54 Disorders of the salivary glands

- Sublingual gland
- Submandibular gland
- TREATMENT OF SALIVARY GLAND MALIGNANCIES
- Trauma
- Xerostomia

Acute necrotising sialometaplasia

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This usually occurs on the palate and primarily a ff ects the minor salivary glands. It initially presents as a swelling that goes on to develop a central crater with rolled out margins, mimicking a malignant ulcer. Clinically it can be mistaken for a malignancy and the biopsy also raises doubt because of the presence of necrosis and hyperplasia. However, the lobular architecture of the glands is generally preserved and the lack of cellular atypia might help the pathologist to reach the right diagnosis. Ultimately , the lesion heals in a few weeks. The exact aetiology is unknown but is suspected to follow trauma or be caused by an injection to that area or by excessive vomiting.
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Benign tumours

Benign tumours

Pleomorphic adenoma These are the most common benign salivary gland tumours. They can occur at all ages, but are most commonly seen between the third and sixth decade. The average age of presentation is 45 years and they are more frequently seen in women. They occur most frequently in the parotid glands (>80%), but are also seen in the submandibular gland and hard palate. Pleo - morphic adenoma presents as a painless, well-defined solitary mobile mass with gradual progression over many years and can reach enormous proportions (Figur e 54.10). Occasionally they can present as metachronous and synchronous tumours. When they arise from the deep lobe of the parotid they may present as a paratonsillar bulge. A sudden increase in size or facial nerve palsy is associated with malignant transformation, which is rare. Treatment involves surgical excision with a cu ff of surrounding normal tissue, where possible, to include the pseudopods from the tumour capsule. Enucleation may result in capsular breach and tumour spillage, increasing the possi bility of local recurrence; it should be avoided. Histopathology On gross examination, pleomorphic adenoma presents as a well-circumscribed, nodular, firm mass with a white to tan cut surface, sometimes showing cartilaginous areas. Large tumours may show areas of degeneration and cystic changes. On microscopy , the tumour comprises mixed epithelial, myoepi thelial and stromal components. A spectrum of architectural and cellular features is seen, including oval, epithelioid, spindle shaped, plasmacytoid and clear cells, in variable amounts of myxoid to chondroid and hyalinised stroma. The pr esence of ductal atypia, di ff use fibrosis and necrosis should be evaluated further to rule out malignancy . In immunohistochemistry , luminal cells express CK7 (strong and di ff use) and myoepithe lial cells express p63, S-100, SOX10 and SMA. Warthin’s tumour Warthin’s tumour, also known as adenolymphoma or cysta denoma lymphomatosum, is a benign tumour composed of oncocytic epithelial cells lining ductal, papillary and cystic spaces in a reactive lymphoid tissue. They are the second most common benign salivary gland tumours (5–15%) and are mainly seen in older men, after the sixth decade of life. They ha ve been associated with cigarette smoking as well as radiation exposure. They are almost exclusively seen in the parotid gland, especially in the inferior pole, and are rarely seen in the periparotid nodes. They can occur synchronously or metachronously in the same or bilateral glands. They are also known to occur with other salivary gland neoplasms such as pleomorphic adenoma and salivary duct carcinoma. Clinically , they present as painless, slow-growing swellings. Facial palsy is rare. Malignant transformation is extremely rare (<1%) and can occur in both the epithelial (Warthin’s adeno carcinoma) and lymphoid (lymphoma) components. Complete surgical excision with an adequate margin is the treatment of tumours. Histopathology On gross examination, they are well circumscribed and ovoid to spherical with a cut surface showing solid and cystic areas containing mucoid to brownish fluid and papillary projections. On microscopy , the tumours have papillary and cystic struc - tures lined by bilayered oncocytic epithelial cells in a lymphoid stroma with germinal centres. The epithelium may show metaplastic changes, including squamous , sebaceous, ciliated and mucous cells.

Figure 54.10 Double head: large pleomorphic adenoma grown over 15 years.

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CLINICAL ANATOMY AND EMBRYOLOGY

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The parotid, submandibular and sublingual glands are three paired glands whereas there are innumerable minor salivary glands (Figure 54.1). The glandular architecture is essentially a series of ducts that open into the oral cavity and are surrounded by acini, which produce the saliva. The extracellular matrix includes the myoepithelial cells, myofibroblasts, immune cells, endothelial cells, stromal cells and nerve fibres. The parotid is ectodermal in origin, while the submandibular and sublingual glands are endodermal. The parotid represents the largest of the salivary glands and is situated in front of the external Niels Stensen , 1638–1686, Danish anatomist, natural scientist and theologist. Thomas Wharton , 1616–1673, physician, St Thomas’ Hospital, London, UK, described the submandibular duct in 1656. Summary box 54.1 Surgical anatomy of the salivary glands /uni25CF /uni25CF acoustic meatus between the ramus of the mandible and the sternocleidomastoid muscle. Each gland is encapsulated and is - composed of fat and cells that secrete mainly serous fluids. The major duct of the parotid gland is called Stensen’s duct, which opens into the vestibule of the mouth opposite the crown of the upper second molar tooth, while the submandibular duct is - Wharton’s duct, which opens into the floor of the mouth para - median to the frenulum. The parotid gland, being primarily serous, secretes watery saliva while the rest are mixed serous and mucinous glands.

The medical and surgical treatment of various pathologies • affecting the salivary glands Three pairs of major salivary glands – parotid, submandibular and sublingual Approximately 800 minor salivary glands

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Cytology

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Fine-needle aspiration cytology Fine-needle aspiration cytology (FNAC) is a widely available, simple and relatively safe diagnostic tool. It is used in the clinical setting of mass-forming lesions, often performed under ultrasound guidance. It can di ff erentiate between inflammatory conditions and neoplasms with a high sensitivity (96%) and specificity (98%). Systematic reviews have reported FNAC to have a high sensitivity (80%) and specificity (97%) in di ff erentiating benign from malignant lesions. The Milan system (Table 54.4) for reporting salivary gland cytopathology is an e ff ective tool to assess the adequacy of the cytopathology specimen and quantify the risk of malignancy . Core needle biopsy FNAC has a high specificity but a lower sensitivity in diagnos ing malignancies. Being operator dependent, there is a high variability in practice. In addition, in lymphoma or high-grade malignancies, ancillary studies such as flow cytometry and immunohistochemistry are required to confirm the diagnosis. In these settings, core needle biopsy has g reater diagnostic - - accuracy than FNAC. It provides more tissue for diagnosis and preserves the cellular architecture for further classification of malignancies. However, as it is a more invasive procedure, it is often reserved as a supplement to FNAC for problem solving. -

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Neoplasm IVA Benign <5% Conservative surgery or clinical follow-up a Conservative surgery IVB SUMP 35% a V Suspicious for 60% Surgery malignancy a (extent depends VI Malignant >90% Surgery on type and grade of malignancy) AUS, atypia of undetermined signi /f_i cance; FNAC, /f_i ne-needle aspira

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Ectopic aberrant salivary gland tissue

Ectopic/aberrant salivary gland tissue

The presence of ectopic salivary tissue in the mandible can manifest as circumscribed unilocular osteolytic radiolucency of the jaw , known as a Stafne bone cyst. They are possibly caused by either congenital entrapment of salivary tissue during mandibular development or pressure resorption from the adjacent ectopic salivary gland and facial artery . On orthopantomogram, the size ranges from 0.5 to 2 /uni00A0 cm, with a median of 1.2 /uni00A0 cm. They are mainly located in the posterior region of the mandible, especially between the first molar and the angle of the mandible. The other sites of ectopic salivary tissue include the cervical lymph nodes, middle ear, parathyroid glands, thyroid gland, pituitary gland, cerebellopontine angle and soft tissue medial to the sternocleidomastoid muscle. Ectopic/aberrant salivary gland tissue

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FURTHER READING

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INVESTIGATIONS Imaging

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Various modalities, from plain radiography to ultrasonography , CT , MRI and sialography are available to clinicians. In most cases, CT scans are considered superior for di ff erentiating neoplasms from inflammatory conditions, while MRI scans give better di ff erentiation between benign and malignant neoplasms. Ultrasonography is a very useful diagnostic tool, especially for lesions of major salivary glands. Acute inflammatory conditions may be picked up by enlarged glands with increased blood flow , compared with chronic inflammation, in which the glands may be smaller in size and hypoechoic. Sialolithiasis will present with distinct acoustic shadowing. Benign tumours such as pleo morphic adenoma are generally visualised as well-lobulated, hypoechoic lesions with some calcifications. Malignant tumours will have irregular shapes and a hypoechoic, inhomogeneous appearance with blurred margins . However, ultrasonography cannot adequately characterise lesions of the deep lobe of the parotid gland and when there is suspected involvement of the skull base; in these cases, cross-sectional imaging such as CT/ MRI is preferred. Computed tomography/magnetic resonance imaging scans These are the best tools for almost complete imaging of the salivary glands. They detect both cystic and solid masses with good accuracy as well as help in diagnosing and localising sialolithiasis. CT scans are especially useful in determining the extent of the tumour, erosion of surrounding osseous structures, extraglandular involvement and the presence of metastatic nodes. MRI scans, especially di ff usion-weighted (DW) and gadolinium-enhanced dynamic MRI, can di ff eren tiate benign and malignant neoplasms based on the apparent di ff usion coe ffi cient (ADC) values, peak enhancement and washout ratios. Positron emission tomography with computed tomography (PET-CT) scans These scans are used mainly in the detection of distant metas tases in high-grade malignancies or if the salivary glands are involved as the site of metastasis with an unknown primary . INVESTIGATIONS Imaging

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Immunological conditions

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Sjögren’s syndrome Sjögren’s syndrome (SjS) is a chronic autoimmune disease with lymphocytic infiltration and autoimmune injury to the salivary and lacrimal glands, leading to dryness of the mouth ology of the disease is evolving and is a complex interaction among genetic elements, environmental factors and abnormal host immunity . It mainly a ff ects women in the fourth decades, who present with enlargement of the salivary glands, especially the parotid. Typically there is a delay in the time taken to diagnosis of the disease. Studies have shown a delay of 2–6 months between the first consultation and diagnosis. SjS can be considered to have four stages: initiation stage, preclinical stage, asymptomatic SjS stage and overt SjS stage. Primary SjS is not associated with any illness or disease, whereas secondary SjS is associated with other autoimmune disorders, including systemic lupus erythematosus, rheumatoid arthritis and scleroderma (Table 54.1). W ith no diagnostic markers, the diagnosis is based on a set of five criteria proposed by the European Alliance of Associ ations for Rheumatology (EULAR). One of them is a biopsy of the sublabial salivary glands , which shows histological fea tures of focal lymphocytic sialadenitis. The criteria required f or diagnosis of SjS involve at least one focus score of more 2 than 50 lymphocytes per 4 /uni00A0 mm of parenchymal tissue. Other tests for early diagnosis are evolving; these include testing for increasing levels of biomarkers such as complement C3 and neutrophil elastase in saliva and tears. In addition, identifica tion of traditional antibodies such as anti-nuclear antibodies (anti-SSA/Ro or anti-SSB/La) and rheumatoid factor can also be used. However, a significant proportion of patients with SjS may be seronegative. Tr eatment depends on disease activity and the organs involved. Tear substitutes can be used and xerostomia is treated by dental and oral surgeons. Randomised controlled trials have not shown any benefit with hy droxychloroquine or disease-modifying antirheumatic drugs (DMARDs). Treat ment with glucocorticoids and/or immunosuppressant drugs should be considered in severe systemic manifestations. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Scleroderma This is an immunologically mediated disease with complex interactions between the vascular network, inflammatory markers and collagen tissue. It mainly a ff ects adults with a female preponderance. Multiple organs may be a ff ected by Christian Frederick Heerfordt , 1871–1953, Danish ophthalmologist, described this syndrome in 1909. Edward C Stafne , 1894–1981, dental surgeon, The Mayo Clinic, Rochester, MN, USA, described these cysts in 1942. is more commonly a ff ected, it can also involve the heart, lungs, kidneys and gastrointestinal tract. Salivary gland parenchyma to fifth may be replaced by collagen tissue, leading to clinical xero - stomia. Biopsy of a minor salivary gland may be useful for diagnosis. Sarcoidosis This is an inflammatory disorder characterised by multiple non-caseating granulomas in multiple systems. A dry cough, fatigue and shortness of breath are its main symptoms. The chest radiograph typically shows bilateral hilar lymphadenop - athy and reticular opacities in the lungs. Skin, heart, kidney , eyes, joints, exocrine glands and the central nervous system may be involved. The aetiology is unclear but one proposed - mec hanism is where an individual with a susceptible genotype is exposed to one or more potential antigens, resulting in a - sustained inflammatory response. The

disease most commonly occurs in those aged 20–60, in people of African or northern European descent and in those with a family history of the disease. Triggers are thought to include infection (mycobac - teria, propionic bacteria and viruses) or exposure to certain chemicals or dust. The function of an organ can begin to be - a ff ected as granulomas form and enlarge. For the salivary glands, the patient can pr esent with a local - ised tumour-like swelling, usually in the parotid – the so-called sarcoid pseudotumour along with xerostomia. In the absence of other disease, the diagnosis is usually made following surgi - cal excision for a presumed neoplasm. Heerfordt's syndrome is a rare manifestation of sarcoidosis that involves parotid swell - ing, anterior uveitis, facial palsy and fe ver. -

TABLE 54.1 Degenerative disorders of the salivary glands. Primary Sjögren's More severe xerostomia syndrome Widespread exocrine gland dysfunction No connective tissue disorder Secondary Sjögren's Male-to-female ratio 1:10 syndrome Middle age Underlying connective tissue disorder Benign lymphoepithelial Diffuse parotid swelling 20% lesion bilateral 5% develop lymphoma

Immunological conditions

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Introduction

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The parotid, submandibular and sublingual glands are the three paired major salivary glands; the minor salivary glands are multiple and situated mainly in the lips, buccal mucosa, tongue and palate, but they can be present anywhere along the aerodigestive tract. Obstructive and inflammatory conditions, tumours and autoimmune-mediated conditions a ff ect these glands. Saliva is an important secretion that performs a number of essential functions, such as clearing substances from the mouth, maintaining pH and tooth mineralisation and influ encing the oral microbiome, which protects the body and helps with wound healing. Moreover , saliva not only neutralises some harmful dietary components but also lubricates and hydrates oral mucosal surfaces . This has been the topic of study for cen turies, with the oldest reference to salivary glands, and more specifically to saliva, found in clay tablets at the Akka Library , which was created by the Assyrian King Assurbanipal from old Mesopotamia around 2500 /uni00A0 /b.sc/c.sc/e.sc . It even refer red to the use of the plant belladonna for curing excessive salivary flow .

Learning objectives

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To understand: The surgical anatomy of the salivary glands • The presentation, pathology and investigation of salivary • gland disease Learning objectives

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Malignant tumours

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Mucoepidermoid carcinoma Mucoepidermoid carcinomas are malignancies consisting of mucinous, intermediate and squamoid tumour cells in vari - able proportions. They are the most common salivary gland malignancies in children and young adults, with a peak inci - dence in the second decade of life. They are known to occur following radiation or chemotherapy in childhood. T hey occur - in both major and minor salivary glands, with the parotid being the most common site involved. The y generally present as soft to firm, painless masses with a gradual increase in size. The tumours are classified as low , intermediate or high grade based on histology . High-grade mucoepidermoid carcinomas tend to be locally aggressive with bone and/or skin involvement and nodal metastases. Distant metastases are seen mainly to the lungs. Complete surgical excision with wide margins is advo - - cated for mucoepidermoid carcinoma. Appropriate adjuvant radiotherapy is the treatment of choice for intermediate- to high-grade mucoe pidermoid carcinomas. On gross examination, the tumours are circumscribed or infiltrative and partially cystic. On histopathology , mucoepi - dermoid carcinomas have squamoid, mucin-producing and intermediate cells in variable proportions. There can be a - cystic and solid growth pattern. Low-grade mucoepider moid carcinomas are generally cystic, well circumscribed and rich in mucous cells. Intermediate-grade tumours are less circum - scribed and more solid, usually with a predominant intermedi - - ate cell component. High-grade mucoepidermoid carcinomas are usually solid, infiltrative and show nuclear atypia, mito - sis, necrosis, perineural invasion and lymphovascular emboli. Demonstra tion of at least focal intracellular mucin is essential for the diagnosis of high-grade mucoepidermoid carcinoma. Summary box 54.5 Mucoepidermoid carcinoma /uni25CF /uni25CF /uni25CF Adenoid cystic carcinoma - Adenoid cystic carcinoma is a slow-growing malignancy composed of both epithelial and myoepithelial cells and

Most common salivary gland malignancy Can occur in minor and major salivary glands Most common site: parotid

It has varying outcomes with good 5-year control but poor 10-year survival owing to the higher incidence of delayed distant metastases. They occur mainly in the fifth to sixth decades, with a slight female preponderance (1.5:1). Most of these malignancies occur in the major salivary glands, but they can also be seen in minor glands in the oral cavity , paranasal sinuses, tracheobronchial tree, etc. Most patients will present with slow-growing masses, with the presence of numbness, paraesthesia or pain. Facial and other neural palsies may be present depending on the site of the tumour. Nodal metastases are seen with high-grade lesions and asymptomatic distant metastases, especially lung metastases, are a frequent presentation. In addition, bone, liver and brain metastases are also seen. Radical surgical excision with or without adjuvant radiotherapy is the treatment of choice. Single-modality radio therapy is associated with inferior control outcomes. There is an emerging role of proton ion and carbon ion therapy , espe cially in unresectable/metastatic disease. Factors influencing surviv al include the tumour site, stage, nodal

disease, presence of perineural spread and grade of tumour. Grossly , adenoid cystic carcinoma presents as a poorly circumscribed, fir m, grey-white and solid mass. On histo pathology , it is an unencapsulated, infiltrative biphasic neoplasm with variable proportions of epithelial and myoepithelial cells and shows cribriform tubular and solid patterns. The cells show small, angulated, hyperchromatic nuclei with scant cytoplasm. The cribriform pattern is characterised by neoplastic cells arranged around small, sharply punched out cylindromatous spaces containing basophilic matrix. The tubular pattern shows bilayered tubules with a true lumen. The solid pattern is less common and shows sheets and nests of tumour cells without lumen formation. Perineural invasion is widely seen in adenoid cystic carcinoma. High-grade transformation can occur. Immunohistochemically , the ductal cells are positive for c-KIT , and myoepithelial cells are positive for p63 and SMA. Acinic cell carcinoma Acinic cell carcinoma is composed of neoplastic acinar cells. It is a low- to intermediate-grade tumour occurring mostly (90%) in the parotid gland. They typically present in the fifth decade and have a slight female predilection (1.5:1). They are generally slow-growing, painless, mobile, solitary tumours and rarely present with facial palsy . A small proportion may be high grade and may metastasise to cervical nodes and lung. Complete excision with an adequate margin is the recommended treatment. Recurrences can occur in cases of incomplete resection, deep lobe involvement and larger size tumours. On histopathology , the tumours show characteristic serous acinar cells and variable proportions of other cell types, including clear, vacuolated, intercalated duct-type oncocytic and hobnail features. They can have solid (most common), follicular or microcystic patterns with a prominent lymphoid infiltrate. Mitoses, necroses and nuclear pleomorphism are rare. Immunohistochemically , the neoplastic cells are positive for DOG1 and SOX10, whereas they are immunonegative for mammaglobin, di ff erentiating them from secretory carcinoma. Carcinoma ex pleomorphic adenoma (epithelial and/or myoepithelial) arises in association with primary or recurrent pleomorphic adenoma. The carcinoma component can be either purely epithelial or myoepithelial in presentation, with infiltration into the surrounding glandular and extraglandular tissue. It occurs mainly in the parotid gland, is more common in women and presents a decade later than pleomorphic adenoma (sixth decade). It often presents as a rapidly growing mass (within a longstanding swelling) associated with pain and facial palsy . Radical surgical excision with or without adjuvant radiotherapy is the treatment of choice. Local and distant metastases occur in 70% of cases with poor 5-year survival outcomes of 25–65%. On histopathology , the tumour shows variable propor - - tions of both pleomorphic adenoma and high-grade adeno - carcinoma such as salivary duct carcinoma or myoepithelial - carcinoma. It is subclassified as non-invasive or intracapsular (tumour is confined within pleomorphic adenoma), minimally invasive (tumour breaching the pleomorphic adenoma cap - sule) and widely invasive into adjacent salivar y gland and soft tissue. TP53 mutations and amplification of HER2 (in salivary - duct carcinoma) with a high degree of genetic instability and copy-number alterations are seen. Salivary duct carcinoma Also known as high-grade ductal carcinoma, salivary duct carcinoma is a high-grade adenocarcinoma, resembling high- grade mammary ductal carcinoma. The tumour may arise de novo or as a malignant component of carcinoma ex pleomor - phic adenoma. It is relatively less common and mostly arises in the parotid. It is commonly seen in elderly men in their sixth or seventh decade. Salivary duct carcinoma is an aggressive tumour presenting as a rapidly gro wing mass, often with facial palsy , pain and cervical lymphadenopathy . Complete excision with wide margins (total parotidectomy) with neck dissection is the treatment of choice. On histopathology , it resembles high-grade invasive ductal breast cancer with a large duct- like configuration with comedo necrosis and cribriform and Roman bridge-like features. Vascular and perineural invasion is seen. Salivary duct

carcinoma also shows androgen receptor and HER2 receptor positivity in a significant percentage of cases, making it a target for treatment, especially in the recur - rent/metastatic setting. It has a high predilection for local recurrence and regional and distant metastases and a poor overall survival. Malignant tumours

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Minor salivary glands

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- Minor salivary glands appear at about the 12th week of gesta - tion, forming directly from upper respiratory ectoderm. They develop as individual units with simple tubuloacinar systems. The minor salivary glands lack a distinct capsule and merge with the surrounding connective tissue in the submu - cosal region. They ar e widely distributed in the head and neck region and are mainly located (70–90%) in the oral cavity and - oropharynx, including the pala te, the tongue, the lips, the buc - cal mucosa and the retromolar trigone. The other sites are the nose, paranasal sinuses, pharynx and the larynx. They con - tribute 8–10% of the saliva and play a major role in saliva production during sleep. - Minor salivary glands
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Mucoceles

Mucoceles

Extravasation mucoceles and retention cysts are formed by - mucous extravasation. Both have similar clinical features but variable distinct pathogenesis (Figure 54.2). - Extravasation mucocele Trauma to the minor salivary gland duct causes accumulation of saliva in the surrounding connective tissue, followed by an inflammatory reaction. They occur commonly in children and adolescents and are mainly found on the lower lip. A ranula is a type of extravasation mucocele. Ranula (little frog) Ranulas, first described by Banister in his surgical compilation of 1585, are caused either by the rupture of the main duct or by the rupture of obstructed acini of the sublingual gland. They appear as a characteristic bluish swelling in the anterior floor of the mouth and resemble the belly or air sac of a frog. They can remain localised or insinuate through the mylohyoid muscle to present as a submental swelling called a ‘plunging ranula’. They are usually soft, fluctuant and painless unless infected. Imaging corroborates the clinical diagnosis and aspiration yields the thick sticky saliva that distinguishes them from a lymphangioma (Figures 54.3 and 54.4). John Banister , 1533–1610, English anatomist, surgeon and teacher. Henrik Samuel Conrad Sjögren , 1899–1986, Professor of Ophthalmology , Gothenburg, Sweden, described this condition in 1933. Treatment should include removal of the sublingual gland as this gland has multiple ducts and ranulas can recur if the gland is left behind. Incision, drainage and marsupialisation have low success rates. Injecting OK-432 at the local site pro - duces inflammation and fibrosis. Injection of botulinum toxin has shown a good success rate but needs further evaluation. Retention cyst These result from obstruction of the duct by periductal scars, sialolithiasis (salivary gland stone formation) or pressure from surrounding tissue. These cysts are mainly found in the ductal system of a minor salivary gland. The cyst cavity may contain fragments of a sialolith or mucous material. Some may regress spontaneously but are best treated by surgical excision.

Figure 54.2 Mucous retention cyst. A translucent swelling on the lower lip is typical. Figure 54.3 Ranulas in the /f_l oor of mouth, transillumination and specimen (courtesy of Dr Shirish Ghan, Nasik, India). Figure 54.4 Magnetic resonance imaging scan showing a ranula in the /f_l oor of mouth.

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NEOPLASMS OF THE SALIVARY GLAND

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Primary salivary gland neoplasms are extremely rare and form less than 3% of head and neck malignancies. The incidence is 0.4–13.5 cases per 100 /uni00A0 000 for benign neoplasms and 0.4–2.6 per 100 /uni00A0 000 for malignant tumours. These neoplasms present after the fourth decade and a ff ect both sexes equally . Warthin’s tumours are more common in older men, while pleomorphic adenomas are slightly more common in women. With a varied spectrum of pathologies, salivary gland neo - plasms present a diagnostic and therapeutic challenge. The World Health Organization (WHO) first classified these in - 1972, and its last update was in 2017 (Table 54.3). Most sali - - vary gland tumours (>80%) occur in the major saliv ary glands and the majority of them are benign. Minor salivary gland tumours, in contrast, are more likely to be malignant (>50%) (Figure 54.9). The commonest benign neoplasm is the pleo - morphic adenoma (mostly seen in the parotid glands), w hile the commonest malignant tumour is the mucoepidermoid carcinoma. Radiation exposure has been implicated in the develop - ment of both benign and malignant salivary gland tumours , while there is a strong association of smoking with Warthin’s tumour. Viral infections , environmental factors and industrial exposure, such as rubber manufacturing, nickel compounds and hair dyes, have been reported to be associated with the development of salivary gland tumours. /uni25CF Thomas Hodgkin , 1798–1866, curator of the museum and demonstrator of Morbid Anatomy , Guy’s Hospital, London, UK, described lymphadenoma in 1832. /uni25CF

Malignant epithelial tumours Acinic cell carcinoma Mucoepidermoid carcinoma Adenoid cystic carcinoma Polymorphous low-grade adenocarcinoma Epithelial–myoepithelial carcinoma Clear cell carcinoma, not otherwise speci /f_i ed Basal cell adenocarcinoma Sebaceous carcinoma Sebaceous lymphadenocarcinoma Cystadenocarcinoma Low-grade cribriform cystadenocarcinoma Mucinous adenocarcinoma Oncocytic carcinoma Salivary duct carcinoma Adenocarcinoma, not otherwise speci /f_i ed Myoepithelial carcinoma Carcinoma ex pleomorphic adenoma Carcinosarcoma Metastasising pleomorphic adenoma Squamous cell carcinoma Small cell carcinoma Large cell carcinoma Lymphoepithelial carcinoma Sialoblastoma Histological grades of salivary gland cancers High grade High-grade mucoepidermoid carcinoma Salivary duct carcinoma Adenoid cystic carcinoma Carcinoma ex pleomorphic adenoma Squamous cell carcinoma Anaplastic or undifferentiated carcinoma Malignant mixed carcinoma Figure 54.9 Mucoepidermoid carcinoma of

the palate. Benign epithelial tumours Pleomorphic adenoma Myoepithelioma Basal cell adenoma Warthin’s tumour Oncocytoma Canalicular adenoma Sebaceous adenoma Lymphadenoma Sebaceous Non-sebaceous Ductal papillomas Inverted ductal papilloma Intraductal papilloma Sialadenoma papilliferum Cystadenoma Soft-tissue tumours Haemangioma Haematolymphoid tumours Hodgkin’s lymphoma Diffuse large B-cell lymphoma Extranodal marginal zone B-cell lymphoma Secondary tumours Low to intermediate grade Low-grade mucoepidermoid carcinoma Acinic cell carcinoma Polymorphous low-grade adenocarcinoma Epithelial–myoepithelial carcinoma

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Warthin’s tumour Oncocytoma Canalicular adenoma Sebaceous adenoma Lymphadenoma Sebaceous Non-sebaceous Ductal papillomas Inverted ductal papilloma Intraductal papilloma Sialadenoma papilliferum Cystadenoma Soft-tissue tumours Haemangioma Haematolymphoid tumours Hodgkin’s lymphoma Diffuse large B-cell lymphoma Extranodal marginal zone B-cell lymphoma Secondary tumours Low to intermediate grade Low-grade mucoepidermoid carcinoma Acinic cell carcinoma Polymorphous low-grade adenocarcinoma Epithelial–myoepithelial carcinoma

Parotid gland

Parotid gland

Understanding a gland’s development gives us insight into the pathophysiology of the various disorders a ffl icting it. The parotid gland is ectodermal in origin and develops in the sixth week of gestation, when the epithelial buds invaginate from the oral mucosa into the surrounding mesenchyme. A tunnel develops from this groove and the gland is formed at its blind end by proliferation, budding and extensive branching. The secretory acini develop from the epithelial tissue, whereas the capsule of the gland and the connective tissue develop from the mesenchyme. The parotid, unlike other glands in the body , does not become encapsulated early to form a regular gland. The other structures in the vicinity , including the vessels, nerves and lymphatics, develop before encapsulation. The gland goes on to envelop the facial nerve, the terminal branches of the external carotid artery , the retromandibular and superficial temporal vein and the lymph nodes. The capsule thus merges with the investing fascia from the zygoma, over the temporomandibular joint and the masseter and reaches the styloid base, posterior digastric belly and the sternocleidomastoid muscle. The super ficial musculoaponeurotic system is closely approximated to this capsule. The parotid gland is, thus, irregular in shape and wedged in a recess between the ramus of the mandible, the base of the skull and the mastoid process . The parotid (Stensen’s) duct passes over the masseter muscle and enters the buccal mucosa through the buccinator muscle at the level of the upper second molar tooth. In some cases, an accessory gland is found along the course of the duct. Salivary tissue that is separated from the main parotid gland is referred to as an accessory parotid gland. These lie on the mas seter muscle in front of Stensen’s duct and have a secondary duct joining Stensen’s duct. According to autopsy studies, the incidence of an accessory parotid gland is 21–61%. Embryo logically , the growing , budding salivary glands originate from the oral cavity epithelium outwards into the mesenchyme as it di ff erentiates into the various facial structures. The late completion of parotid gland encapsulation needs to be con sidered when planning surgery for accessory gland pathology that might also require removal of a superficial parotid gland. Following superficial parotid gland removal, the remnant unencapsula ted acinar system in the glandular structure may also account for sialocele formation (a localised cavity or cyst containing saliva). Also after parotid gland surgery the acinar system with its ductules can be exposed to the wound, resulting in breakdown and leakage of saliva with fistula formation. The facial nerve gets enveloped within the substance of the gland as it grows laterally , dividing the parotid gland into Lucja Frey , 1896–1944, physician, The Neurological Clinic, Warsaw , Poland. Ludwig Levin Jacobson , 1783–1843, Danish anatomist. the superficial and deep lobes. Generally , the major functional component (80%) is superficial whereas the deep lobe is usu - ally the r etromandibular component with minimal functional gland tissue. The lymphatic system develops within the parotid glandu - - lar tissue after encapsulation of the submandibular and sub - lingual glands. This results in the majority of the lymphatic structures, including the lymph nodes, being embedded within the parotid. The retromandibular vein, which drains these lympha tics, generally lies deep to the facial nerve and is a con - stant landmark that is useful during the retrograde method of identification of the main trunk of the facial nerve. Most of the lymph nodes lie in the

superficial (preauricular) lobe of the parotid gland lateral to the masseter while very few lie in the deep (retromandibular) lobe of the parotid. - Summary box 54.2 - Parotid gland /uni25CF - /uni25CF /uni25CF Parotid innervation and Frey’s syndrome The glossopharyngeal nerve (cranial nerve IX) carries preganglionic parasympathetic fibres from the inferior salivatory nucleus. The Jacobson nerve, a branch of cranial

Parotid gland Sublingual ducts Sublingual gland Submandibular duct Submandibular gland Figure 54.1 Anatomical position of the three major salivary glands. Parotid duct Parotid gland Parotid gland and minor salivary glands are ectodermal in origin whereas the submandibular and sublingual glands are endodermal Parotid gland develops in the sixth week but has delayed encapsulation Facial vessels, facial nerve and lymphatic tissue are embedded in the substance of the parotid gland before the capsule fuses

the tympanic plexus in the middle ear. The lesser petrosal nerve carrying the preganglionic fibres from here exits via the foramen ovale, where it synapses with the postganglionic secretomotor parasympathetic fibres in the otic ganglion. These fibres exit the otic ganglion and join the auriculotemporal nerve in the infratemporal fossa, which innervates the parotid gland for the secretion of saliva. Within the gland, acetylcholine (ACh) stimulates both aci nar activity and ductal transport, leading to vasodilatation of the glands and contraction of the myoepithelial cells. Atro pine decreases salivation by competing with ACh for the sali vary r eceptor site and is useful in reducing salivary secretion. Regeneration of parasympathetic fibres to the sweat glands leads to abnormal autonomic reinnervation. ACh can act as a neurotransmitter for both postgang lionic sympathetic and parasympathetic fibres; this might contribute to ‘gustatory sweating’ (Frey syndrome), which involves sweating and flush ing of the skin overlying the parotid region while eating in some patients following parotidectomy . Parotid gland

Understanding a gland’s development gives us insight into the pathophysiology of the various disorders a ffl icting it. The parotid gland is ectodermal in origin and develops in the sixth week of gestation, when the epithelial buds invaginate from the oral mucosa into the surrounding mesenchyme. A tunnel develops from this groove and the gland is formed at its blind end by proliferation, budding and extensive branching. The secretory acini develop from the epithelial tissue, whereas the capsule of the gland and the connective tissue develop from the mesenchyme. The parotid, unlike other glands in the body , does not become encapsulated early to form a regular gland. The other structures in the vicinity , including the vessels, nerves and lymphatics, develop before encapsulation. The gland goes on to envelop the facial nerve, the terminal branches of the external carotid artery , the retromandibular and superficial temporal vein and the lymph nodes. The capsule thus merges with the investing fascia from the zygoma, over the temporomandibular joint and the masseter and reaches the styloid base, posterior digastric belly and the sternocleidomastoid muscle. The super ficial musculoaponeurotic system is closely approximated to this capsule. The parotid gland is, thus, irregular in shape and wedged in a recess between the ramus of the mandible, the base of the skull and the mastoid process . The parotid (Stensen’s) duct passes over the masseter muscle and enters the buccal mucosa through the buccinator muscle at the level of the upper second molar tooth. In some cases, an accessory gland is found along the course of the duct. Salivary tissue that is separated from the main parotid gland is referred to as an accessory parotid gland. These lie on the mas seter muscle in front of Stensen’s duct and have a secondary duct joining Stensen’s duct. According to autopsy studies, the incidence

of an accessory parotid gland is 21–61%. Embryo logically , the growing , budding salivary glands originate from the oral cavity epithelium outwards into the mesenchyme as it di ff erentiates into the various facial structures. The late completion of parotid gland encapsulation needs to be con sidered when planning surgery for accessory gland pathology that might also require removal of a superficial parotid gland. Following superficial parotid gland removal, the remnant unencapsula ted acinar system in the glandular structure may also account for sialocele formation (a localised cavity or cyst containing saliva). Also after parotid gland surgery the acinar system with its ductules can be exposed to the wound, resulting in breakdown and leakage of saliva with fistula formation. The facial nerve gets enveloped within the substance of the gland as it grows laterally , dividing the parotid gland into Lucja Frey , 1896–1944, physician, The Neurological Clinic, Warsaw , Poland. Ludwig Levin Jacobson , 1783–1843, Danish anatomist. the superficial and deep lobes. Generally , the major functional component (80%) is superficial whereas the deep lobe is usu - ally the r etromandibular component with minimal functional gland tissue. The lymphatic system develops within the parotid glandu - - lar tissue after encapsulation of the submandibular and sub - lingual glands. This results in the majority of the lymphatic structures, including the lymph nodes, being embedded within the parotid. The retromandibular vein, which drains these lympha tics, generally lies deep to the facial nerve and is a con - stant landmark that is useful during the retrograde method of identification of the main trunk of the facial nerve. Most of the lymph nodes lie in the superficial (preauricular) lobe of the parotid gland lateral to the masseter while very few lie in the deep (retromandibular) lobe of the parotid. - Summary box 54.2 - Parotid gland /uni25CF - /uni25CF /uni25CF Parotid innervation and Frey’s syndrome The glossopharyngeal nerve (cranial nerve IX) carries preganglionic parasympathetic fibres from the inferior salivatory nucleus. The Jacobson nerve, a branch of cranial

Parotid gland Sublingual ducts Sublingual gland Submandibular duct Submandibular gland Figure 54.1 Anatomical position of the three major salivary glands. Parotid duct Parotid gland Parotid gland and minor salivary glands are ectodermal in origin whereas the submandibular and sublingual glands are endodermal Parotid gland develops in the sixth week but has delayed encapsulation Facial vessels, facial nerve and lymphatic tissue are embedded in the substance of the parotid gland before the capsule fuses

the tympanic plexus in the middle ear. The lesser petrosal nerve carrying the preganglionic fibres from here exits via the foramen ovale, where it synapses with the postganglionic secretomotor parasympathetic fibres in the otic ganglion. These fibres exit the otic ganglion and join the auriculotemporal nerve in the infratemporal fossa, which innervates the parotid gland for the secretion of saliva. Within the gland, acetylcholine (ACh) stimulates both aci nar activity and ductal transport, leading to vasodilatation of the glands and contraction of the myoepithelial cells. Atro pine decreases salivation by competing with ACh for the sali vary r eceptor site and is useful in reducing salivary secretion. Regeneration of parasympathetic fibres to the sweat glands leads to abnormal autonomic reinnervation. ACh can act as a neurotransmitter for both postgang lionic sympathetic and parasympathetic fibres; this might contribute to ‘gustatory sweating’ (Frey syndrome), which involves sweating and flush ing of the skin overlying the parotid region while eating in some patients following parotidectomy .

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In parotidectomy , the tumour is removed with a cu ff of normal surrounding tissue where possible. The embryological develop - ment of the parotid with its late encapsulation not only embeds vessels, nerves and lymph nodes within the capsule but also fuses widely with the investing fascia from the temporalis above to the digastric below and from the buccinator anteriorly to the mastoid posteriorly . T he facial nerve traverses the parotid, making the removal of this gland di ffi cult. Types of parotid surgery as per the extent of resection (conservative to radical) /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Planning the surgery Decision making should take into consideration: 1 presumed tumour histology; 2 relation to the facial nerve plane (Patey’s faciovenous plane); 3 location of the tumour (deep lobe of the parotid); 4 facial nerve function. Traditionally , the type of parotidectomy is based on the location of the tumour either lateral to the plane of the facial nerve, necessitating a superficial parotidectomy , or deep to it, requiring a total parotidectomy . A total conservativ e paroti dectomy usually preserves the facial nerve whereas a radical parotidectomy involves its sacrifice. Today , with a focus towards reducing morbidity and pre serving gland function, varia tions of parotidectomy have evolved in the management of benign tumours: /uni25CF Extracapsular tumour dissection does not require a formal facial nerve dissection; this reduces the incidence of tem porary facial nerve palsy . /uni25CF Adequate parotidectomy involves removal of the tumour with a cu ff of normal tissue in tail of parotid lesions, pre serving function. It may not be possible to excise the tu mour with a cu ff of normal tissue in all the cases, especially when the lump is abutting the facial nerve. /uni25CF Deep lobe parotid tumours can be removed, preserving the entire superficial parotid gland, which is functional as well as cosmetic. Anaesthesia General anaesthesia is usually preferred unless there is any contraindication, in which case local anaesthesia may rarely be considered. Incision The planned incision should ensure complete access to the tumour, be extendable to encompass intraoperative surprises and be safe and cosmetically acceptable. The Blair incision is a straight preauricular incision curving slightly below the ear lobule. Bailey modified the inferior segment towards the mastoid and along the anterior border of the sternocleidomastoid. David Howard Patey , 1899–1976, surgeon, The Middlesex Hospital, London, UK. Vilray Papin Blair , 1871–1955, described the incision that bears his name in 1912. Henry Hamilton Bailey , 1894–1961, surgeon, The Royal Northern Hospital, London, UK. John J Conley , 1912–1999, otolaryngologist, St. Vincent’s Hospital and Medical Center, USA, made important contributions in the treatment of head and neck cancer. along the natural skin crease of the neck (Figure 54.12a) and has three components: (i) the horizontal part along the skin crease two finger breadths from the angle of the mandible, (ii) the vertical part close to the tragus in a skin crease if present, and (iii) the communicating part connecting the horizontal and vertical components in a gentle curve. The facelift incision has two components: the anterior pre - auricular component is similar to the modified Blair incision while the posterior limb curves at right angles, reaching the hair line and avoiding any neck incision. Flap elevation The horizontal component of the modified Blair incision is incised first to identify the platysma, the external jugular vein and the greater auricular nerve. The vertical component is then incised and connected inferiorly . The

platysma is divided and the subplatysmal flap is raised until the parotid gland is visualised and continued above, remaining below the superfi - cial musculoaponeurotic fascia, which connects the temporalis fascia and the platysma. This allows a suitable flap to be raised with the entire subcutaneous fat lifted o ff the parotid gland. The flap is developed anteriorly with elevation o ver the parotid - gland but not onto the masseteric fascia to prevent damage to the nerve branches as they exit the gland (Figure 54.12b). Parotid gland mobilisation - The fascia between the sternocleidomastoid and the parotid is dissected in the avascular plane. The greater auricular nerve is dissected up to the ear lobule and its anterior branches are divided (Figure 54.12c). The gland is then dissected o ff the - sternomastoid muscle and fibrofatty tissue overlying the inter - nal jugular vein to identify the posterior belly of the digastric muscle, which is then traced to the mastoid process. The e xter - - nal jugular vein is preserved as it provides a good landmark - for the facial nerve plane. The parotid is then mobilised from the tragal cartilage and the bony external auditor y meatus, exposing its tip – the ‘tragal pointer’. The two avascular planes are then connected by blunt and sharp dissection, identifying the tympanomastoid suture. Facial nerve localisation The facial nerve is usually identified by the antegrade tech - nique using anatomical landmarks, which are fairly constant, and is delineated from the trunk to the peripheral branches (Figure 54.12d): /uni25CF tragal (Conley’s) pointer: the facial nerve lies 1 /uni00A0 cm deep and inferior to the tip of the tragal cartilage; /uni25CF digastric muscle: the facial nerve can be identified above the upper border of the posterior belly of the digastric muscle; /uni25CF tympanomastoid suture: the facial nerve lies inferior to this suture line as it overlies the stylomastoid foramen.

Extracapsular dissection Adequate parotidectomy Super /f_i cial parotidectomy Total conservative parotidectomy Radical parotidectomy

Modi /f_i ed Blair incision Retroauricular incision External jugular vein (c) (d) Greater auricular nerve traced to ear lobule Mastoid tip (e) Patey’s fascia (f) Centripetal dissection Figure 54.12 (a) Incision planning showing the modi /f_i ed Blair incision and the retroauricular incision. the super /f_i cial musculoaponeurotic system layer, preserving the external jugular vein. dividing the branches going on to the parotid gland. (d) Tragal pointer and the digastric muscle used in combination to identify the facial nerve exiting the stylomastoid foramen in the centre. (e) Dissection of the nerve in Patey’s fascia: dissecting the nerves of the main trunk from the periphery to the centre. (f) Centripetal dissection of the parotid gland towards the parotid duct in the centre. Subplatysmal plane Incising the platysma Parotid tissue Tragal pointer Facial Digastric nerve muscle Cervicomandibular division (g) Ligating the parotid duct (b) Subplatysmal /f_l ap elevation to identify (c) Greater auricular nerve traced as far as the ear lobule, (g) Ligation of the parotid duct.

the parotid gland, fibrofatty tissue and the stylomastoid branch of the posterior auricular artery . Dissection of the glandular tissue and artery exposes the nerve. Bleeding can be brisk; this can be controlled with pressure, adrenaline (epinephrine) neuropatties and bipolar electrocautery . Identification of the facial trunk by a retrograde dissec tion technique is useful in revision cases with altered anatomy and fibrosis. It relies on the identification of one of the main branches of the nerv e (usually the buccal branch in relation to the parotid duct), which is then traced proximally to the main trunk. Centripetal dissection Once the main trunk is identified, a curved fine artery forceps is used to dissect in Patey’s plane above the nerve, taking care to avoid stretching the nerve (Figure 54.12e). The glandular tissue is dissected o ff the nerve and divided laterally .

Starting with the lower cervicomandibular division and its further divisions into the cervical, marginal mandibular and lower buccal branches, the entire course of each branch is identified until it exits the gland. Each of the branches is dissected in a sequential manner in a centripetal dissection working towards the parotid duct (Figure 54.12f). Thereafter, the upper temporozygomatic divi sion is traced onto its temporal, zygomatic and upper buccal branches, similarly reaching the duct. Thus at the end of the dissection the entire superficial part of the gland remains attached to the parotid duct, which can be clamped and lig to deliver the specimen (Figure 54.12g). Total conservative parotidectomy In a tumour straddling the superficial and deep lobes across Patey's plane, following removal of the superficial lobe, the deep lobe is dissected o ff the temporal veins and terminal branches of the external carotid artery . This results in complete removal of the suprafacial and subfacial parotid gland with preservation of the facial nerves – a total conservative parotidectomy . Radical parotidectomy In malignant tumours with extraparenchymal spread and facial nerve invasion, it is imperative to remove the involved struc tures. Radical parotidectomy involves removal of all parotid gland tissue and elective division of the involved facial nerve branches as well as the structures inv olved, most commonly the masseter muscle. It is imperative to repair the facial nerves when a segment has been removed. The branches supplying the orbicularis oculi and oris are prioritised in the reconstruc tion using cable grafts from either the greater auricular nerve or the sural nerve. Extracapsular dissection Extracapsular dissection for select benign parotid tumours is practised to avoid facial nerve dissection. It is reported to be as safe as parotidectomy . With a similar incision the subcutaneous flap is elevated above the platysma. At the site of the tumour Victor Minor , Russian neurologist, described the starch iodine test in 1928. expose the tumour. The tumour is dissected carefully in an extracapsular plane, visualising the facial nerve branches. Use of intraoperative nerve monitoring makes this safer. Howev er, in the absence of monitoring, careful dissection and a high level of suspicion before cutting any tissue is critical for uneventful - surgery . After excision the fascia is sutured with absorbable sutures, followed by skin closure. Drain placement and closure Sternomastoid muscle flaps or acellular dermal sheets may be used to cover the parotid bed. This attempts to prevent cross-innervation of the subcutaneous gland and overlying skin by the auriculotemporal nerve to avoid Frey's syndrome. A suction drain tube is placed and anchored beneath the posterior belly of the digastric muscle. The drain is brought out posterior to the suture line. The skin is sutured in layers. Complications Temporary facial palsy is commonly seen in the lower branches, especially the marginal mandibular nerve. Most patients tend to recover over time. When the zygomaticotemporal division is a ff ected, eye care is essential to protect the cornea. - Parotidectomy is a clean operation and hence infection is rare. Postoperative management of the drains following aseptic precautions and timely removal decreases the risk of infection. Haematoma is uncommon and is usually pre ventable ated with appropriate intraoperative haemostasis. Small haema - tomas usually resolve without intervention. Extreme collection causing discoloration suggests a possible bleeding diathesis that requir es appropriate management and evacuation of the haematoma. Sialocele is pre ventable by underrunning the capsule, where possible, to avoid exposure of the main acinar ductal system. Treatment includes the use of anticholinergics to reduce salivar y secretion, aspiration and pressure dressings. Longstanding salivary fistulae may require low-dose radiation. Hollowing of the retromandibular area can be reduced with autologous fat grafts, which can be placed at the time of primary surgery . - Frey's syndrome (gusta tory sweating) results from cross-innervation of the dermal sweat glands by the regener - ating postganglionic parasympathetic nerve fibres of the auriculotemporal nerve. It occurs in most patients but is often not significant; patients rarely mention it unless it is ex cessive.

Common presentations are sweating and flushing in the - preauricular region during meals. Minor’s starch iodine test identifies the region a ff ected. In it iodine is painted in the preauricular region, dried and covered with starch. Salivary stimulation causes sweating, which turns the starch blue. Frey’s syndrome can be reduced either by raising a thick flap with all subcutaneous fat over the parotid or by interposition of tissue, such as a sternocleidomastoid flap, temporalis fascia, allo - dermal tissue or autologous fat between the skin and the surgical bed. Its incidence can be reduced by extracapsular aponeurotic system. Treatment includes use of antiperspirants in mild cases; in more severe cases, tympanic neurectomy or botulinum toxin injection at the site of perspiration is used, which is ver y e ff ective. Summary box 54.8 Complications of parotid gland surgery /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Management of established Frey’s syndrome /uni25CF /uni25CF /uni25CF

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Major salivary gland malignancies are staged using the eighth edition of the American Joint Committee on Cancer (AJCC) staging. However, minor salivary gland malignancies are staged as per their site of origin. The primary tumour (T) staging is as given in Table 54.5 . The nodal (N) and distant metastasis (M) staging is similar to that for head and neck squamous cell carcinomas.
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The submandibular gland is surrounded by important struc tures and its removal is fairly straightforward if planned well. The approaches to the submandibular gland are transcutane ous (such as lateral transcervical, submental and retroauricular) or transoral. Visualisation during the various approaches can be impro ved with endoscopic or robotic assistance, potentially reducing complications. Summary box 54.6 Important anatomical relations to the submandibular gland /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Planning the surgery Decision making is determined by the nature of the disease. In benign lesions only the gland is removed, preserving stage and histology , there may be a consideration for a supra - omohyoid neck dissection involving levels I–III. Anaesthesia General anaesthesia is usually preferred unless there is any contraindication, in which case local anaesthesia may be considered. Incision The incision planned should ensure complete access to the tumour, be extendable to encompass intraoperative surprises and be safe and cosmetically acceptable. The standard incision is placed in a neck crease at least two finger breadths below the mandible. The horizontal incision is along the lines of relaxation, which results in a cosmetic scar (Figure 54.11a). Flap elevation After incising the skin the platysma is exposed and divided in the same plane. The skin is retracted with skin hooks and the flap is elevated in the avascular subplatysmal plane, taking care that the veins remain in the investing fascia. The flap is elevated up to the attachment of the platysma on the mandible, which helps in identification of the marginal mandibular nerve (Figure 54.11b). Marginal mandibular nerve preservation The fascia above the facial vessels is palpated in the midportion of the body of the mandible. The nerve is usually identified in the fascia above the vessels and is dissected along its path horizontally to expose it as it exits the parotid and traverses onto the muscles anteriorly . Gland mobilisation The facial vein and artery are ligated below the marginal nerve, exposing the lower edge of the body of the mandible. - The submandibular gland is now retracted downwards and outwards to expose the mylohyoid muscle (Figure 54.11c); the - fascia overlying the submandibular gland is dissected, exposing the submental artery and vein, which are secured and ligated. The lateral edge of the mylohyoid is delineated and retracted with a curved retractor. This exposes the deeper portion of the submandibular gland as well as Wharton’s duct. The submandibular gland is then retracted downwards to expose the lingual nerve (Figure 54.11d) and the connecting subman - dibular ganglion, which is divided. The gland is then retracted superiorly and posteriorly , exposing the deep fascia alongside the ranine veins above the hypoglossal nerve (Figure 54.11e). Wharton’s duct is then clamped, divided and liga ted close to the floor of the mouth to prevent retention of

debris or stones, reducing the chance of infection. The gland is retracted downwards and laterally , exposing the facial artery above the common tendon of the digastric muscle. The facial artery is clamped, divided and ligated, completing the gland excision. Malignant submandibular tumours Treatment for low-grade tumours that are less than 4 /uni00A0 cm with - out extraparenchymal spread is submandibular gland removal with clearance of the perivascular group of lymph nodes around

Lingual nerve Hypoglossal nerve Marginal mandibular branch of the facial nerve Anterior facial vein Facial artery

(b) Skin crease incision Subplatysmal /f_l ap (c) Mylohyoid muscle retracted Mylohyoid muscle SM gland retracted down and laterally Figure 54.11 (a) Submandibular (SM) pleomorphic adenoma: clinical presentation and T1-weighted magnetic resonance imaging with contrast view showing the tumour. (b) A horizontal skin crease incision, subplatysmal /f_l ap elevation and identi /f_i cation of the marginal mandibular nerve. (c) The gland is retracted downwards and outwards to identify and retract the mylohyoid muscle to locate the lingual nerve. Marginal nerve Lingual nerve and SM ganglion Deep part of SM gland

the facial vein. In high-grade tumours, supraomohyoid neck dissection (levels I–III) is recommended, whereas node-positive tumours require a comprehensive neck dissection (levels I–V). For tumours with extraparenchymal spread, radical clearance of the involved structures along with a formal comprehensive neck dissection is required. Closure Haemostasis is achieved and an antiseptic wash is given. Intraoral communication is ruled out before placing a suction drain over the mylohyoid with the tip pointing laterally to prevent migration into the oral cavity . The wound is closed by approximating the platysma with absorbable sutures and the skin with absorbable or non-absorbable sutures. Complications Nerve palsy The proximity of the three nerves (marginal division of the facial, lingual and hypoglossal) to the submandibular gland makes them susceptible to injury . Care should be taken, espe - cially in recurrent sialadenitis, which causes dense adhesions, increasing the possibility of nerve injury . Haemorrhage The facial artery and the ranine veins along the hypoglossal are notorious for postoperative haemorrhage and should be double-checked prior to closure.

Hypoglossal nerve (e) SM fossa Figure 54.11 (d) The gland is retracted downwards and outwards to identify the hypoglossal nerve and the facial vein and artery before removing the gland. (e) The submandibular fossa after tumour-laden gland removal and closure with subcuticular sutures. SM gland retracted up Facial vein above the Facial artery exposed on digastric retracting SM gland

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Sialadenitis

Sialadenitis

Inflammation of a salivary gland can be acute or chronic (Table 54.2). Acute causes include viral and bacterial infec - tion. It mainly a ff ects the young adolescent population. Parotid glands are more commonly involved than submandibular glands. /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF Viral infections are more common than bacterial infections. The causative organisms are most commonly paramyxovirus (mumps), followed by cytomegalovirus, coxsackie virus, human immunodeficiency virus (HIV), parainfluenza virus types I and II, influenza virus A and herpesvirus. Staphylococcus aureus is the most common Among bacteria, organism and usually results in a retrograde spread of infec tion through the duct. Duct obstruction and xerostomia in the elderly population are predisposing factors. The patient will present with a painful red swelling over the gland region. Viral sialadenitis is self-limiting in most cases , requiring symptomatic supportive care, while bacterial infection will require antibiotics. Human immunodeficiency virus sialadenitis This is mainly characterised by bilateral enlargement of the parotid glands. This may mimic SjS. HIV sialadenitis is usually seen in young individuals with an absence of any serological antibodies, whereas SjS is mainly seen in middle-aged women. The prevalence is between 5% and 10% and it has been postulated that it is more common in women on highly active antiretroviral therapy (HAART) (mainly protease inhibitors). HAART or parenchymal disease of the salivary gland as a result of HIV (Figure 54.5). Biopsy reveals perivascular, periductal and periacinar areas predominantly infiltrated with CD8 cells. Abnormal deposi - tion of fat seen in the parotid gland (parotid lipomatosis) as well as in the abdomen and dorsal cervical areas may be associated with the use of protease inhibitors. Benign lymphoepithelial cysts are HIV-related reactive lymphoproliferation, which may occur in the intraparotid lymph nodes (Table 54.1). The parotid g landular epithelium may get trapped within the normal intraparotid lymph nodes, resulting in cystic enlargement or migration of HIV-infected cells into the parotid gland, which could trigger lymphoid proliferation, salivary duct dysplasia, ductal obstruction and cyst formation. Ultrasonography is usually diagnostic. There may be a rare conversion into lymphoma. Di ff use infiltrative lymphocytosis syndrome manifests with bilateral enlargement caused by constant infiltration of CD8 in the parotid glands. Summary box 54.4 - HIV sialadenitis /uni25CF /uni25CF /uni25CF /uni25CF Recurrent parotitis of childhood This is characterised by rapid swelling of one or both parotid glands that is aggravated by chewing and eating. Systemic upset with fever and malaise is variable. The symptoms usually last about a week and are followed by a quiescent period for weeks to several months. It is mainly seen between the ages of 3 and 6 years. It is postulated to be caused by an incompetent parotid duct punctum, leading to ductal contamination with oral fluids. The diagnosis is based on the characteristic history . In addition, sialography shows a characteristic punctate sialectasis (snowstorm). The condition is di ffi cult to manage if it becomes established and the initial treatment is important. The treatment consists of long courses of antibiotics and endoscopic washouts. Sialadenosis This is a non-inflammatory bilateral enlargement of the parotid glands. The swelling is generally painless with reduced saliva and is associated with chronic

malnutrition, obesity , alcoholism, liver disease, diabetes and drugs such as guaneth - idine, thioridazine or isoprenaline. It requires di ff erentiation from any neoplastic disorder. Treatment mainly consists of management of the underlying systemic disorder.

sialadenitis. Acute sialadenitis Chronic sialadenitis Viral: Granulomatous: Mumps TB Coxsackie Cat scratch disease Cytomegalovirus Actinomycosis Paramyxovirus Sarcoidosis Bacterial: HIV Staphylococcus aureus Abscess (parotid and (acute suppurative submandibular) parotitis) Recurrent subacute parotitis Radiation sialadenitis HIV, human immunode /f_i ciency virus; TB, tuberculosis. Figure 54.5 Lymphoepithelial cysts in a human immunode /f_i ciency virus-infected patient. Incidence 5–10% More common in women on HAART Mimic SjS, but absent antibody In /f_i ltration of CD8 in the parotid glands

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Sialolithiasis

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Salivary gland stones can form in the gland ducts. Patients between the ages of 30 and 60 with sialolithiasis typically pres - ent with cyclical postprandial swelling of the major salivary glands and dryness possibly due to decreased salivary flow . Duct abnormality , inflammation and increased calcium content of the saliva may contribute to an increased risk of sialolithiasis. Chronic dehydration and pharmacological causes of decreased salivary flow are often implicated. The submandibular gland is most commonly a ff ected (85%) owing to the ascending course of its duct, predisposing it to stagnation of the mucinous as well as the more viscous saliva it produces (Figure 54.6). The alka line saliva precipitates calcium and phosphate and predisposes to stone formation. On examination, there is an asymmetrical enlargement of the gland and a large proximal stone may be palpated in certain cases. Submandibular gland duct stones ar e mainly proximal and parotid gland duct stones are mainly distal (Figure 54.7). Sialolithiasis complicated by a secondary bacterial infection may present with an abscess. Conventional radiographs are considered as an initial diagnostic test (Figure 54.8). However, small stones may be - missed and only 80% of stones are radio-opaque. In such cases, computed tomography (CT) scanning, ultrasonography and a magnetic resonance sialogram will be more sensitive for diagnosis and localisation. Sialography is the gold standard for diagnosis and involves injecting a dye into the duct of the salivary gland. It not only helps in diagnosis of sialolithiasis but also identifies any pathology in the duct. In addition, it may be therapeutic in certain cases. Sialendoscopy provides

(b) Figure 54.6 (a) A submandibular gland sialolithiasis. (b) endoscopy with submandibular gland calculus removal (courtesy of Dr /uni00A0 Shirish Ghan, Nasik, India). (b) Figure 54.7 (a) Parotid

gland swelling due to lithiasis and secondary stricture. (b) Sialendoscopic stricture dilatation (courtesy of Dr Shirish Ghan, Nasik, India). Sialo

Figure 54.8 Occlusal view showing a stone in the submandibular gland.

of stones. It is a safe procedure that can be performed under local anaesthesia with better outcomes than open surgery . The smaller (<5 /uni00A0 mm) distal stones can be removed with endoscopy while the larger (>5 /uni00A0 mm) distal stones may require duct slit ting. For an impacted stone, the transoral route is used. Intra parenchymal stones between 5 and 7 /uni00A0 mm can be extracted endoscopically while larger stones require transoral slitting. Stones that are not palpable and not visualised endoscop ically can be remo ved using external shock wave lithotripsy (ESWL). However, ESWL is not suitable for stones larger than 7–10 /uni00A0 mm. Hilar stones are removed using an endoscope. Excision of the submandibular gland should be considered as a last resort. Parotid stones (<7 /uni00A0 mm) can be removed endo scopically and di ffi cult cases might need a combined trans cutaneous approach. ESWL can be considered for impacted stones. Again gland removal should be considered only as a final r emedy . Sialolithiasis

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Sialorrhoea

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Sublingual gland

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The sublingual glands contribute around 5% of saliva produc - tion and are the smallest of the major salivary glands. They - lie above the mylohyoid muscle and below the floor of mouth mucosa and are bordered by the mandible laterally and by - the genioglossus m uscle medially . Their secretions are drained - by small ducts (Rivinus’s ducts) that exit along the sublingual fold at the floor of the mouth. A few anterior ducts may join together to form a common duct called Bartholin’s duct, which empties close to or into Wharton’s duct near the sublingual caruncle. The pathology of the sublingual glands mainly involves the - formation of a mucous retention cyst (ranula). Tumours of sublingual glands are v ery rare. Summary box 54.3 Sublingual gland /uni25CF /uni25CF /uni25CF /uni25CF - -

Problems are rare Minor mucous retention cysts may need surgery A plunging ranula is a retention cyst that tunnels deep Nearly all tumours are malignant

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The submandibular glands originate from the junctional tissue between ectoderm and endoderm from the floor of the mouth. They grow from the 18th to the 25th embryonic week and acquire connective capsules. They lie in the submandibular space between the digastric muscles and extend upwards deep to the mandible. They con sist of a larger superficial and a smaller deep lobe that is con tinuous around the posterior bor der of the mylohyoid muscle. The deep part of the gland lies on the hyoglossus muscle in close relation to the lingual nerve. The submandibular gan glion innervates the submandibular gland by the postganglionic parasympathetic fibres from the superior salivatory nucleus of the pons, through the chorda tympani and lingual nerve. The ganglion connections are r equired to be separated to free the gland and preserve the lingual nerve during excision. The gland is surrounded by a well-defined capsule derived from the deep cervical fascia, which splits to enclose it. Whar ton’s (submandibular) duct lies between the hyoglossus and mylohyoid muscle after arising from the deep part of the gland. It drains at the sublingual papilla into the anterior floor of the mouth. The facial vein lies superficial to the g land to reach the anterior border of the mandible. The facial artery enters deep to the posterior belly of the digastric and stylohyoid mus cles and passes through or superficial to the gland to reach the anterior border of the mandible. The glandular branches need to be ligated when preserving the facial artery during subman dibular gland removal. The facial artery is commonly used as the recipient artery in microvascular anastomosis in free tis sue transfer in head and neck reconstruction. The marginal mandibular branch of the facial nerve lies in the superficial Hayes Martin , 1892–1977 Attending Surgeon, Memorial Hospital; Chief, Head and Neck Surgery , Memorial Sloan Kettering Cancer Centre; Professor of Sur gery , Cornell University Medical College, New Y ork, NY , USA. Augustus Quirinus Rivinus , 1652–1723, German physician and botanist, studied the anatomy of salivary glands and also developed ways of classifying plants. T homas Bartholin , 1616–1680, Professor of Anatomy , Copenhagen University , Copenhagen, Denmark. technique of ligating these vessels and flipping them abov e in order to preserve the mandibular division of the facial nerve during neck dissection. Submandibular gland

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TREATMENT OF SALIVARY GLAND MALIGNANCIES

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The treatment guidelines are based on retrospective studies, with very little randomised evidence to guide treatment deci - sions. Surgery forms the mainstay of treatment with a goal to extirpate the tumour with microscopic margins of at least 0.5 /uni00A0 cm. The extent of resection is determined not only by the size and stage of the malignancy but also by the g rade of di ff erentiation. The preservation of the facial nerve should - be planned, but not at the cost of residual disease. Elective neck dissection should be o ff ered for T3/T4 and high-grade tumours. In node-positive disease, comprehensive neck dissection is mandatory . Adjuvant radiotherapy is advocated for stage III and IV tumours, high grade of di ff erentiation as well as the presence of high-risk features such as close/positive surgical margins, the presence of perineural or lymphovascular invasion and nodal metastases with extranodal extension. The role of chemoradiation in the adjuvant setting is still under investigation. In unresectable or metastatic tumours, palliative chemotherapy and/or targeted therapy is being explored.

the American Joint Committee on Cancer system. T category T criteria TX Primary tumour cannot be assessed T0 No evidence of primary tumour Tis Carcinoma in situ T1 Tumour 2 /uni00A0 cm or smaller in greatest dimension a without extraparenchymal extension T2 Tumour larger than 2 /uni00A0 cm but not larger than 4 /uni00A0 cm in greatest dimension without extraparenchymal a extension T3 Tumour larger than 4 /uni00A0 cm and/or tumour having a extraparenchymal extension T4a Moderately advanced disease Tumour invades skin, mandible, ear canal and/or facial nerve T4b Very advanced disease Tumour invades skull base and/or pterygoid plates and/or encases carotid artery a Extraparenchymal extension is clinical or macroscopic evidence of invasion of soft tissues. Microscopic evidence alone does not consti tute extraparenchymal extension for classi /f_i cation purposes.

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Trauma

Trauma

Trauma to the salivary glands or ducts is uncommon and is usually associated with polytrauma due to penetrating injuries, blasts or vehicular accidents. This can result in injury to the salivary glandular tissue, the duct and the surrounding nerves, Aldred Scott Warthin , 1866–1931, Professor of Pathology , University of Michigan, Ann Arbor, MI, USA. go unnoticed and manifest later with a salivary fistula, nerve palsy and/or sialoceles. The basic principles of all wound care management apply - to salivary gland injuries, including removal of foreign bod - - ies, wound washout, debridement and tension-free closure of the wound. Besides this, the g landular tissue, the duct and the nerves in the vicinity require utmost attention to prev ent late - complications. These are often overlooked and missed. Facial nerve A cranial nerve examination guides the clinician towards the possibility of nerve injuries. The wound is examined for nerve - injuries. If the branches are severed anteriorly to an imaginary - line dropped vertically down from the lateral canthus of the eye, they are not repaired. If the injury is posterior to this line, a direct nerve repair or grafting is carried out depending on the wound status. If not feasible at that time the nerve endings are tagged for later. Salivary duct Within the first 72 hours a duct injury should be repaired with - a direct end-to-end anastomosis over a cannula. If there is a loss of duct tissue of over 1 /uni00A0 cm, the proximal portion is either cannulated for subsequent marsupialisation into the oral cavity or a duct rerouting is considered. In cases with significant parenchyma and duct injury a ductal ligation along with gland excision should be considered. Trauma

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Xerostomia

Xerostomia

Normal salivary flow decreases with age. Typical complaints are of a dry mouth, di ffi culty swallowing and speaking, intol erance to spicy , acidic and crunchy food, a loss of taste and denture-wearing issues. Xerostomia is more common among postmenopausal women, who complain of a burning tongue or mouth. Common causes of xerostomia are chronic anxiety states and de pression; dehydration; anticholinergic drugs, especially antidepressants; salivary gland disorders (e.g. SjS); and radiotherapy to the head and neck region. Xerostomia and salivary gland hypofunction diagnosis requires a thorough history and examination as well as documentation with one of the several questionnaires available. Treatments include proper hydration, humidification at night, avoiding harmful dentifrices and crunchy/hard foods and the use of sugar-free chewing gums and lozenges. Medications include lubricants, saliva substitutes or stimulants. Xerostomia

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