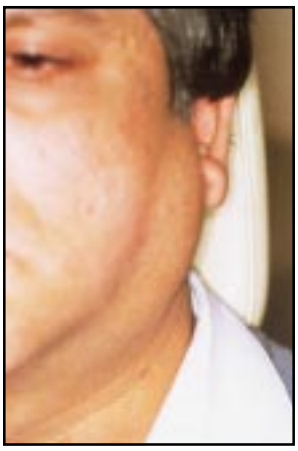

***Figure 5.*** *Salivary swelling.*

### *Aetiology*

Sjögren's syndrome is an autoimmune inflammatory exocrinopathy which appears to be the result of lymphocyte-mediated destruction of salivary, lacrimal and other exocrine glands (Figure 4).There may be a viral aetiology and a genetic predisposition. Sjögren's syndrome is characterized by several autoantibodies—particularly antinuclear antibodies (ANA) and rheumatoid factor (RF) and antinuclear antibodies known as Sjögren's syndrome A (Ro or SS-A) and B (La or SS-B).

### *Clinical Features*

Sjögren's syndrome is a chronic multi-system disease which presents mainly with eye and oral complaints.

*Eye complaints* include sensations of grittiness, soreness or dryness. The eyes may be red, with infection of the conjunctivae and soft crusts at the angles (keratoconjunctivitis sicca). The

- Connective tissue disease: absent
- Oral involvement: more severe
- Recurrent sialadenitis: more common
- Eye involvement: more common
- Lymphoma: more common

***Table 4.*** *Features of primary Sjögren's syndrome compared with those of secondary Sjögren's syndrome.*

| Investigation | Findings |
|---|---|
| Salivary flow rate (sialometry) | Reduced |
| Labial salivary gland biopsy | Focal lymphocytic infiltrate. May help to predict lymphomas |
| Scintiscanning (scintigraphy) | Reduced uptake of technetium. |
| Sialography | Sialectasis |

***Table 5.*** *Sjögren's syndrome: salivary studies*

lacrimal glands may swell. Ophthalmological examination is essential.

*Oral complaints* (often the presenting feature) include xerostomia and associated features (see above). The tongue typically also develops a characteristic lobulated, usually red, surface with partial or complete depapillation (Figure 4).

Other complaints due to Sjögren's syndrome include arthritis, dry vagina, purpura and many others.

These clinical features in the absence of systemic disease are often now referred to as *primary* Sjögren's syndrome (SS-1), frequently termed sicca syndrome (Table 4). The most common type of Sjögren's syndrome however, is secondary Sjögren's syndrome (SS-2), which comprises dry eyes and dry mouth and a connective tissue or autoimmune disease, such as:

- rheumatoid arthritis;
- systemic lupus erythematosus;
- systemic sclerosis;
- mixed connective tissue disease;
- primary biliary cirrhosis.

### *Complications*

- Candidosis, which is common may cause soreness and redness of the oral mucosa.
- Dental caries, which tends to be severe and difficult to control.
- Salivary gland enlargement (Figure 5).
- Ascending (suppurative) sialadenitis may result from bacterial infection ascending the salivary duct.
- Pseudolymphoma, which is a massive swelling of the salivary glands, associated with enlargement of the regional lymph nodes.
- True lymphoma may result if the B cell lymphoproliferation becomes malignant.

### *Diagnosis and Management*

The diagnosis of Sjögren's syndrome is made mainly from the history and clinical examination, but investigations may also be needed, and thus specialist referral warranted. A similar syndrome may be seen in HIV disease.

Investigations may include:

- autoantibody profile (for rheumatoid and antinuclear factors, and anti-Ro and anti-La antibodies). Anti-Ro in particular is found, and if other antinuclear antibodies are present (such as RF) the patient may have secondary Sjögren's syndrome;
- haematological investigation to exclude anaemia;
- erythrocyte sedimentation rate or plasma viscosity (raised);
- biopsy of labial salivary glands;
- a rinse or swab from the oral mucosa to confirm the presence of candidosis if there is soreness;
- salivary studies (see Table 5).

Although it is desirable to control the underlying autoimmune disease, this is at present experimental only (e.g. cyclosporin). The patient should, however, be followed up regularly, particularly because of the possibility of development of lymphoma.

It is most important that the eyes are examined by a specialist (Figure 6). Methylcellulose eye drops or, rarely, ligation or cautery of the nasolacrimal duct may be needed. Dry mouth should be treated as discussed and the patient must be kept informed about their condition (Table 6).

## Sialorrhoea (Ptyalism)

The complaint of excess salivation is very uncommon.

### *Aetiology*

Sialorrhoea is common in infants, especially when 'teething', and is common at any age where there are any painful lesions or foreign bodies in the mouth—such as ulcers or new dentures. In some cases it is not due to excess saliva production but to an inability to swallow saliva as a result of poor neuromuscular co-ordination as occurs in neurological disorders such as Parkinson's disease and cerebral palsy. Patients with learning disability, pharyngeal obstruction, or reduced swallowing rate may also exhibit sialorrhoea. Drugs such as anticholinesterases and cocaine, and rabies, are rare causes. Some psychogenic cases have been reported.

### *Clinical Features*

Typically, drooling is seen in patients whose underlying condition is often neurological or muscular. In other patients the complaint of sialorrhoea seems to have no physical basis and cannot be confirmed by sialometry, and appears to reflect an underlying psychological problem.

### *Management*

Atropinics, although theoretically useful to control sialorrhoea, are not often of practical value because of adverse side-effects, and therefore antihistamines are sometimes used. Surgical techniques have been devised to reroute the submandibular gland duct to open posteriorly.

## SALIVARY SWELLINGS

It can be difficult to establish whether a salivary gland is genuinely swollen, especially in obese patients. A useful guide to whether the patient is simply obese or has parotid enlargement is to observe the outward deflection of the ear lobe, which is seen in true parotid swelling (Figure 5).The causes of swelling of the salivary glands are summarized in Table 7.

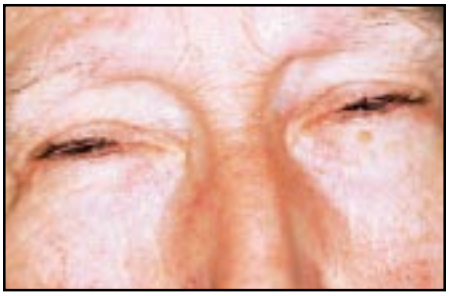

***Figure 6.*** *Eye involvement in Sjogren's syndrome.*

## Mumps (Acute Viral Sialadenitis: Parotitis)

By far the most common cause of salivary gland swelling is infection—usually mumps, an acute infectious disease which principally affects the parotid salivary glands of children.

### *Aetiology*

RNA paramyxovirus, the mumps virus; rarely Coxsackie, ECHOviruses, EBV or HIV infection. Transmission of mumps is by direct contact or by droplet spread from saliva.

### *Clinical Features*

An incubation period of 2 to 3 weeks elapses before clinical features appear but many infections are subclinical.

Typically mumps presents as:

- parotitis—acute onset of painfully and usually bilaterally enlarged parotids, although in the early stages only one parotid gland may appear to be involved. The submandibular glands are also affected. The skin over the affected glands appears normal, as does the saliva—features which help distinguish from acute bacterial sialadenitis;
- trismus;
- fever, anorexia and malaise.

Extrasalivary manifestations may include:

- inflammation of the testes (orchitis)—ensuing infertility is rare;
- oophoritis;
- pancreatitis;
- meningitis or meningoencephalitis;
- deafness.

### *Diagnosis and Management*

The diagnosis is clinical but confirmation, if needed, is by demonstrating a fourfold rise in serum antibody titres between acute serum and convalescent serum taken 3 weeks later. Raised levels of serum amylases or lipases are found.

No specific antiviral agents are available. Treatment is symptomatic, involving analgesics, adequate hydration and reducing the fever. Patient isolation for 6 to 10 days may be advised because the virus is in saliva during this time. Prevention is by immunization in childhood.

- *This is an uncommon condition*
- *The cause is unknown but it is immunological and possibly viral*
- *It is not known to be contagious*
- *It is not usually inherited*
- *Dry eyes are commonly found*
- *Joint and other problems may be associated*
- *Rarely, a very few patients may, after years, develop a tumour. You should get yourself checked regularly*
- *X-rays, blood tests and biopsy may be required*
- *Symptoms can usually be controlled, but not cured, with simple drugs*

***Table 6.*** *Information sheet for the patient with Sjögren's syndrome.*

## Bacterial Salivary Gland Infection

This is also known as acute bacterial ascending sialadenitis. It is rare and seen mainly in older patients.

### *Aetiology*

The parotid glands are most commonly affected by ascending sialadenitis, which may be seen in:

- hospital in-patients. It was previously not uncommon following gastrointestinal surgery because of dehydration and dry mouth, infection ascending from the mouth. With better understanding of fluid balance and oral hygiene, and more widespread use of prophylactic antibiotics, this is now uncommon;
- patients who have undergone radiotherapy to the head and neck;
- people with Sjögren's syndrome;
- otherwise apparently healthy patients with abnormalities such as calculi, mucus plugs and duct strictures.

- Inflammatory:
  - Mumps
  - Ascending sialadenitis
  - Recurrent parotitis
  - HIV parotitis
  - Sjögren's syndrome
  - Sarcoidosis
- Cystic fibrosis
- Neoplasms
- Duct obstruction
- Sialosis
- Drugs
- Deposits e.g. amyloid

***Table 7.*** *Causes of salivary gland swelling*

The organisms most commonly isolated are *Streptococcus viridans* and *Staphylococcus aureus*, the latter frequently being penicillin resistant.

### *Clinical Features*

Acute sialadenitis typically presents with:

- painful and tender enlargement of one gland;
- possible reddening of the overlying skin;
- pus exuding from, or milked from, the duct orifice;
- trismus;
- cervical lymphadenopathy;
- pyrexia.

If the infection localizes as an abscess, it may point externally through the overlying skin or, rarely, into the external acoustic meatus.

### *Diagnosis and Management*

The diagnosis is essentially clinical but pus should be sent for culture and sensitivity testing. Specialist referral may be indicated. Prompt antimicrobial therapy is indicated (flucloxacillin if caused by *Staphylococcus* and the patient is not allergic to penicillin) and, where fluctuation is present, surgical

drainage is needed as there may be extensive glandular damage. Hydration must be ensured. Salivation should be stimulated by chewing gum or use of sialogogues.

## Chronic Bacterial Sialadenitis

Chronic bacterial sialadenitis may develop after acute sialadenitis, particularly if inappropriate antibiotics are used or predisposing factors not eliminated. Chronic sialadenitis may follow salivary calculus formation. Unfortunately, serous acini may atrophy when salivary outflow is chronically obstructed and this further reduces function. Surgical excision is often needed.

## Recurrent Parotitis of Childhood

Chronic recurrent parotitis of childhood is an uncommon condition, characterized by repeated parotitis and sialectasis, which often improves spontaneously at puberty. No reliably effective treatment has been reported; repeated courses of antimicrobials are often used.

## Sarcoidosis

Sarcoidosis is an uncommon chronic disease of unknown cause, in which granulomas form particularly in the lungs, lymph nodes (especially the hilar nodes), salivary glands and other sites such as the mouth. The prevalence is highest in adult Black women.

### *Clinical Features*

*Orofacial features* include:

- cervical lymphadenopathy;
- occasionally enlarged salivary glands—Heerfordt's syndrome (salivary and lacrimal swelling, facial palsy and uveitis) is rare;
- xerostomia;
- mucosal nodules;
- gingival hyperplasia;
- labial swelling.

***Figure 7.*** *Submandibular swelling.*

*Systemic features* include:

- erythema nodosum;
- lymphadenopathy;
- lung involvement.

### *Diagnosis and Management*

Specialist referral is indicated if this multisystem disease is suspected. Biopsy of affected tissue shows characteristic granulomas, as may labial salivary gland biopsy. In over 50% of patients with bilateral hilar lymphadenopathy, biopsy of a labial salivary gland shows typical granulomas which are non-caseating, contain multinucleated giant cells and are surrounded by lymphocytes.

Lymphadenopathy can be revealed by chest radiography (showing hilar lymphadenopathy) or gallium scan. Gallium is taken up by macrophages in the granulomas; a scan may show uptake in involved lymph nodes, salivary and lacrimal glands.

The Kveim test used to be carried out by intracutaneous injection of a heat-sterilized suspension of human lymphoid tissue affected with sarcoidosis. After 4 to 6 weeks the area was biopsied and, if positive, showed well formed epithelioid non-caseating granulomas. The test is positive in about 80% of patients with sarcoidosis. The Kveim test has now been superseded by serum angiotensin-converting enzyme and adenosine deaminase levels, both of which are raised in sarcoidosis.

Patients with only minor symptoms of sarcoidosis often require no treatment. Corticosteroids are used if there is active disease of the lungs or eyes, cerebral involvement, or other serious complications such as hypercalcaemia.

## Salivary Duct Obstruction

### *Aetiology*

Salivary duct obstruction is fairly common, and usually caused by a calculus in the submandibular duct. Strictures, mucus plugs or neoplasms are occasional causes. Rarely, patients present with 'physiological' duct obstruction due either to duct spasm or an abnormal passage of the parotid duct through the buccinator or in relation to the masseter muscles.

### *Clinical Features*

These include salivary gland swelling, which is unilateral, painful and intermittent, appearing just before or at mealtimes (Figure 7). In older patients this history is not always obtained; there may just be dull pain over the affected gland, referred elsewhere. There is a lack of swelling at other times, and some obstructions are completely asymptomatic.

Prolonged duct obstruction produces atrophy, particularly of serous acini.

### *Diagnosis and Management*

It is rarely possible clinically to determine the cause of major duct obstruction except when a calculus is palpable. Plain radiographs may reveal a calculus but nearly 50% are radiolucent. If a calculus is not obvious, specialist referral is usually indicated. Sialography should help to differentiate the various causes of major duct obstruction. Extraductal causes of obstruction may be apparent only on sialography or combined sialography and computed tomography (CT) scanning.

Treatment is surgical removal of the obstruction (such as a calculus), lithotripsy, or duct dilatation.

## Obstruction of Minor Salivary Glands

Minor gland outlet obstruction is most commonly encountered when a mucocele is present. Mucoceles are

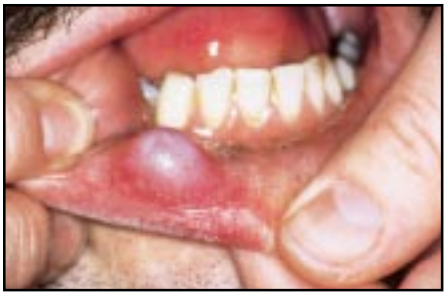

***Figure 8.*** *Mucocele.*

fairly common, mostly inside the lower lip and in young adults/children, particularly males.

### *Aetiology*

Most mucoceles are caused by trauma to the duct of a minor salivary gland leading to extravasation of mucus and the appearance of a cystic lesion—which is not, however, lined by epithelium, and therefore is not a true cyst. Occasional mucoceles are caused by saliva retention.

### *Clinical Features*

Most mucoceles appear in the lower labial mucosa, buccal mucosa or ventrum of the tongue. They are dome-shaped, bluish, translucent, fluctuant painless swellings, usually up to 1 cm in diameter (Figure 8). These rupture easily to release viscid salty mucus, but frequently recur. Ranula is a term used for the 'frog belly' appearance of rare large mucoceles in the floor of the mouth, which may involve the sublingual gland or, rarely, burrow through the mylohyoid muscle (plunging ranula). Superficial mucoceles are small intraepithelial lesions simulating a vesiculobullous disorder.

| | |
|---|---|
| **Drugs:** | Alcohol<br>Others |
| **Endocrine:** | Diabetes mellitus<br>Acromegaly<br>Pregnancy |
| **Metabolic:** | Liver cirrhosis<br>Starvation, e.g. anorexia nervosa/bulimia<br>Cystic fibrosis |

***Table 8.*** *Causes of sialosis.*

### *Diagnosis and Management*

Diagnosis is clear-cut but a salivary gland neoplasm must be excluded, particularly in cystic swellings in the *upper* lip. Mucoceles can resolve spontaneously; alternatively they can be excised, or treated with cryosurgery.

## Sialosis (Sialadenosis)

Sialosis is a rare benign, non-inflammatory, non-neoplastic, bilaterally symmetrical and painless enlargement of salivary glands which usually affects the parotids in adults.

### *Aetiology*

Although many cases are idiopathic, a variety of causes of sialosis are recognized (Table 8), dysregulation of the autonomic innervation of the salivary glands being the unifying factor in all. The main causes include:

- sympathomimetic drugs such as isoprenaline;
- alcohol abuse with or without accompanying liver cirrhosis;
- endocrine changes, including diabetes mellitus, acromegaly, thyroid disease and pregnancy;
- malnutrition in starvation, anorexia nervosa, bulimia and cystic fibrosis.

### *Clinical Features*

These include:

- salivary gland swelling: soft, painless, generally bilateral (usually the parotids) (Figure 9);
- no xerostomia;
- no trismus;
- no fever.

### *Diagnosis and Management*

The diagnosis of sialosis is one of exclusion, based mainly on history and clinical examination, and specialist referral may be indicated. Salivary gland function is normal but sialography is likely to show enlarged normal glands. Sialochemistry may show raised potassium and calcium levels, which would not be present in salivary enlargement due to other causes. Biopsy is not usually needed but blood

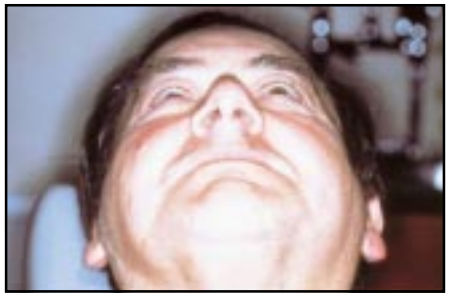

***Figure 9.*** *Sialosis (sialadenosis)*

examination for raised glucose levels, possibly growth hormone levels or abnormal liver function may point to an underlying cause.

No specific treatment is available but sialosis may resolve when alcohol intake is reduced or glucose control instituted.

## SALIVARY NEOPLASMS

### *Aetiology*

The aetiology of salivary neoplasms is unclear. Viruses may be involved; polyoma viruses have been implicated in animal models, and other viruses, such as Epstein-Barr virus, in some human neoplasms. Irradiation has been implicated in some tumours.

### *Clinical Features*

A wide range of different neoplasms can affect the salivary glands.
*Tumours of the major salivary glands* mostly:

- present as unilateral swelling of the parotid;
- are benign;
- are pleomorphic adenomas. The next most common tumour is

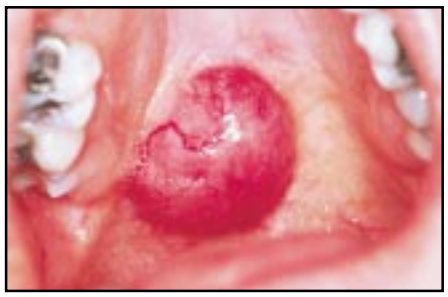

***Figure 10.*** *Pleomorphic salivary adenoma.*

**Epithelial tumours**
- Adenomas (benign)
  Pleomorphic (allied condition benign lympho-epithelial lesion)
  Monomorphic (adenolymphoma—allied condition sialosis, oxyphilic, others—allied condition oncocytosis)
- Mucoepidermoid (intermediate)
- Acinic cell tumours (intermediate)
- Carcinomas (malignant)
  Adenoid cystic
  Adenocarcinoma
  Epidermoid
  Undifferentiated
  Carcinoma in pleomorphic adenoma

***Table 9.*** *Classification of salivary gland tumours (after World Health Organization)*

carcinoma which, in some cases, arises in a long-standing pleomorphic salivary adenoma.

The 'rule of nines' is an approximation that states that 9 out of 10 tumours affect the parotid, 9 out of 10 are benign, and 9 out of 10 are pleomorphic salivary adenomas (PSAs). *Intra-oral salivary gland neoplasms* are:

- less common than in major glands;
- more often malignant;
- typically unilateral;
- mainly pleomorphic adenoma, but adenoid cystic carcinoma and mucoepidermoid carcinoma are relatively more common in the mouth than in the major glands;
- most common in the palate (Figure 10) but may be seen in the buccal mucosa or upper lip, and rarely in the tongue or lower lip.

### *Malignant Potential*

Most tumours in the parotid gland are PSAs and benign. Most submandibular gland tumours are PSAs and benign but one-third are malignant. Most sublingual gland tumours are malignant. Tumours of the tongue are usually malignant—especially adenoid cystic carcinoma. Tumours on the lips are generally benign (pleomorphic or other adenoma) and seen in the upper lip.

### *Classification*

The World Health Organization classification is the most widely used (see Table 9), and the epithelial tumours, which are the most important, can be memorized by the mnemonic *A Most Acceptable Classification* (Adenomas; Mucoepidermoids; Acinic cell tumours; Carcinomas).

## Adenomas

*The pleomorphic salivary adenoma* (PSA; mixed salivary gland tumour)is the most common salivary gland neoplasm, usually slow-growing and benign. The tumour is poorly encapsulated and parotid adenomas are in intimate relationship with the facial nerve—both facts making complete excision difficult. Most pleomorphic adenomas are lobulated, rubbery swellings with normal overlying skin or mucosa but a bluish appearance if inside the mouth. They are not fixed to deeper tissues. Malignant change is uncommon but is suggested clinically by:

- rapid growth;
- pain;
- fixation to deep tissues;
- facial palsy.

*Monomorphic adenomas*, unlike pleomorphic adenomas, have a uniform cellular structure of epithelial elements. They include:

- Adenolymphoma (papillary cystadenoma lymphomatosum or Warthin's tumour). This neoplasm is found only in the parotid and is benign, and may be bilateral.
- Oxyphil adenoma. This rare neoplasm is found virtually only in the parotid, affects mainly the elderly and is benign.

## Mucoepidermoid Tumours

This tumour is usually slow-growing, of low grade malignancy or is benign.

## Acinic Cell Tumours

Acinic cell tumours are very rare and usually benign, though all grades of malignancy have been reported.

## Carcinomas

Salivary carcinomas are uncommon, malignant, and usually adenoid cystic or adenocarcinomas.

- Adenoid cystic carcinoma (cylindroma) is a slow-growing malignant tumour which infiltrates perineurally and metastasizes.
- Adenocarcinoma is a rapidly growing tumour of more malignant behaviour than adenoid cystic carcinoma.
- Epidermoid carcinoma is often an undifferentiated, highly malignant tumour.

## Diagnosis and Management of Salivary Neoplasms

A swelling of a salivary gland, especially if localized, firm and persistent, may be a neoplasm and it would be prudent to seek a specialist opinion. A long history of gradual gland enlargement suggests a benign process, while pain or facial nerve palsy is ominous and suggests carcinoma (see above and Table 10). Some tumours may be small and the presentation may be of pain only.

Sialography may reveal an obvious filling defect or displacement of the

| | |
|---|---|
| ***Aetiology:*** | *Unknown* |
| ***Clinical features:*** | *Typically in parotid*<br>*Asymptomatic swelling and eversion of ear lobe*<br>*No xerostomia.*<br>*Pleomorphic adenomas rubbery and often lobulated*<br>*Malignant tumours painful and ulcerate* |
| ***Incidence:*** | *Rare; mainly older women* |
| ***Management:*** | *Surgical excision; radiotherapy also for some*<br>*Microscopy after gland excision (biopsy may allow seeding and recurrence)* |

***Table 10.*** *Salivary neoplasms.*

gland but is a relatively imprecise means of tumour detection. CT scanning is a more sensitive means of tumour detection. Ultrasonography has a limited application. Preoperative needle biopsy, sometimes CT guided, has a high tumour detection rate in experienced hands. The diagnosis can often be firmly established by open biopsy but is best carried out at the time of definitive operation, in order to avoid seeding malignant cells.

Early detection carries a good prognosis because most tumours metastasize late. Some tumours, such as adenoid cystic carcinoma, invade bone and neural tissues preferentially.

The treatment of choice for salivary gland tumours is surgical excision; radiotherapy is sometimes an adjunct.

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## BOOK REVIEW

**Essential Endodontology.** By D. Orstavik and T.R. Pitt Ford. Blackwell Science Ltd. (www.blackwell-science.com), 1999 (£79.50) ISBN 0-632-04089-0.

I have developed the technique, when I carry out book reviews, of comparing what I read in the book to the 'mission-statement' contained in the authors' preface. The two main authors, who are both leading and respected figures in the field of endodontology, make the following statement in the preface: "The study of the disease, apical periodontitis, has flourished as a field of research in the last quarter century. Information on its aetiology, pathogenesis, microbiology, epidemiology, treatment and factors affecting prognosis, has accumulated in the scientific literature. The time has come to present the state of the science of the subject in a text for graduate and undergraduate dental students as well as for practitioners and specialists." In the case of this book they have certainly fulfilled their statement of "presenting the state of the science of the subject".

The list of 19 internationally recognized contributors reads like a 'who's who' in endodontology, with an inevitable swing towards the Scandinavian. To balance things up, however, some of the Scandinavians have American addresses.

I was disappointed that there were several contentious issues that were not discussed. The reason for this is, presumably, that there is no scientific evidence, which is after all the very strength of this book: patency is one such issue. Given the plethora of new, mainly American, rotational preparation techniques that are usually taught involving patency, it would have been very useful to have seen how this stood up in the scheme of things. Again, I would have liked to see greater elaboration of working length estimation. The use of modern electronic apex locators are, after all, part of the accepted treatment technique, with substantial research material available.

There is an excellent chapter on the treatment of the exposed dentine pulp complex; when indirect pulp capping is being discussed, it includes a quote from the late Professor Nygaard Ostby: "to intend to leave caries permanently is malpractice and this kind of procedure should be pronounced indirect pulp crapping!"

Although contemporary dentine bonding was discussed, the pros and cons of acid etching deep dentine or pulp were not. This book does not set out to be a practical manual, but an elaboration of the acid etch dilemma would have been welcome.

These comparatively trivial points are the only aspects of this tome I can find to criticize. It is a really splendid book – I just wish it had been around when I was doing my postgraduate studies. It will make a very useful reference book, particularly with over two thousand references. It is, in essence, an endodontological encyclopaedia.

**Chris Emery**
**Endodontic Practitioner**