

## **Salivary glands**

### **Developmental disorders**

#### **Aplasia/agenesis**

Congenital absence of one or more of the major salivary glands can occur but is very rare. When it does it is usually the parotid gland that is affected.

#### **Duct atresia**

Again this disorder is extremely rare. Usually the submandibular duct in the floor of the mouth fails to canalate during embryological development. The newborn infant presents within 2 or 3 days of life with submandibular swelling on the affected side due to a retention cyst in the submandibular salivary gland.

#### **Congenital fistula**

Patients with branchial cleft anomalies present usually with unilateral painless swellings in the region of the parotid. Rarely are they bilateral. They form sinus tracts either in the crease behind the pinna or in front of the tragus. They discharge saliva intermittently. Abscess formation due to secondary infection may occur. Complete surgical excision of the sinus tract is essential. The dissection is often very extensive and full dissection of the facial nerve may be required.

#### **Ectopic and aberrant salivary tissue**

Ectopic salivary tissue can develop anywhere within the territory of the first and second branchial arches in the lateral neck, pharynx or middle ear. Salivary tissue is regularly found in lymph nodes within the neck and can be mistaken for metastatic disease when found in a neck dissection specimen.

Although rare, the most commonly recognised ectopic salivary tissue is the Stafne bone cyst. This presents as an asymptomatic clearly demarcated radiolucency at the angle of the mandible below the inferior dental canal. It is formed by an invagination into the bone of the lingual aspect of the mandible by an ectopic lobe of the adjacent submandibular salivary gland.

#### **Accessory lobes**

An accessory parotid lobe is the most common developmental anomaly. It occurs in as many as 20 per cent of subjects. Its position is constant arising from the horizontal component of the parotid duct as it crosses the masseter muscle. Its importance lies in the fact that any of the diseases that can affect the salivary glands may involve the accessory lobe and lead to diagnostic

confusion as the possibility is not considered. This is because the symptoms and signs are not within the normal anatomical territory of the parotid.

### **Inflammatory disorders** مهمه

#### **Viral**

##### **Mumps**

The mumps virus is a paramyxovirus and is the most common cause of acute painful parotid swelling affecting children. The disease starts with a prodromal period of 1 or 2 days during which the child experiences feverishness, chills, nausea, anorexia and headache. This is typically followed by pain and swelling of one or both parotid glands. The parotid pain can be very severe and is exacerbated by eating or drinking. Symptoms resolve spontaneously after 5—10 days.

In a classical case of mumps the diagnosis is based on the history and clinical examination. However, the presentation may be atypical or sporadic or have predominantly unilateral or even submandibular involvement. In this situation, paired blood specimens taken approximately 10 days apart are used to confirm the diagnosis. One episode of infection confers lifelong immunity.

A number of other viral agents —Coxsackie A and B, parainfluenza 1 and 3, enteric cytopathogenic human orphan viruses (ECHO) and lymphocytic choriomeningitis —can all cause identical signs and symptoms.

##### **Bacterial**

***Acute ascending bacterial sialadenitis*** affects mostly the parotid glands. The reduced salivary flow and oral sepsis result in bacteria colonising the parotid duct and subsequently involving the parotid parenchyma. With current medical practice and improved oral hygiene patients are rarely allowed to become dehydrated and this clinical pattern is uncommon. The typical patient presenting with an acute ascending bacterial parotitis now is an otherwise fit young adult with no obvious predisposing factors. The clinical presentation is of the onset of tender, red, painful parotid swelling over a few hours. There is associated malaise, pyrexia and often regional lymphadenopathy. Pain is exacerbated on attempting to eat or drink. The parotid swelling may be diffuse but often it is localised to the lower pole of the gland presumably because the infection tends to localise under the effect of gravity.

If the gland is gently 'milked' by massaging the cheek, cloudy turbid saliva can be expressed from the parotid duct and this should be cultured. The infecting organism is usually *Staphylococcus aureus* or *Streptococcus viridans*. Sialography must never be undertaken during the acute phase of infection as the retrograde injection of infected material into the duct system will result in

bacteraemia. Ultrasound imaging shows the characteristic dilatation of the acinae.

If the patient presents at an early stage before abscess formation, the infection can usually be controlled with antibiotics. In a patient not allergic to penicillin a combination of a broad-spectrum penicillin and a penicillinase-resistant agent is usually effective. If the gland becomes fluctuant indicating abscess formation, the pus must be drained. Occasionally it is possible to drain the abscess by aspirating the pus through a large-bore hypodermic needle but usually it is necessary to undertake formal surgical drainage under general anaesthesia.

**Chronic bacterial sialadenitis** is far more common in the submandibular salivary gland and it usually occurs secondary to chronic obstruction. Unfortunately the submandibular gland has a poor capacity for recovery following infection and, in most cases following control of any acute symptoms with antibiotics, the gland itself must be removed. During the operation great care must be taken not to damage the mandibular branch of the facial nerve when making the incision, the lingual nerve when mobilising the gland and clamping the duct and the hypoglossal nerve when separating the gland from the floor of the submandibular triangle.

### **Recurrent sialadenitis of childhood**

It is characterised by the rapid swelling of usually one parotid gland accompanied by pain and difficulty in chewing as well as systemic symptoms such as fever and malaise. Although each episode of parotid swelling is normally unilateral the opposite side may be involved in subsequent episodes. Each episode of pain and swelling lasts for 3—7 days and is followed by a quiescent period of a few weeks to several months. Occasionally episodes are so frequent that the child loses a considerable amount of schooling. The onset is usually between 3 and 6 years although it has been reported in infants as young as 4 months. The diagnosis is based on the characteristic history and is confirmed by sialography which shows a very characteristic punctate sialectasis often likened to a snow storm against a dark night sky.

Traditionally the episodes of parotitis have been treated with antibiotic and symptoms settle within 3—5 days on such a regime. Occasionally recurrent episodes are so frequent that prophylactic antibiotics are required for a period of months or years. Spontaneous resolution of symptoms seems to occur at puberty.

**Specific 'infections' (granulomatous sialadenitis)****Mycobacterial infections**

***Tuberculosis and nontuberculous parotitis*** typically presents as a tumour-like swelling of the gland. Symptoms are usually minimal with little pain and no pyrexia. Often the diagnosis is not suspected and the mass is excised by formal parotidectomy.

***Cat-scratch disease*** للأطلاع

Cat-scratch disease is caused by *Bartonella henselae*. Children are usually affected. Symptoms follow a scratch by a cat when a small pustule forms at the site of the scratch. There is an associated lymphadenitis usually affecting the cervical nodes and mild pyrexia and encephalopathy with occasional transient cranial nerve palsies. The parotid glands are swollen in 3 per cent of cases. The condition is self-limiting and resolves without treatment.

***Syphilis***

The glands can be involved in the acute early stages but are more often involved in tertiary syphilis with gumma formation, gland destruction and dense fibrosis.

***Toxoplasmosis***

Toxoplasmosis is due to the protozoan organism *Toxoplasma gondii*. In most cases infection is not recognised and is asymptomatic. When symptoms do occur the patient usually presents with lymphadenopathy and malaise sometimes accompanied by a headache and sore throat. The enlarged lymph nodes are rubbery and are not tender. On occasion the patient presents with isolated unilateral parotid swelling some weeks before the lymphadenopathy develops. In this situation a parotidectomy is often performed which leads to the diagnosis. Diagnosis is further confirmed by the detection of a positive Sabin—Feldman dye test on the serum. The disease follows a self-limiting course and resolves spontaneously after weeks or months. If symptoms are severe the patient is treated with a 3- or 4-week course of pyrimethamine and sulphadiazine.

***Deep mycoses*** للأطلاع

Fungal infections of the salivary glands occur only in immunocompromised patients and are most commonly seen in human immunodeficiency virus (HIV)-positive patients. Salivary gland involvement is usually just one manifestation of a more generalised infection. The patient presents with a tumour-like swelling of the affected gland. Often there is extensive central

necrosis. Fresh material is needed for culture and identification of the organism. Treatment is by appropriate systemic antifungal chemotherapy.

***Wegener's granulomatosis*** للأطلاع

Although the typical presentation is chronic granulomatous ulceration and destruction in the nasopharynx or sometimes the oral cavity, Wegener's granulomatosis can involve the major salivary glands. Diagnosis is based on the histological finding of necrotising arteritis often associated with numerous giant cells and granulomas. Pulmonary and renal involvement is very common. Treatment is by cytotoxic chemotherapy such as cyclophosphamide or azathioprine. The prognosis is poor.

***Granulomatous disease of minor salivary glands*** للأطلاع

Granulomatous cheilitis, Melkersson—Rosenthal syndrome (recurrent facial palsy/facial swelling/fissured tongue) and Crohn's disease all affect the minor salivary glands of the lips. Cheilitis glandularis is a rare disorder mainly of adult males in whom the lower lip becomes swollen and hard. The labial salivary glands become nodular and their orifices are inflamed and swollen.

***Allergic sialadenitis*** للأطلاع

A variety of potential allergens causing acute parotid swelling has been identified. Some foods, drugs (most frequently chloramphenicol and tetracycline), metals such as nickel and pollens have been incriminated.

***Radiation sialadenitis*** للأطلاع

Following the start of therapeutic irradiation when the parotid glands are within the radiation field the patient develops an acute parotitis usually after 24 hours. The glands are swollen and tender and there is a marked rise in salivary amylase and the salivary flow rate is reduced. The reaction is self-limiting and resolves after 2 or 3 days even though the radiotherapy continues. This reaction is quite distinct from the permanent radiation atrophy that occurs with therapeutic doses above 50 Gy, which develops progressively some weeks after the radiation has been completed.

***Human immunodeficiency virus-associated sialadenitis*** للأطلاع

Chronic parotitis in children is almost pathognomonic of HIV infection. In adults a sicca syndrome and lymphocytic infiltration of the salivary glands are more usual. The presentation of HIV-associated sialadenitis is very similar to classic-al Sjogren's syndrome. Dry mouth, dry eyes and swelling of the salivary glands together with lymphadenopathy suggest the diagnosis.

Histologically the condition closely resembles Sjogren's syndrome and differentiation may be difficult. However, autoantibodies including antinuclear, rheumatoid factor, SS-A and SS-B are absent unless the patient coincidentally has a connective tissue disorder. Acquired immunodeficiency syndrome (AIDS)-associated lymphoma presenting as salivary gland swelling has also been described.

Another presentation of salivary gland disease in HIV-positive patients is multiple parotid cysts causing gross parotid swelling and significant facial disfigurement. On imaging with computerised tomography (CT) or magnetic resonance imaging (MRI) the parotids have the appearance of Swiss cheese with multiple large cystic lesions. The glands are not painful and there is no reduction in salivary flow rates. Surgery may be indicated to improve the appearance.

### **Sialadenitis of minor salivary glands**

It occurs only on the hard palate in the molar region in the vault of the palate midway between the midline and the gingival margin. It is only seen in heavy smokers. It has a characteristic appearance which resembles a carcinoma with central ulceration and raised erythematous margins. The ulcer may be as much as 3 cm in diameter. As it closely resembles a carcinoma the diagnosis is often made on the basis of a surgical biopsy. The lesions are self-healing but often take 10—12 weeks to resolve.

### **Obstruction and trauma**

#### **Papillary obstruction**

Occasionally a rough upper molar tooth or an overextended denture flange will irritate the parotid papilla. If this is sufficient to cause ulceration with consequent inflammation and oedema this may obstruct salivary flow, particularly at meal times when the flow rate is increased. In this situation the patient has classical rapid onset pain and swelling at meal times.

If the trauma to the parotid papilla continues there will be progressive scarring and fibrosis in the soft tissues and permanent stenosis of the papilla can occur. A papillotomy will be required.

للأطلاع This is a simple procedure performed under local anaesthesia. A probe is inserted into the orifice of the papilla and with a scalpel blade the papilla is split open by incising down on to the probe. This lays open the papilla and divides the stenosis allowing free drainage of saliva.

**Stone formation (sialolithiasis) مهمه**

Eighty per cent of all salivary stones occur in the sub-mandibular gland, 10 per cent occur in the parotid, 7 per cent in the sublingual gland and the remainder occurs in the minor salivary glands. It is believed that the majority of stones occurs in the submandibular glands because their secretions contain mucus and the viscosity is higher. Eighty per cent of submandibular stones are radio-opaque and can be identified using plane radiographs. By contrast the majority of parotid stones are radiolucent and cannot be detected on plane radiography.

The classical presentation is of acute pain and swelling at meal times. Onset is rapid within a minute of starting the meal and the swelling resolves over a period of about 1 hour after the meal is completed.

However, this classical picture only occurs when the stone causes almost complete obstruction often when it is impacted at the opening of Wharton's duct. More often the stone causes only partial obstruction and is lying either within the hilum of the gland or within the duct in the floor of the mouth. In this situation the patient may complain of occasional swelling often with minimal discomfort or of a chronically enlarged mass in the submandibular triangle with episodes of dull aching pain. This results from chronic bacterial infection arising in an obstructed gland with salivary stasis and poor emptying. Often a salivary stone is totally asymptomatic and is discovered coincidentally during radiography for other reasons. If a stone is identified on plane radiographs, no other investigation is necessary. Parotid stones often impact at the parotid papilla or alternatively take on a 'stag-horn' shape and form at the junction of the two main collecting ducts and the Stenson's duct. If the stone is trapped at the duct papilla it can often be released by gently probing and carrying out dilatation of the papilla. It may be necessary to slit the duct in order to release the stone.

للأطلاع If the stone is lying in the submandibular duct in the floor of the mouth anterior to the point at which the duct crosses the lingual nerve (second molar region) the stone can be released by opening the duct longitudinally. It is important to pass a large suture around the duct proximal to the stone so that during the operative procedure the stone cannot be displaced backwards in the duct. Once the stone has been released the wall of the duct should be sutured to the mucosa of the floor of the mouth to maintain an opening for the free drainage of saliva. No attempt should be made to repair the duct wall as this will lead to stricture formation. A parotid stone located at the confluence of the collecting ducts can be released surgically by raising a preauricular flap, exposing the parotid duct and again incising it longitudinally to release the stone.

**Obstruction in and around the duct wall مهمه**

Scarring and fibrosis in the duct wall stricture formation will also result in obstruction to salivary flow. It often results as a complication of long-standing sialolithiasis but it may occur as a result of trauma particularly to the floor of the mouth. Subsequent healing and scarring can result in a stenosis of the duct. In patients with masseteric hypertrophy the parotid duct may be stretched around the anterior border of the muscle and this may cause obstruction of salivary flow at meal times.

**Mucoceles**

Mucus retention cysts and mucus extravasation cysts arise in the minor salivary glands as a result of mechanical damage to the gland or its duct. The common sites are on the mucosal aspect of the lower lip particularly in patients with a deep overbite and in the buccal mucosa posteriorly where an upper wisdom tooth is erupting buccally. Typically the patient presents with a history of recurrent swellings that develop over days or weeks, rupture and then recur after a few weeks. The cysts rarely exceed 1 cm in diameter and are tense bluish sessile swellings. The treatment is not to the cyst itself but to the underlying minor gland which should be excised under local anaesthesia.

**A ranula** is no more than a large mucocele arising from the sublingual gland. Classically the ranula presents as a large tense bluish swelling in the floor of the mouth anteriorly often displacing the tongue. However, the ranula may push its way through the midline mylohyoid dehiscence in the floor of the mouth and enter the submental space presenting as a midline swelling in the upper neck. This is the 'plunging ranula'. The treatment of a ranula is excision of the sublingual gland.



### Salivary neoplasms

Salivary gland tumours can be classified into: **Important**

#### **Adenoma:**

- Pleomorphic adenoma: pleomorphic adenoma.
- Monorphic: warthin's tumour, adenolymphoma.

#### **Carcinoma:**

✚ Epithelial : Low or high grade

- Low grade: acinic cell carcinoma, adenoid cystic carcinoma, mucoepidermoid carcinoma.
- High grade: adenocarcinoma, squamous cell carcinoma, mucoepidermoid carcinoma.

✚ Non epithelial tumour: haemangioma, lymphangioma.

✚ Lymphoma: non-Hodgkin's lymphoma, lymphoma in sjogren syndrome.

✚ Tumour like lesions: solid tumours as adenomatoid hyperplasia & benign lymphoepithelial lesion, cystic tumours as salivary gland cysts.

#### **Clinical features**

**Nearly all salivary neoplasms** present as slowly growing masses which have often been present for several years. Even malignant salivary tumours usually grow slowly. Unfortunately pain is not a reliable indication of malignancy. Certainly if a malignant salivary neoplasm is invading a sensory nerve pain or paraesthesia can occur but frequently at surgery one sees a nerve macroscopically invaded by tumour but which has been functioning normally pre-operatively. Furthermore benign tumours often present with pain and aching in the affected gland presumably due to capsular distension and possibly also due to an element of outflow obstruction. Therefore the only reliable clinical indication of malignancy is facial nerve palsy in the case of the parotid, induration and/or ulceration of the overlying skin or mucosa and regional lymphatic metastasis.

**Epithelial tumours**

Seventy-five per cent of all salivary epithelial tumours arise in the parotid glands and, of these, only 15 per cent are malignant. Just over 10 per cent occur in the submandibular glands and, of these, approximately one-third are malignant. About 15 per cent of tumours occur in the minor salivary glands and nearly half of these will be malignant. Tumours arising in the sublingual glands are rare (0.3 per cent) but nearly all of them will be malignant. Both benign tumours adenomas and malignant tumours carcinomas occur.

**❖ Tumours of the minor salivary glands:**

They are 90% malignant. Common sites are the upper lip, palate, & retro molar regions. Less commonly nasal & pharyngeal cavities. Benign tumours present as painless firm, slow growing swelling in which the overlying ulceration is extremely rare.

If benign tumour of the salivary glands of the upper lip, do excision of the tumour which include the overlying mucosa with primary closure.

If benign tumour of the salivary glands of the palate is <1cm, then do excisional biopsy & the defect is allowed to heal by secondary intention. If >1cm, do incisional biopsy to establish the diagnosis before excision.

Malignant minor salivary gland tumours have firmer consistency & undergo necrosis and ulceration as a late presentation. The overlying mucosa may be colored pink, blue or black.

Malignant tumour of the palate treated by wide excision which may involve low level or total maxillectomy & the defect managed by prosthetic obstruction or immediate reconstruction.

**❖ Tumours of the sublingual salivary glands**

They are 90% malignant. Present as hard or firm painless swelling in the floor of the mouth. They require wide excision involving the overlying mucosa with the mass & simultaneous neck dissection with immediate reconstruction of the intraoral defect.

**❖ Tumours of the submandibular salivary glands**

They are 50% malignant. Present as slow growing painless swelling with in the submandibular triangle. Even malignant tumours can be slow growing and painless. Benign tumours can be painful due to capsular distension or outflow salivary obstruction.

***Clinical features of malignant submandibular salivary glands tumour are:***

1. Fascial nerve weakness.
2. Rapid enlargement of swelling.
3. Induration & or ulceration of the overlying skin.
4. Cervical lymphadenopathy.

***Investigations of submandibular salivary glands tumour include:***

1. **CT scan & MRT.** They are most helpful. They can show the tumour as being circumscribed (benign) or diffuse & invasive (malignant) & also shows the relationship of the tumour with other structures, this help planning surgery.
2. **FNA biopsy** is safe using needle gauge not >18G (have no risk of seeding the tumour).

Disadvantages of FNA are **1.** Rarely alter the surgical management **2.** Only influence the treatment planning if malignancy is confirmed on cytological analysis.

**Note:** Open biopsy is contra indicated. It causes seeding.

**Management:**

If the tumour is benign & small & is entirely encased within the submandibular gland parenchyma, we do intracapsular submandibular gland excision. If the tumour is large & benign & is beyond the submandibular gland, we do suprahyoid neck dissection preserving the marginal branch of the mandibular nerve, lingual nerve & hypoglossal nerve. This entails full clearance of the submandibular triangle.

If overt malignant tumour, do modified neck dissection or radical neck dissection which the latter necessitate sacrificing the lingual & the hypoglossal nerve, if the tumour is adherent to the deep bed of the gland.

**❖ Tumours of the parotid gland:**

Parotid gland is the most common site for salivary tumour. Most arise in the superficial lobe as slow growing painless swelling below the ear or in front of the ear or in the upper aspect of the neck.

Rarely arise from the deep lobe which then present as Para pharyngeal masses. Here it cause difficulty in swallowing & snoring & the examination will shows diffuse firm swelling in the soft palate & tonsil.

80-90% of parotid gland tumours are benign mainly pleomorphic adenoma.

Tumours of the superficial lobe present as discrete mass with infiltration in to the overlying skin or present as diffuse hard swelling of the gland or as an advanced disease with cervical lymphadenopathy or as fascial nerve weakness (mostly indicate malignancy).

Investigations include CT scan & MRI (most helpful imaging) and FNA which aid obtaining preoperative diagnosis. Open biopsy is contraindicated.

**Management:**

All space occupying lesions of the superficial lobe of the parotid gland should be managed by superficial parotidectomy & not by enucleation of the swelling, even if benign lesion is suspected.

The aim is to remove the tumour with cuff of normal surrounding tissue.

***Complications of superficial parotidectomy include:***

1. Haematoma
2. Infection
3. Temporary and permanent facial nerve weakness
4. Facial numbness
5. Numbness of the ear lobe due to transaction of the great auricular nerve
6. Frey syndrome.

**Adenomas**

Of the variety of benign adenomas that has been described only two — the pleomorphic adenoma and Warthin's tumour—arise with any frequency.

**Pleomorphic adenomas**

At least 75 per cent of all parotid tumours and more than 50 per cent of all submandibular gland tumours will prove to be benign pleomorphic adenomas. It accounts for rather less than 50 per cent of minor gland tumours. Clinically the tumour has the texture of cartilage and has an irregular and bosselated surface. In the palate, the overlying mucosa is rarely ulcerated. Very rarely

after a number of years the tumour may undergo malignant change and for this reason all patients presenting with pleomorphic adenomas should be advised to undergo surgical removal of the tumour. This tumour which is only poorly encapsulated is very tense and if an incision is made into it the contents of the tumour burst into the surrounding tissue planes and it is impossible to eradicate the microscopic spillage of tumour cells. If this happens the patient will develop multiple local tumour recurrences over many years unless they are subjected to radical postoperative radiotherapy, which is best avoided in the management of benign disease. *Clearly if there is skin infiltration or ulceration an open biopsy is essential to establish a preoperative diagnosis upon which to plan surgery.* For tumours of the minor salivary glands particularly in the palate there is a much higher chance of the tumour being malignant and as it is not necessary to open up other tissue planes to gain access to the tumour as open incisional biopsy is important.

***Fine needle aspiration (FNA) biopsy is a safe alternative to open biopsy of a major gland.*** Evidence suggests that provided the needle gauge does not exceed 18 G there is no risk of seeding viable tumour cells. Although advocates of this technique claim high accuracy and specificity, there is inevitably a high risk of sampling error.

### **Warthin's tumour** مهم

The Warthin's tumour occurs only in the parotid gland where it accounts for approximately 15 per cent of all neoplasms. It is a disease of the elderly with a mean age of presentation of 60 years. Historically it had a male: female ratio of 4:1 but it is now becoming increasingly common in females. Recent evidence suggests that this tumour is related to cigarette smoking. It is also unusual in that in 10 per cent of cases it arises either bilaterally or is multicentric in the one gland. It does not undergo malignant change.

### **Carcinoma** مهم

The acinic cell carcinoma and the mucoepidermoid carcinoma, although undoubtedly malignant tumours with a potential for local invasion and metastatic spread, are frequently very low grade histologically and do not require the radical treatment needed for more aggressive tumours. Together they account for only 5 per cent of all tumours at any time. The mucoepidermoid tumour is much more common in the USA where it forms 10 per cent of all salivary neoplasms.

The adenoid cystic carcinoma, adenocarcinoma, squamous cell carcinoma and undifferentiated carcinoma are all aggressive malignant tumours that carry a poor prognosis regardless of treatment. The adenoid cystic carcinoma is characterised by relentless perineural spread along the cranial nerves and into the brain. However, it grows extremely slowly and although inevitably fatal

the 5- and 10-year survival figures are 70 per cent and 40 per cent, respectively. It is also unusual in having a predilection for distant metastasis to the lungs where it produces often multiple cannon ball tumours which remain symptomless for many years. The other carcinomas mentioned above have 5-year survival figures of around 25—35 per cent.

### Management of epithelial tumours

Both benign and malignant tumours arising in the parotid or submandibular glands are treated surgically by excision with surgical clearance. In the parotid gland this is by either superficial or total parotidectomy according to the location of the tumour. Unless the patient presents with facial nerve palsy (indicating a malignant tumour) the facial nerve is always preserved. In the submandibular gland treatment is always by excision of the gland. If when a definitive patho-logical diagnosis is received the tumour is malignant then the patient should receive radical postoperative radiotherapy. In those cases when the tumour involves skin or other adjacent structures or where there is lymphatic metastasis the patient should undergo radical excision, including a neck dissection and sacrificing any structures invaded by tumour, and again treated with postoperative radiotherapy.

***Pleomorphic adenomas*** arising in the minor salivary glands can be treated by local excision with a 5-mm margin. They do not invade periosteum and so in the palate they should be excised subperiosteally. Mucoepidermoid carcinomas and acinic cell carcinomas require rather more radical excision with a 10-mm margin and, when they are situated in the palate, palatal fenestration should be undertaken. Postoperative radiotherapy is only indicated for high-grade tumours or if the margins are not clear. For the remaining carcinomas arising in the minor salivary glands radical surgical excision and postoperative radiotherapy are indicated. In the palate this will be by maxillectomy.

### Nonepithelial tumours للأطلاع

A variety of nonepithelial tumours can arise in the salivary glands. Haemangiomas and lymphangiomas (cystic hygromas) occur in childhood. Haemangiomas occur mostly in the parotid and appear shortly after birth and grow progressively for several months. The majority undergoes spontaneous regression by 2 years of age. Females are more frequently affected. Lymphangiomas are less common. They may affect any of the salivary glands. They form sponge-like multicystic lesions. Fifty per cent are manifest by 12 months and 90 per cent will be evident by the end of the second year.

They do not undergo spontaneous involution. They frequently extend into the neck and mediastinum, and can undergo dramatically rapid growth causing respiratory obstruction. Treatment is by complete surgical excision but this may be technically very difficult.

Neurofibromas and neurilemmomas are the commonest nonepithelial tumours arising in adults. Clinically they are not distinguishable from other salivary tumours and are only diagnosed following surgery for a presumed epithelial tumour. Lipomas occur in the parotids particularly in adult males. They are treated by surgical excision.

### ***Malignant lymphomas*** للأطلاع

True extranodal lymphoma arising in the salivary glands usually the parotids is rare. More common is lymphoma arising from the lymph nodes either on the surface of the glands or within the parenchyma of the gland. Lymphoma also arises in the salivary glands, as a complication of HIV disease, and also in benign lymphoepithelial lesion and Sjogren's syndrome. The peak incidence for nonHodgkin's lymphoma is the sixth and seventh decades and females are twice as likely as males to be affected.

Salivary gland lymphomas usually present as firm painless swellings and more than 90 per cent occur in the parotids. If the lymphoma is confined to the parotid, treatment is by parotidectomy with postoperative radiotherapy. If there is evidence of spread beyond the salivary gland, treatment is by polychemotherapy according to the accepted protocols based on histological characterization.

### **Unclassified and allied conditions** للأطلاع

***Sialosis*** is an uncommon noninflammatory cause of salivary swelling usually affecting the parotid glands symmetrically. It is usually associated with metabolic and endocrine conditions such as alcohol abuse, diabetes mellitus, pregnancy, malnutrition and some drugs (usually sympathomimetics). It usually affects middle aged and elderly adults who present with bilateral soft parotid swellings. Biopsy of the glands reveals extensive fatty replacement but otherwise normal tissues. No treatment is known to be effective but sometimes parotidectomy is required to correct the disfigurement.

***Necrotising sialometaplasia, benign lymphoepithelial lesion, salivary duct cysts, Kuttner tumour and cystic lymphoid hyperplasia of HIV disease*** can all mimic salivary gland neoplasia. Similarly, branchial cysts and dermoids can present diagnostic confusion on occasion. As has already been

discussed both sarcoid and toxoplasmosis can present as parotid pseudotumours.

### **Degenerative conditions**

#### **Sjogren's syndrome للاطلاع**

Sjogren's syndrome is an autoimmune condition causing progressive destruction of the salivary and lachrymal glands. In 1933 Sjogren first described the association of keratoconjunctivitis sicca (dry eyes) and Xerostomia (dry mouth). Shortly thereafter he noted that these symptoms frequently occurred in patients with rheumatoid arthritis (RA). It has since been realised that Sjogren's syndrome can occur in association with any connective tissue disorder. Indeed the association is very much commoner in many connective tissue disorders than it is with rheumatoid arthritis. Only 15 per cent of patients with RA develop Sjogren's syndrome whereas 30 per cent of patients with systemic lupus erythematosus and nearly all patients with primary biliary cirrhosis do so. This combination of dry eyes, dry mouth and a connective tissue disorder most often RA as this is by far the most frequent connective tissue disorder is called secondary Sjogren's syndrome. The same combination of dry eyes and dry mouth but without association with a connective tissue disorder is known as Primary Sjogren's syndrome. Primary Sjogren's syndrome also differs from secondary Sjogren's syndrome by virtue of more severe xerostomia and xerophthalmia, more widespread dysfunction of other exocrine glands, a higher incidence of developing lymphoma and a different antibody profile.

Females are affected more often than males in the ratio of 10:1. Typically they are middle aged. The presenting complaint is usually of the underlying connective tissue disorder and only later does the patient become aware of a gritty feeling in the eyes due to dry eyes or of dry mouth. Occasionally there is enlargement of the parotid glands bilaterally and even more rarely the enlarged parotids are painful. Superinfection of the mouth with *Candida albicans* is frequent. Less frequently the patient develops bacterial sialadenitis due to ascending infection from the mouth. The condition does not invariably progress to total xerostomia and for any individual patient it is not possible to predict the outcome. The characteristic features of the condition are progressive lymphocytic infiltration, acinar destruction and proliferation of duct epithelium of all salivary and lachrymal tissue.

The diagnosis is often based on the characteristic history. No laboratory investigation is pathognomonic of either primary or secondary Sjogren's syndrome. However, the following investigations are usually undertaken:

1. Sialography reveals the progressive damage from punctate sialectasis to total parenchymal destruction leaving no more than a grossly dilated duct.



2. Labial salivary gland biopsy can be misleading particularly if only one minor gland is harvested. The characteristic lymphocytic infiltration is focal and a single gland may not show the changes. A minimum of three glands should be submitted to the pathologist.
3. Estimation of salivary flow may be unhelpful as the normal variation in flow rates makes the interpretation of the results difficult.
4. Viral staining of the cornea with rose Bengal and examination of the cornea with a slit-lamp is a very sensitive assessment of a dry eye.
5. Autoantibody screen.
6. Blood tests usually show a moderately raised erythrocyte sedimentation rate (ESR) and a mild microcytic anaemia (the anaemia of chronic disease).

**The management of Sjogren's syndrome** must be symptomatic. No known treatment modifies or reverses the xerostomia and keratoconjunctivitis sicca. Artificial tears are essential to protect the cornea. For the dry mouth various artificial saliva preparations are available but often the patient prefers to use frequent drinks and learns to carry a bottle of water with them at all times. If patients are to use saliva substitutes it is important that, if they are dentate, the product should not have a low pH and should contain fluoride as rampant dental caries are a frequent complication. There is also increased incidence of developing lymphoma in patients with Sjogren's syndrome. The risk is highest in those with primary Sjogren's syndrome. Monocytoid B-cell lymphoma is the usual complication. Its onset is often heralded by immunological changes (falling immunoglobulin levels, falling titre of rheumatoid factor, rising B2-microglobulin titre, rising serum macroglobulin titre and the appearance of monoclonal light chains in the serum and urine), lymphadenopathy and weight loss.

#### **'Benign' lymphoepithelial lesion** للاطلاع

The term 'benign lymphoepithelial lesion' was coined by Godwin in 1952. Use of the word 'benign' to describe the lesion is misleading as approximately 20 per cent of patients with benign lymphoepithelial lesion or Sjogren's syndrome ultimately develop lymphoma. Histologically it is not possible to distinguish benign epithelial lesions from Sjogren's syndrome. Both are characterised by lymphocytic infiltration, acinar atrophy and ductal epithelial proliferation. Indeed they may well be manifestations of the same condition.

Clinically benign lymphoepithelial lesion presents as diffuse swelling of the parotid. The swelling is firm and often painful. In 20 per cent of cases the parotid swelling is bilateral. Eighty per cent of patients are female and most are over 50 years old at presentation. Often there is an associated connective tissue disorder and the risk of developing lymphoma is particularly high in

those with rheumatoid arthritis. Most patients will be treated by parotidectomy in order to establish the diagnosis but if any parotid remnants are left, the swelling may recur again with the risk of lymphomatous change. Prolonged follow-up is essential.

**Mikulicz' syndrome** للأطلاع

In 1888 Mikulicz described benign, asymptomatic, symmetrical enlargement of the lacrimal and salivary glands. His original publication described a series of patients who clearly had a variety of different conditions. Benign lymphoepithelial lesion, Sjogren's syndrome, lymphoma, lymphocytic leukemia, sarcoid and Sialosis can all present in this way. The term Mikulicz' syndrome is not helpful and should not be used.

**Xerostomia** للأطلاع

A complaint of dry mouth is common. It seems to be particularly frequent in postmenopausal women who also complain of a burning tongue or mouth. Normal salivary flow decreases with age in both men and women. The situation is further confused as patients with Sjogren's syndrome are frequently unaware of having a dry mouth and patients who complain of dry mouth frequently have normal salivary flow rates. The most common causes of xerostomia in order of frequency are:

- Chronic anxiety states and depression;
- Dehydration;
- Drugs — many drugs have been implicated in causing xerostomia as an undesirable side effect;
- Salivary gland diseases as described earlier.

Xerostomia can be difficult to treat. Treatment is aimed at the relief of symptoms and the avoidance or control of complications. Frequent sips of water help most patients. Artificial salivas are not well accepted but their lubricant properties may be particularly useful at meal times. Cholinergic drugs such as pilocarpine can be tried but their side effects — diarrhoea and pupillary dilatation often outweigh any benefit.

Rampant caries and destructive periodontal disease are major complications due to oral infection. Meticulous oral hygiene and the weekly use of topical fluoride are essential. There is a high incidence of oral candidiasis and antifungal drugs are necessary.

**Sialorrhoea** للاطلاع

Some drugs and painful lesions in the mouth increase salivary flow rates. In normal health this is rarely noticed as the excess saliva is swallowed spontaneously. 'False ptilyism' is more common and is a well-recognised delusional symptom or occurs due to faulty neuromuscular control leading to drool-ing despite normal saliva production. Uncontrollable drooling is usually treated surgically. As the submandibular gland contributes most resting saliva, attention is directed at these glands bilaterally. The submandibular ducts can be mobilised and repositioned in the base of the anterior pillars of the fauces. Alternatively the two glands may be excised.

**Surgery of salivary gland disease** للاطلاع

The most common indication for removal of the sublingual salivary gland is in the management of a ranula, which is a mucous extravasation/retention cyst of the gland. Neoplasms of the sublingual gland occur only rarely but nearly all tumours at this site will be malignant. In this situation sur-gery is the same as that for any other malignancy in the floor of the mouth — resection with a clear margin often involving the mandible and when necessary en bloc with a neck dissection. Before an incision is made it is helpful to infiltrate the floor of the mouth with a local anaesthetic containing a vasoconstrictor. For simple excision of the sublingual gland, a linear incision is made in the floor of the mouth parallel to and just lateral to the submandibular duct, with care taken not to extend the incision more posteriorly than the first molar tooth so as to avoid damage to the lingual nerve. The incision should open the cavity of the ranula and allow the mucinous contents to be aspirated. The submandibular duct is now carefully identified and retracted medially. Stay sutures passed through the margins of the mucosa are helpful to aid retraction. Using blunt dissection with scissors the lingual nerve is identified. The sublingual gland which lies adjacent to the inner cortex of the mandible is then mobilised and its multiple ducts which drain into the submandibular duct are divided carefully in order not to damage the duct itself. The anterolateral part of the sublingual gland may be attached to the periosteum of the mandible by fibrous tissue and this must be divided carefully. Following removal of the gland, the mucosa of the floor of the mouth is loosely closed with two or three plain gut sutures. When sublingual gland excision is necessary for a tumour, it should be removed with a wide margin including a rim resection of the mandible.

**Complications** للأطلاع

Damage to the lingual nerve posteriorly or the submandibular duct medially is avoided by careful surgical technique. Meticulous haemostasis is required to avoid a postoperative haematoma in the floor of the mouth.

**Submandibular gland excision** للأطلاع

The patient is positioned supine on the operating table with moderate neck extension and the chin rotated to the opposite side. It is helpful to have head-up tilt on the operating table as this reduces venous engorgement. Following routine skin preparation and draping the incision is mapped out. The line should run within a skin crease in the neck at least 3 cm below the lower border of the mandible in order to avoid risk of damaging the mandibular branch of the facial nerve as it loops down below the lower border of the mandible. The incision should be approximately 7 cm long. The incision line is then infiltrated with conventional dental local anaesthetic solution containing 2 per cent lignocaine and 1:80 000 adrenaline. This results in some vasoconstriction which limits capillary ooze and helps to define tissue planes.

The incision is made with either a number 15 blade or a fine cutting diathermy whilst the assistant puts tension across the incision line. The incision is made directly down to platysma. The subcutaneous fat is stripped with firm pressure and a swab from the underlying muscle for approximately 1 cm on each side of the incision as this facilitates a layered closure later. The underlying platysma is then incised to the full extent of the skin incision again with either a blade or cutting diathermy. The assistant can now retract the wound margins using 'cat paws' or Allis forceps applied to the cur edge of the platysma muscle (never the skin edges!).

The underlying investing layer of the deep cervical fascia is next divided, preferably with scissors, after the fascia is first tented outwards with toothed forceps. Often the fascia consists of a series of separate laminae like an onion skin but occasionally it is composed of a single thicker sheet. Again the fascia should be divided along the full length of the incision to avoid the operative field becoming ever smaller.

Posteriorly, the fascial incision approaches the angular tract where the deep cervical fascia splits to form the investing layer that has just been incised and the deeper layer that forms the floor of the submandibular triangle containing the submandibular gland.

The mandibular branch of the facial nerve normally runs on the deep aspect of the investing layer of fascia although occasionally it lies between the

platysma and the fascia. Great care must be taken to protect the mandibular branch.

The anterior facial vein which lies in the connective tissue overlying the submandibular gland -is clamped, divided and tied. The loose connective tissue is separated with scissors to expose the submandibular gland. The dissection from now on continues on the capsular surface of the gland. For chronically infected glands there is frequently extensive fibrosis, and care and patience are required to maintain this plane. For all tumours contained within the submandibular gland capsule, this plane is safe as it forms an effective barrier. For malignant tumours that have infiltrated beyond the capsule, a full submandibular clearance, usually as part of a neck dissection, and often including the periosteum of the lower and inner aspect of the mandible, is needed.

The anterior pole of the superficial lobe of the submandibular gland is first mobilised and retracted upwards with Allis forceps (Fig. 42.18). This reveals the posterior belly of the digastric muscle which is then gently retracted downwards with a small Langenbeck retractor. This exposes the facial artery which emerges from behind the stylohyoid muscle and passes upwards and forwards to enter the deep surface of the submandibular gland. The artery is then clamped, divided and tied. Great care must be taken to secure the proximal ligature. As the vessel is divided it retracts out of sight and, if the ligature slips, the bleeding end of the vessel can be very difficult to identify.

The course of the facial artery is variable. Often it deeply penetrates the substance of the gland to emerge again at its upper border. Sometimes the artery lies in a groove in the deep aspect of the gland. The dissection in the plane of the submandibular gland capsule continues to mobilise the anterior pole of the superficial lobe of the gland, which is then gently retracted posteriorly. During this dissection a number of small arteries and veins will be identified entering the gland. These should be carefully clamped, divided and tied or diathermised according to their size. As the dissection continues posteriorly along the lower border of the mandible, the facial artery and anterior facial vein are encountered as they hook around the mandible. The vessels are again clamped, divided and ligated at this point.

At this stage in the operation, the anterior pole of the superficial lobe of the gland can be retracted posteriorly to reveal the groove between the superficial and deep lobes of the submandibular gland. The posterior border of the mylohyoid muscle lies within this groove. It is gently freed with scissors and then retracted forwards with a Langenbeck retractor. The deep lobe of the submandibular gland can now be mobilised either with a finger or by opening the blades of the scissors applied to the surface of the gland. On the deep aspect of the deep lobe, one or two small veins may be encountered running

from the gland through the underlying hyoglossus into the lingual veins. If these veins are not tied or adequately diathermised, troublesome bleeding may be encountered.

The submandibular salivary gland can now be pulled downwards revealing the V-shaped lingual nerve. The apex of the V is the point at which parasympathetic secretomotor fibres tether the lingual nerve to the salivary gland. It is very important to identify carefully the V of the lingual nerve and its parasympathetic fibres as the latter must be transected to free the gland. As these fibres are cut, the lingual nerve springs forwards. Finally the submandibular duct is clamped, divided and tied as far forward as possible with just enough left to drain the major sublingual gland which empties into the duct. A thin layer of loose connective tissue remains in the gland bed overlying the hypoglossal nerve.

The wound is inspected for any bleeding points, a vacuum drain inserted and the wound closed in layers using a subcuticular suture to close the skin. The wound edges are reinforced with skin closure tapes.

### **Complications**

- 1) Three cranial nerves are at risk during removal of the sub-mandibular salivary gland; the mandibular branch of the facial nerve, the lingual nerve (a branch of the third division of the trigeminal nerve) and the hypoglossal nerve.
- 2) When chronic infection and subsequent fibrosis have occurred, it is sometimes difficult to identify the lingual nerve and the deep aspect of the deep lobe may be attached to the hypoglossal nerve. At these stages of the operation, the surgeon must be convinced that these structures have been identified before using any sharp dissection.
- 3) Meticulous haemostasis is required throughout the operation as many of the vessels entering and leaving the sub-mandibular gland are only apparent when the gland is under traction and as soon as they are divided the vessels retract into the adjacent muscle planes.

### **Parotidectomy** للأطلاع

Treatment of parotid tumours is by superficial parotidectomy for all benign tumours in the superficial lobe and total parotidectomy for all benign deep lobe and dumb-bell tumours. Such tumours including deep lobe tumours should never be approached from the pharyngeal aspect. The facial nerve is preserved in all cases.

The prognosis for malignant parotid tumours is poor. There is little evidence that radical parotidectomy, which includes sacrificing the entire facial nerve, adds significantly to the patient's survival. It does, however, considerably

increase the morbidity. For this reason, superficial or total parotidectomy for malignant tumours is undertaken with preservation of those branches of the facial nerve not macroscopically invaded by tumour. This is followed in all cases of malignant parotid tumours by radical radiotherapy.

Similarly, 'supraradical' surgery for adenoid cystic carcinomas is not advocated. This tumour, although probably always fatal in the long term, is compatible with a useful 10-year survival rate. It is difficult, therefore, to justify extensive mutilating surgery without offering a cure. Adenoid cystic carcinomas whose macroscopic margins remain within the parotid are treated by total parotidectomy followed by radical radiotherapy. For more extensive tumours, radical dissection with as wide a margin as is anatomically appropriate whilst being compatible with reasonable rehabilitation followed by radical radiotherapy will ensure excellent local control of tumour. The radiotherapy field should include the skull base in order to control the perineural tumour extensions.

For any malignant parotid tumours with skin involvement, facial nerve weakness, mandibular invasion, extension into the infratemporal fossa or lymph node metastasis, radical resection often in continuity with radical neck dissection must be undertaken with reconstruction with the use of appropriate flaps and followed by radical postoperative radiotherapy.

### **Surgical technique** للأطلاع

Whenever the facility is available and the patient fit, hypotensive anaesthesia is used, as this considerably reduces oozing and thus makes it easier to trace the facial nerve fibres. The incision line is infiltrated with lignocaine hydrochloride and 1:80 000 adrenaline and the incision made with a knife or fine cutting diathermy. Following a preauricular incision extending downwards to continue in a suitable skin crease in the neck, the skin flap is raised in the plane of the pre parotid fascia and then held forward by suturing the margins for the flap to the adjacent towels. The blood-free plane anterior to the external auditory meatus is opened up by blunt dissection and this leads the surgeon down to the base of skull just superficial to the styloid process and the stylomastoid foramen. This plane is then gently opened up in an inferior direction by blunt dissection until the trunk of the facial nerve is seen. With large posterior tumours this plane may be difficult to open up. In this situation it is helpful to identify the posterior belly of the digastric muscle in the cervical extension of the incision. The anterior border of the sternocleidomastoid muscle is mobilised and retracted inferiorly to display the digastric muscle beneath it. This manoeuvre necessitates sectioning the great auricular nerve. The posterior belly of the digastric is traced upwards and backwards to its insertion on to the mastoid, which lies immediately below the stylomastoid foramen, thus leading the operator to the facial nerve from below.

There are four anatomical landmarks leading to the identification of the trunk of the facial nerve as it leaves the stylomastoid foramen (Fig. 42.21).

1. The cartilaginous external auditory meatus forms a pointer' at its anterior, inferior border indicating the direction of the nerve trunk.
2. Just deep to the cartilaginous pointer is a reliable bony landmark formed by the curve of the bony external meatus and its abutment with the mastoid process. This forms a palpable groove leading directly to the stylomastoid foramen. Unfortunately this groove is filled with fibrofatty lobules that often mimic the trunk of the facial nerve which can lie as much as 1 cm deep to this landmark.
3. The anterior, superior aspect of the posterior belly of the digastric muscle is inserted just behind the stylomastoid foramen.
4. The styloid process itself can be palpated superficial to the stylomastoid foramen and just superior to it. The nerve is always lateral to this plane and passes obliquely across the styloid process. A branch of the postauricular artery is usually encountered just lateral to the nerve.

Once the facial nerve trunk has been identified the superficial lobe of the parotid can be exteriorised by opening up the plane in which the branches of the facial nerve run between the two lobes by blunt dissection. Initially, as it leaves the stylomastoid foramen, the trunk of the facial nerve turns abruptly to become more superficial and also divides into the larger zygomaticofacial trunk and smaller cervicofacial trunk. The five main branches of the nerve are then followed peripherally through the parotid until the superficial lobe is completely freed. This part of the operation is performed using fine scissors, opened up in the plane of the facial nerve branches, with care always taken to identify the nerve fibre before dividing parotid tissue.

During the lower part of the dissection, branches of the posterior facial vein will be encountered immediately deep to the marginal mandibular branch. Great care must be taken when vascular clamps are applied to these branches to avoid damaging the facial nerve.

If the superficial parotidectomy is being performed for chronic infection, the duct should be tied off as far forward as possible to prevent recurrent ascending infection from the oral cavity.

If the tumour lies in the deep lobe of the gland a conventional superficial parotidectomy is performed as described. Next, the branches of the facial nerve are mobilised and lifted on nylon tapes to enable the deep lobe to be freed around its margins and removed when the mass is dropped downwards.



As this space is wedge shaped with its apex superior, it is almost invariably possible to do this. The deep lobe is covered by a capsule (the deep layer of the deep cervical fascia which splits to envelope the parotid) and is surrounded by the parapharyngeal fat. Thus, it is relatively easy to mobilise the deep lobe by blunt dissection with either scissors or a finger. Only very rarely is it necessary to perform a mandibulotomy to gain access to the deep lobe.

Very rarely most often after recurrent infection with fibrosis or previous radiotherapy — the trunk of the facial nerve cannot be confidently identified. In this situation the peripheral branches of the nerve are identified at the anterior border of the parotid and traced centrally towards the stylomastoid foramen.

Following removal of the parotid gland the blood pressure is returned to normal, all bleeding points are controlled, a vacuum drain is placed and the wound closed in layers. A pressure dressing is then applied for 48 hours.

### **Complications**

**Permanent facial nerve paralysis** following superficial or total parotidectomy is rare except when branches of the facial nerve have been deliberately sacrificed. When the facial nerve or its branches are sacrificed as a result of macroscopic tumour involvement, an immediate nerve graft may be undertaken using conventional microneural techniques.

**Temporary weakness** due to neuropraxia occurs in approximately 30 per cent of operations but recovers rapidly, usually within 6 weeks. Anaesthesia of the skin flap slowly resolves as the sensory nerves regenerate from the periphery.

**Anaesthesia of the ear lobe** due to sectioning of the great auricular nerve can be troublesome, particularly in females who find it difficult to wear earrings. Recovery can take up to 18 months and sometimes is never complete.

**Gustatory sweating (Frey's syndrome)** is a regular sequel to parotidectomy occurring in up to 54 per cent of cases. Surgical manoeuvres to treat it once established are not successful and most patients either learn to live with it or alternatively use an antiperspirant containing aluminium chloride.

**Spillage of a benign pleomorphic adenoma** should not occur if a formal superficial parotidectomy is undertaken. However, there are four circumstances where even with meticulous surgical technique this can happen:

- ✚ Extremely large pleomorphic adenomas occupying the entire superficial lobe making mobilisation of the gland difficult;
- ✚ Tumours that are intimately associated with branches of the facial nerve requiring very delicate dissection along the capsule of the tumour to release the nerve;
- ✚ tumours with lobular extensions extending beneath the mastoid, zygomatic arch or mandible;
- ✚ Some tumours that are abnormally friable with even routine retraction of the superficial lobe resulting in rupture. If rupture occurs an extremely careful inspection of the wound must be undertaken and the area thoroughly irrigated. In all such cases postoperative radiotherapy should be undertaken in order to avoid multiple recurrences due to tumour seeding.

Other rare complications such as **sialocele** or **salivary fistula** occasionally follow parotidectomy. Both complications are managed conservatively and resolve spontaneously after days or weeks. Very rarely a parotid fistula persists despite attempts at surgical closure. In this situation postoperative radiotherapy will destroy the residual functioning acinar tissue and allow the fistula to close.

### Radiotherapy معلومه مهمه

Parotid tumours are often considered to be 'radioresistant'. This is not true: regression after radiotherapy is usually slow, but this reflects the slow cell turnover time of the majority of these tumours, rather than the inability of radiation to effect a cure. There are many reports of long-term local control of large inoperable tumours by radiotherapy. Nevertheless, the chance of successful radiotherapy does seem lower than in the case of squamous cell carcinoma, and therefore the primary treatment should be surgical wherever possible.

Radiotherapy is of value for the inoperable tumour, and also should be used postoperatively whenever there is a risk of incomplete excision such as rupture of a pleomorphic adenoma. It should also be used prophylactically to radical dosage following excision of any malignant parotid tumour. In cases where reoperation is required for recurrence or where there is gross residual tumour, radiation in high doses increases survival significantly.

Adenoid cystic carcinoma has been reported to be the most, consistently radio responsive tumour type. In view of the propensity of this tumour for later recurrence, it is doubtful whether high local control rates at 3 or 5 years really indicate radio curability.

A wide volume around the tumour should be irradiated, especially in the case of adenoid cystic carcinoma. A dose close to the limits of normal tissue tolerance is necessary.



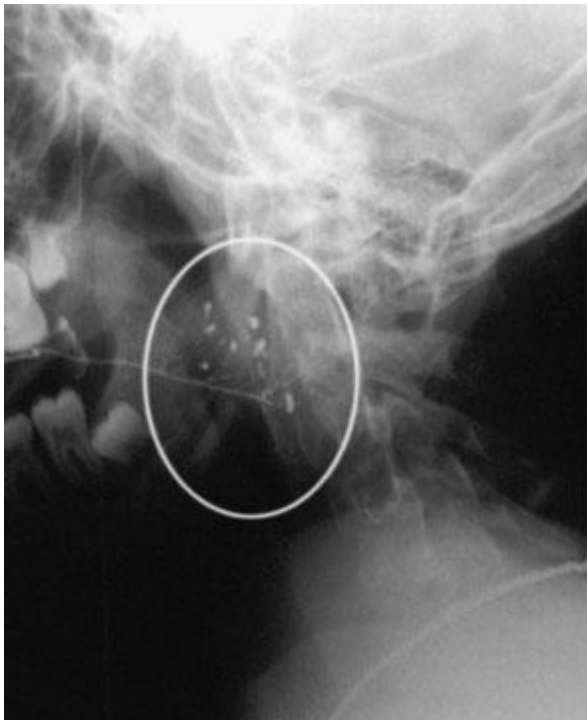
**Figure 47.1** Mucous retention cyst. A translucent swelling on the lower lip is typical.



**Figure 47.10** Acute left submandibular sialadenitis.



**Figure 47.18** Acute left bacterial parotitis.



**Figure 47.19** Characteristic 'snowstorm' appearance of recurrent parotitis of childhood (circled).



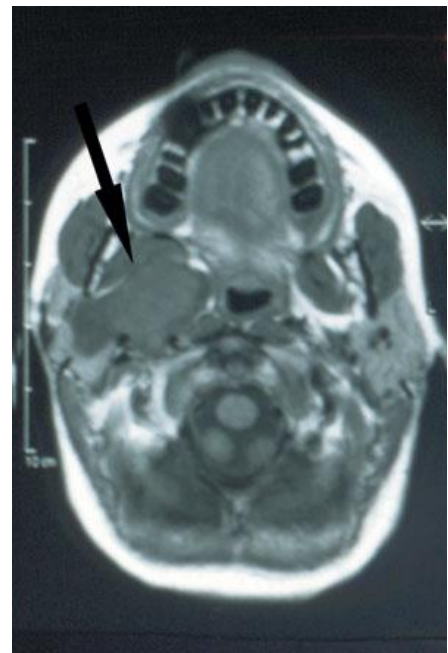
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**Figure 47.21** (a) Benign tumour of the left parotid gland producing characteristic deflection of the ear lobe. (b) Pleomorphic adenoma arising from the upper pole of the left parotid gland producing a pre-auricular swelling. (c) Deep lobe tumour of the right parotid presenting with a swelling of the right soft palate. (d) Magnetic resonance imaging (MRI) scan revealing a large deep lobe tumour (arrow) of the right parotid gland, occupying the parapharyngeal space.

**Muqdad fuad**