

CHAPTER

Diagnosis and Management of Salivary Gland Disorders

Michael Miloro *and* Sterling R. Schow

CHAPTER OUTLINE

EMBRYOLOGY, ANATOMY, AND PHYSIOLOGY
DIAGNOSTIC MODALITIES
History and Clinical Examination Salivary Gland
Radiology Plain Film Radiographs Sialography
Computed Tomography, Magnetic Resonance Imaging, and Ultrasound
Salivary Scintigraphy (Radioactive Isotope Scanning)
Salivary Gland Endoscopy (Sialoendoscopy)
Sialochernistry
Fine-Needle Aspiration Biopsy
Salivary Gland Biopsy
OBSTRUCTIVE SALIVARY GLAND DISEASE
Sialolithiasis
MUCOUS RETENTION AND EXTRAVASATION PHENOMENA
Mucocele
Ranula
SALIVARY GLAND INFECTIONS
NECROTIZING SIALOMETAPLASIA SJOGREN'S SYNDROME
TRAUMATIC SALIVARY GLAND INJURIES
NEOPLASTIC SALIVARY GLAND DISORDERS
Benign Salivary Gland Tumors
Malignant Salivary Gland Tumors

The clinician is frequently confronted with the necessity of assessing and managing salivary gland disorders. A thorough knowledge of the embryology, anatomy, and pathophysiology is necessary to treat patients appropriately. This chapter examines the cause, diagnostic methodology, radiographic evaluation, and management of a variety of salivary gland disorders, including sialolithiasis and obstructive phenomena (e.g., mucocele and ranula), acute and chronic salivary gland infections, traumatic salivary gland disorders, S]6gren's syndrome (SS), necrotizing sialometaplasia, and benign and malignant salivary gland tumors.

EMBRYOLOGY, ANATOMY, AND PHYSIOLOG

The salivary glands can be divided into two groups: the minor and major glands. All salivary glands develop from the embryonic oral cavity as buds of epithelium that extend into the underlying mesenchymal tissues. The epithelial ingrowths branch to form a primitive ductal system that eventually becomes canalized to provide for drainage of salivary secretions. The minor salivary glands begin to develop around the fortieth day in utero, where- as the larger major glands begin to develop slightly earlier, at about the thirty-fifth day in utero. At around the seventh or eighth month in utero, secretory cells called

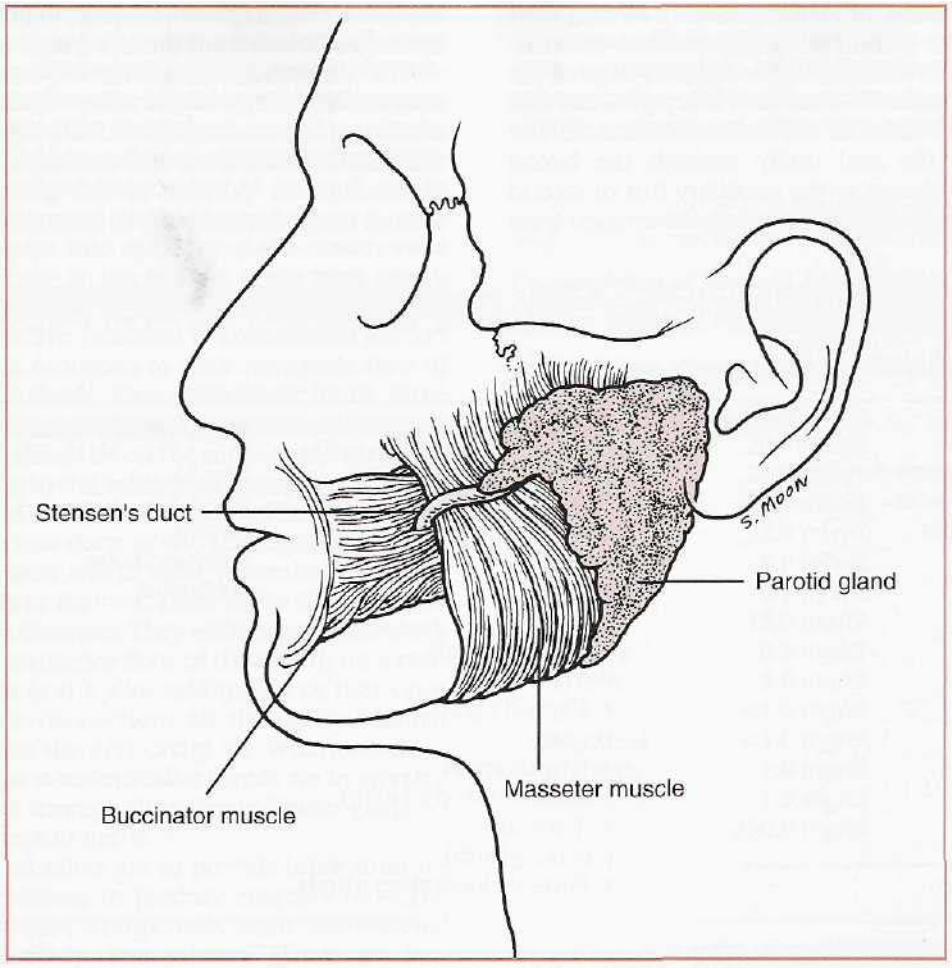


FIG. 20-1 Parotid gland anatomy. The course of Stensen's duct runs superficial to the masseter muscle and then curves sharply anteriorly to pierce the buccinator muscle fibers and enter the oral cavity.

acini begin to develop around the ductal system. The acinar cells of the salivary glands are classified as either *serous cells,* which produce a thin, watery serous secretion, or *mucous cells,* which produce a thicker, viscous mucous secretion. The minor salivary glands are well developed and functional in the newborn infant. The acini of the minor salivary glands primarily produce mucous secretions, although some are made up of serous cells, as well. The major salivary glands are paired structures and are the parotid, submandibular, and sublingual glands. The parotid glands contain primarily serous acini with few mucous cells. Conversely, the sublingual glands are for the most part composed of mucous cells. The submandibular glands are mixed glands, made up of approximately equal numbers of serous and mucous acini. Between 800 and 1000 minor salivary glands are found throughout the portions of the oral cavity that are covered by mucous membranes, with a few exceptions, such as the anterior third of the hard palate, the attached gingiva, and the dorsal surface of the anterior third of the tongue. The minor salivary glands are referred to as the *labial, buccal, palatine, tonsillar* (Weber's glands), *retromolar* (Carmalt's glands), and *lingual glands,* which are divided into three groups: (1) inferior apical (glands of Blandin Nuhn), (2) taste buds (Ebner's glands), and (3) posterior lubricating glands (Table 20-1).

The parotid glands, the largest salivary glands, lie superficial to the posterior aspect of the masseter muscle and the ascending ramus of the mandible. Peripheral portions of the parotid gland extend to the mastoid process, along the anterior aspect of the sternocleidomastoid muscle, and around the posterior border of the mandible into the pterygomandibular space (Fig. 20-1). The major branches of the seventh cranial (facial) nerve roughly divide the parotid gland into a superficial lobe and a deep lobe while coursing anteriorly from their exit at the stylomastoid foramen to innervate the muscles of facial expression. Small ducts from various regions of the gland coalesce at the anterosuperior aspect of the parotid to form Stensen's duct, which is the major duct of the parotid gland. Stensen's duct is about 1 to 3 mm in diameter and 6 cm in length.

Occasionally, a normal anatomic variation occurs in which an accessory parotid duct may aid Stensen's duct in drainage of salivary secretions. Additionally, an accessory portion of the parotid gland may be present some-

where along the course of Stensen's duct. The duct runs anteriorly from the gland and is superficial to the masseter muscle. At the location of the anterior edge of the masseter muscle, Stensen's duct turns sharply medial and passes through the fibers of the buccinator muscle. The duct opens into the oral cavity through the buccal mucosa, usually adjacent to the maxillary first or second molar tooth. The parotid gland receives innervation from the ninth cranial (glossopharyngeal) nerve via the auriculotemporal nerve from the otic ganglion.

The submandibular glands are located in the submandibular triangle of the neck, which is formed by the anterior and posterior bellies of the digastric muscles anc the inferior border of the mandible (Fig. 20-2). The posterosuperior portion of the gland curves upward around the posterior border of the mylohyoid muscle and

TABLE 20-1

Salivary Gland Embryology and Anatomy

	Minor Salivary Glands	Major Salivary Glands
In utera development	Day 40	Day 35
Number	800-1000	6
Types	Labial	Parotid
	Buccal	Submandibular
	Palatine	Sublingual
	Tonsillar	
	• Weber's glands	
	Retromolar	
	• Carmalt's glands	
	Lingual	
	• Inferior apical	
	(Glands of Blandin Nuhn)	
	• Taste buds	
	(Ebner's glands)	
	• Posterior lubricating glands	

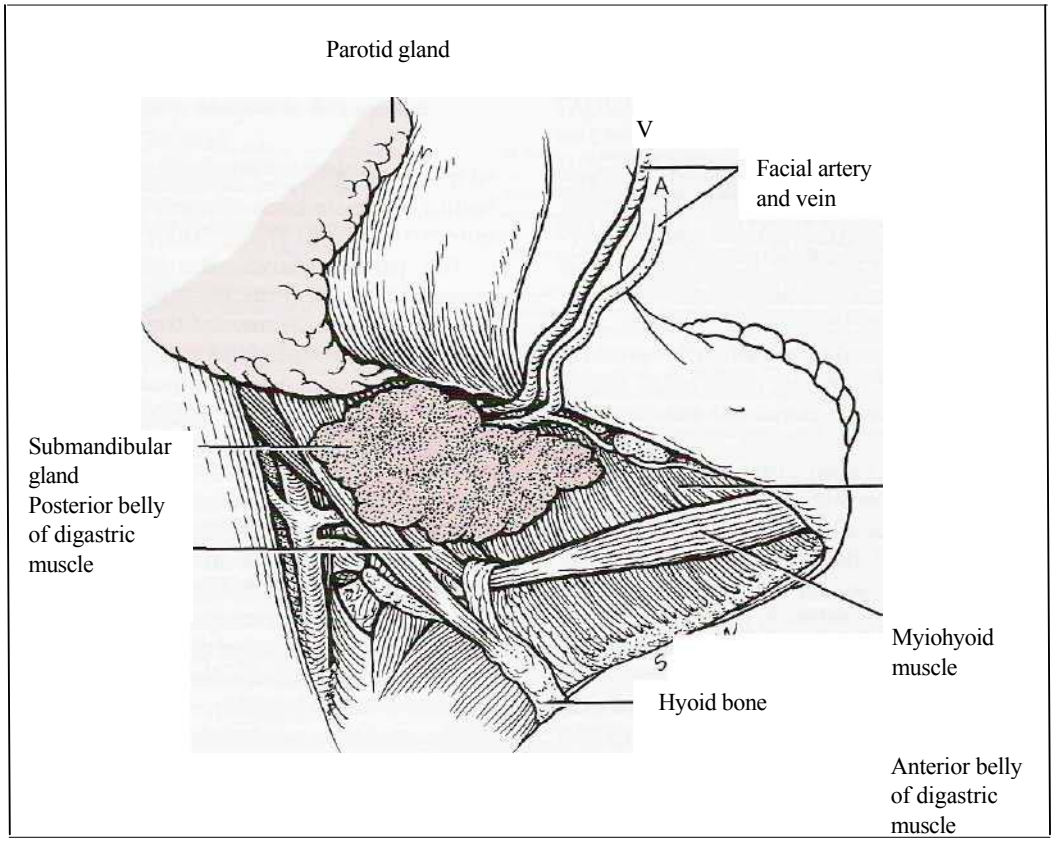


FIG. 20-2 Submandibular gland anatomy. The anterior and posterior bellies of the digastric muscles and the inferior border of the mandible form the submandibular triangle.

gives rise to the major duct of the submandibular gland known as *Whartoris duct.* This duct passes forward along the superior surface of the mylohyoid muscle in the sublingual space, adjacent to the lingual nerve. The anatomic relationship is such that the lingual nerve loops under Wharton's duct, from lateral to medial, in the posterior floor of the mouth. Wharton's duct is about 5 cm in length, and the diameter of its lumen is 2 to 4 mm. Wharton's duct opens into the floor of the mouth via a punctum located close to the incisors at the most anterior aspect of the junction of the lingual frenum and the floor of the mouth. The punctum is a constricted portion of the duct, and it functions to limit retrograde flow of bacteria-laden oral fluids. This particularly limits those bacteria that tend to colonize around the ductal orifices.

The sublingual glands lie on the superior surface of the mylohyoid muscle, in the sublingual space, and are separated from the oral cavity by a thin layer of oral mucosa (Fig. 20-3). The acinar ducts of the sublingual glands are called *Bartholin's ducts* and in most instances coalesce to form 8 to 20 ducts of Rivinus. These ducts of Rivinus are short and small in diameter. They either open individually directly into the anterior floor of the mouth on a crest of mucosa, known as the *plica sublingualis,* or they open indirectly through connections to the submandibular duct and then into the oral cavity via Wharton's duct. The sublingual and submandibular glands are innervated by the facial nerve through the submandibular ganglion via the chorda tympani nerve.

The functions of saliva are to provide lubrication for speech and mastication, to produce enzymes for digestion, and to produce compounds with antibacterial properties (Table 20-2). The salivary glands produce approximately 1000 to 1500 ml of saliva per day, with the highest flow rates occurring during meals. The relative contributions of each salivary gland to total daily production varies, with the submandibular gland providing 70%, the parotid gland 25%, the sublingual gland 3% to 4%, and the minor salivary glands contributing only trace amounts of saliva (Box 20-1). The electrolyte composition of saliva also varies between salivary glands, with parotid gland concentrations generally higher than the submandibular gland, except for submandibular calcium concentration, which is approximately twice the concentration of parotid calcium (see Table 20-2). The relative viscosities of saliva vary according to gland and

TABLE 20-2

Composition of Normal Adult Saliva

	Parotid Gfand	Submandibular Gland
Sodium	23.0 mEq/L	21.0 mEq/L
Potassium	20.0 mEq/L	17.0 mEq/L
Chloride	23.0 mEq/L	20.0 mEq/L
Bicarbonate	20.0 mEq/L	18.0 mEq/L
Calcium	2.0 mEq/L	3.6 mEq/L
Phosphate	6.0 mEq/L	4.5 mEq/L
Magnesium	0.2 mEq/L	0.3 mEq/L
Urea	15.0 mg/dl	7.0 mg/dl
Ammonia	0.3 mg/dl	0.2 mg/dl
Uric acid	3.0 mg/dl	2.0 mg/dl
Glucose	<1.0 mg/dl	<1.0 mg/dl
Cholesterol	< 1.0 mg/dl	<1.0 mg/dl
Fatty acids	1.0 mg/dl	<1.0 mg/dl
Amino acids	1.5 mg/dl	<1.0 mg/dl
Proteins	250.0 mg/dl	150.0 mg/dl

BOX 20-1

Daily Saliva Production by Salivary Gland

Submandibular gland	70%
Parotid gland	25%
Sublingual gland	3%-4%
Minor glands	Trace

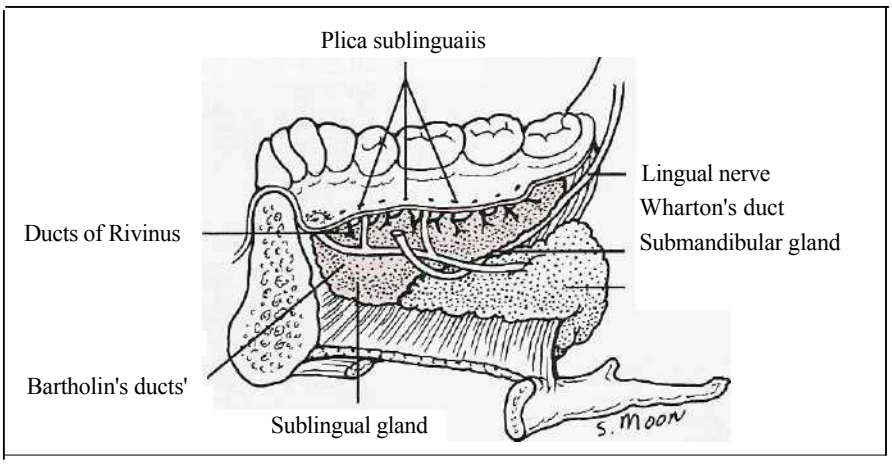


FIG. 20-3 Sublingual gland anatomy. The interrelationships between the ductal systems of the submandibular and the sublingual glands and the relationship of the lingual nerve to Wharton's duct are demonstrated.

BOX 20-2

Incidence of Radiopaque Stones

Submandibular gland	80%
Parotid gland	40%

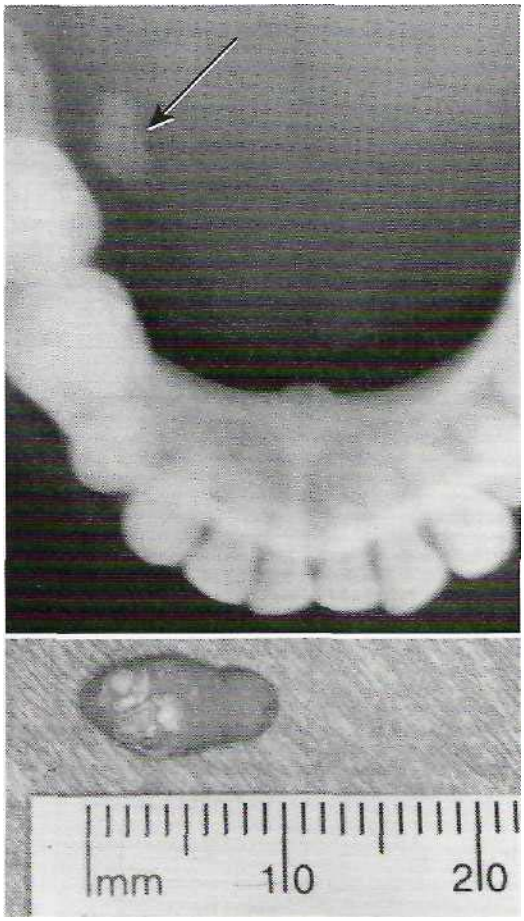


FIG. 20-4 A, Mandibular ocdusal radiograph shows a radiopaque sialoiith *(arrow)*. B, Submandibular sialoiith after intraoral removal is demonstrated.

correspond to the percentage of mucous and serous cell; therefore the highest viscosity is in the sublingual gland, followed by the submandibular gland, and, lastly. I parotid gland, which is composed mainly of serous eel Interestingly, the daily production of saliva begins decrease gradually after the age of 20.

DIAGNOSTIC MODALITIES

History and Clinical Examination

The most important component of diagnosis in salivary gland disorders, as with most other disease processes, is the patient history and the clinical examination. In most cases the patient will guide the doctor to the diagnosis merely by relating the events that have occurred in association with the presenting complaint. The astute clinician must perform a thorough evaluation, and, in mar instances, the diagnosis can be determined without the necessity of further diagnostic evaluation. At the very least, the clinician may be able to categorize the problem as reactive, obstructive, inflammatory, infectious, metabolic, neoplastic, developmental, or traumatic in origin and guide further diagnostic testing. Occasionally, the clinician may find it necessary to use any of several diagnostic modalities.

Salivary Gland Radiology

Plain film radiographs. The primary purpose of plain films in the assessment of salivary gland disease is to identify salivary stones (calculi), although only 80% to 85% of all stones are radiopaque and therefore visible radiographically. The incidence of radiopaque stones varies, depending on the specific gland involved (Box 20-2). A mandibular occlusal film is most useful for detecting sublingual and submandibular gland calculi in the anterior floor of the mouth (Fig. 20-4, *A)*. A "puffed cheek view," in which the patient forcibly blows the cheek laterally to distend the soft tissues overlying the lateral ramus, can demonstrate parotid stones. Panoramic radiographs can reveal stones in the parotid gland a posteriorly located submandibular stones (Fig. 20-5).

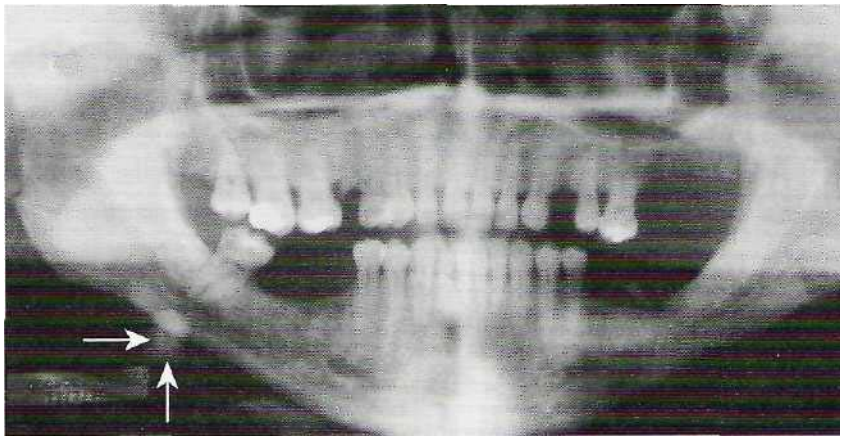


FIG. 20-5 Panoramic radiograph demonstrates a right submandibular sialoiith farrows).

Periapical radiographs can show calculi in each salivary gland or duct, including minor salivary glands, depending on film placement. In most instances, the radiographic image corresponds in size and shape to the actual stone (see Fig. 20-4, *B).*

Siatography. The gold standard in diagnostic salivary gland radiology may be the sialogram. Sialography is indicated as an aid in the detection of radiopaque stones. In addition, when 15% to 20% of stones are radiolucent; sialography is also useful in the assessment of the extent of destruction of the salivary duct or gland or both as a result of obstructive, inflammatory, traumatic, and neoplastic diseases. In addition to its diagnostic role, sialography may be used as a therapeutic maneuver, because the ductal system is dilated during the study, and small mucous plugs or necrotic debris may be cleared during injection of contrast.

Sialography is a technique in which the salivary duct is cannulated with a plastic or metal catheter (Fig. 20-6), a radiographic contrast medium is injected into the ductal system and the substance of the gland, and a series of radiographs are obtained during this process. Approximately 0.5 to 1 ml of contrast material can be injected into the duct and gland before the patient begins to experience pain. The two types of contrast media available for sialographic studies are water-soluble and oil-based. Both types of contrast material contain relatively high concentrations (25% to 40%) of iodine . Most clinicians prefer to use water-soluble media, which are more mistible with salivary secretions, more easily injected into the finer portions of the ductal system, and more readily eliminated from the gland after the study is completed, either by drainage through the duct or systemic absorption from the gland and excretion through the kidneys. The oil-based media are more viscous and require a higher injection pressure to visualize the finer ductules than do the water-soluble media. As a result, they usually produce more discomfort to the patient during injection. Oil-based media are poorly eliminated from the ductal system and may cause iatrogenic ductal obstruction.

Residual oil-based contrast medium is not absorbed by the gland and may produce severe foreign-body reactions and glandular necrosis. Additionally, if the patient has ductal disruption secondary to chronic inflammatory changes, the extravasation of oil-based media may cause significantly more soft tissue damage than water-soluble material.

A complete sialogram consists of three distinct phases, depending on the time at which the radiograph is obtained after injection of the contrast material:

1. Ductal phase (Fig. 20-7), which occurs almost immediately after injection of contrast material and allows visualization of the major ducts
2. Acinar phase (Fig. 20-8), which begins after the ductal system has become fully opacified with con trast and the gland parenchyma becomes filled subsequently
3. Evacuation phase, which assesses normal secretory clearance function of the gland to determine whether any evidence of retention of contrast remains in the gland or ductal system after the sialogram

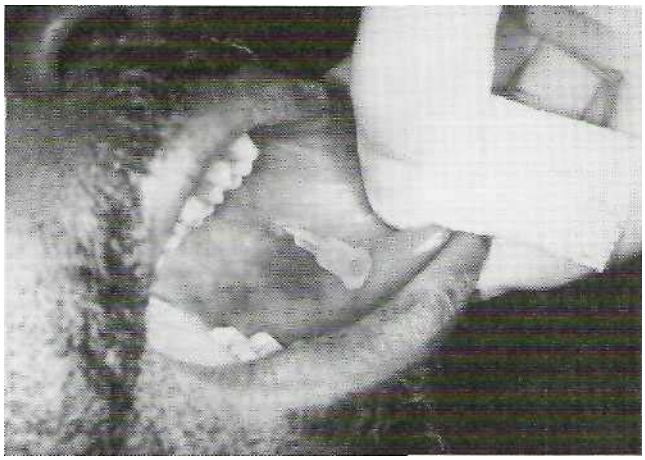


FIG. 20-6 Cannulation of Stensen's duct with a plastic catheter.

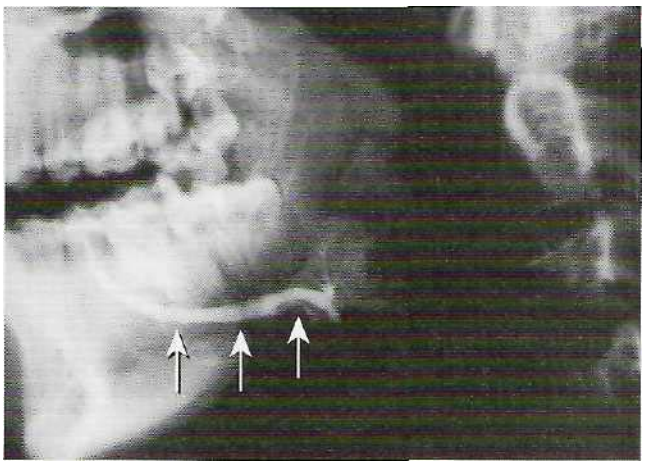


FIG. 20-7 Ductal phase of a submandibular sialogram. Contrast is contained only within the main salivary ducts *(arrows).*

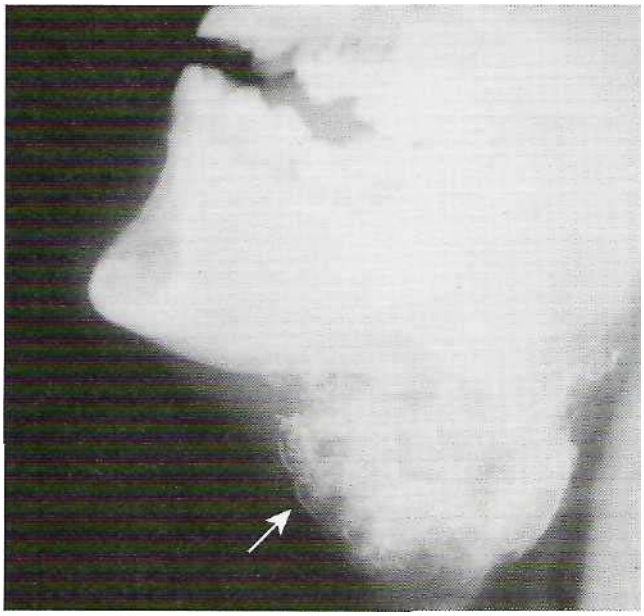


FIG. 20-8 Acinar phase of a submandibular sialogram. Normal arborization of the entire ductal system of the gland *(arrow)* is demonstrated.

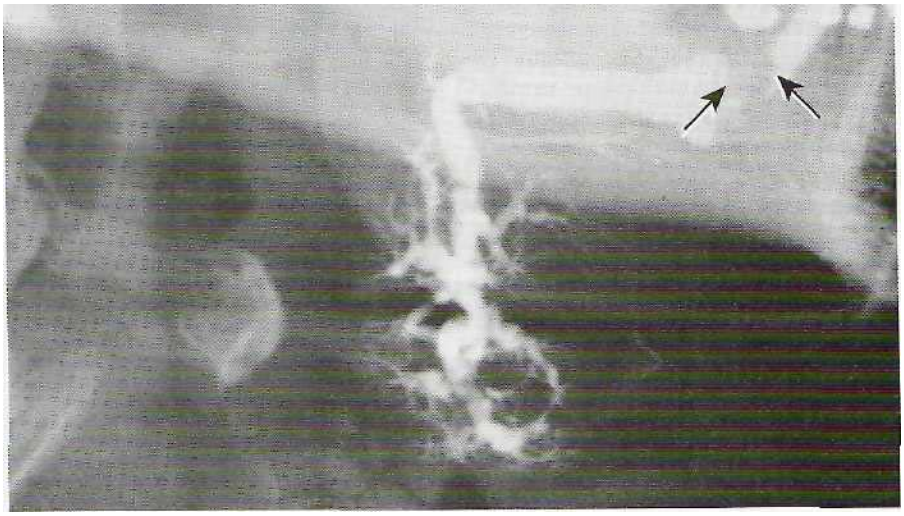


FIG. 20-9 Siaiogram of right submandibular gland. Obstruction of duct by a radiolucent sialolith *(arrows)* has caused dilation of the duct and loss of normal parenchyma of the gland.

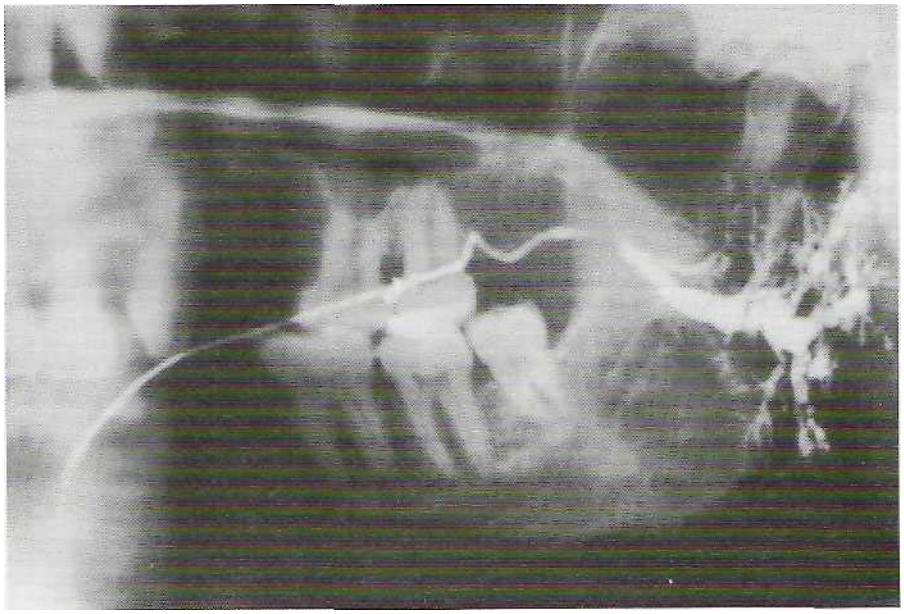


FIG. 20-10 Siaiogram of right parotid gland. The characteristic "sausage link" appearance of the duct is demonstrated, which indicates ductal damage from obstructive disease with irregular narrowing of duct caused by reparative fibrosis.

The retention of contrast in the gland or ductal system beyond 5 minutes is considered abnormal. A normal siaiogram shows a large primary duct branching gradually and smoothly into secondary and terminal ductules. Evenly distributed contrast will result in opacification of the acinoparenchyma that will outline the gland and its lobules. When a stone obstructs a salivary duct, continued secretion by the gland produces distension of the ductal system proximal to the obstruction and finally leads to pressure atrophy of the parenchyma of the gland (Fig. 20-9).

Sialodochitis is a dilation of the salivary duct secondary to epithelial atrophy as a result of repeated inflammatory or infectious processes, with irregular narrowing caused by reparative fibrosis (i.e., "sausage link" pattern) (Fig. 20-10). Sialadenitis represents inflammation mainly involving the acinoparenchyma of the gland. Patients with sialadenitis experience sacculardilation of the acini

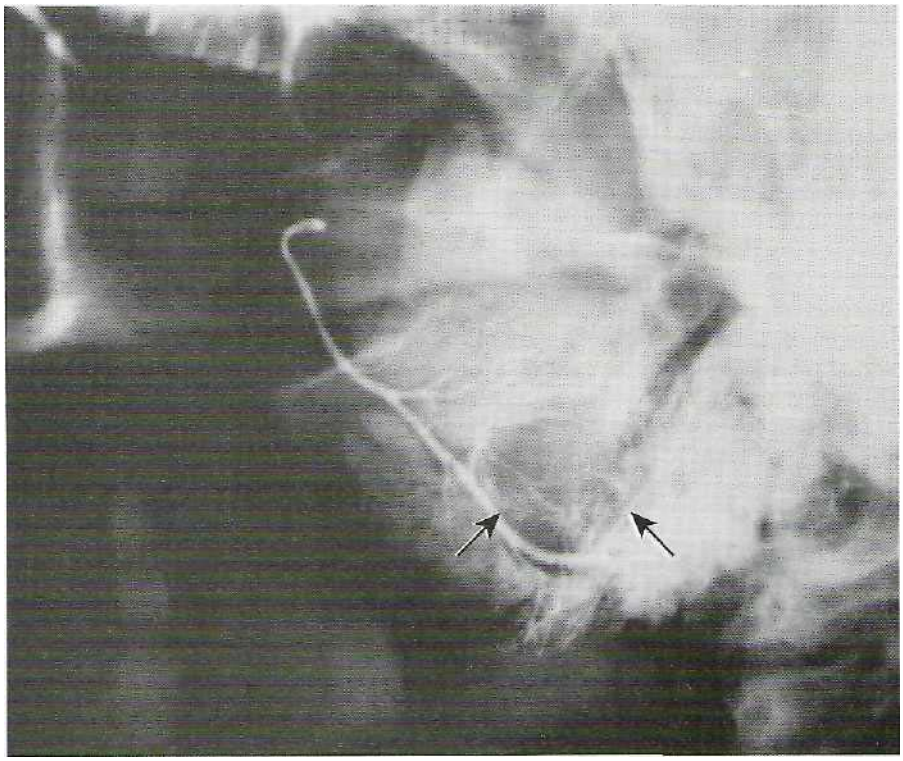


FIG. 20-11 Sialogram of right parotid gland illustrates "ball-in-hand" phenomenon *(arrows)*. The filling defect in this sialogram locates a tumor of the gland with displacement of normal surrounding ductal anatomy.

of the gland secondary to acinar atrophy and infection, which results in "pruning" of the normal arborization of the small ductal system of the gland. Centrally located lesions or tumors that occupy a part of the gland or impinge on its surface displace the normal ductal anatomy. On sialography, ducts adjacent to the lesion are curvilinearly draped and stretched around the mass, producing a characteristic "ball-In-hand" appearance (Fig. 20-11).

Sialograms are specialized radiologic studies performed by oral and maxillofacial surgeons and some interventional radiologists trained in the technique. Those inexperienced in its performance or its proper interpretation should not attempt this examination. The three contraindications to performing a sialogram are (1) acute salivary gland infections, because a disrupted ductal epithelium may allow extravasation of contrast into the soft tissues and cause severe pain and possibly a foreign-body reaction; (2) patients with a history of iodine sensitivity, especially a severe allergic reaction after a previous radiologic examination using contrast; and (3) before a thyroid gland study, because retained iodine in the salivary gland or ducts may interfere with the thyroid scan.

Computed tomography, magnetic resonance imaging, and ultrasound. The use of computed tomography (CT) has been generally reserved for the assessment of mass lesions of the salivary glands. Although CT scanning results in radiation exposure to patients, it is less invasive than sialography and does not require the use of contrast material. Additionally, CT scanning can demonstrate salivary gland calculi, especially submandibular

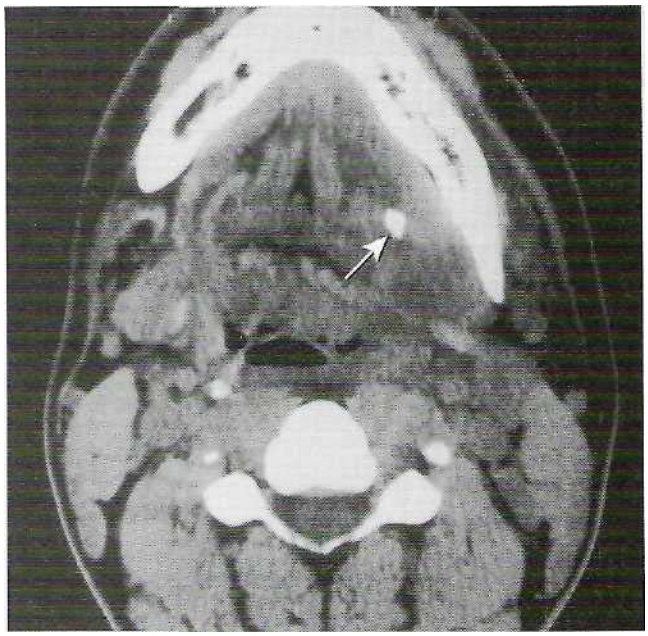


FIG. 20-12 Computerized tomographic scan of the mandible and floor of mouth shows a posterior submandibular sialolith *(arrow)*.

stones that are located posteriorly in the duct, at the hilum of the gland, or in the substance of the gland itself (Fig. 20-12).

Magnetic resonance imaging (MRI) is superior to CT scanning in delineating the soft tissue detail of

salivary gland lesions, specifically tumors, with no radiation exposure to the patient or the necessity of contrast enhancement.

Ultrasonography is a relatively simple, noninvasive imaging modality, with poor detail resolution. The primary role of ultrasonography is in the assessment of superficial structures to determine whether a mass lesion that is being evaluated is solid or cystic (fluid-filled) in nature.

Salivary scintigraphy (radioactive isotope scanning). The use of nuclear imaging in the form of radioactive isotope scanning, or salivary scintigraphy, allows a thorough evaluation of the salivary gland parenchyma, with respect to the presence of mass lesions and the function of the gland itself. This study uses a radioactive isotope (usually, technetium [Tc] 99m) injected intravenously (IV), which is distributed throughout the body and taken up by a variety of tissues, including the salivary glands. The major limitation of this study, aside from patient radiation exposure, is the poor resolution of the images obtained. Salivary gland scintigraphy may demonstrate increased uptake of radioactive isotope in an acutely inflamed gland or decreased uptake in a chronically inflamed gland, as well as the presence of a mass lesion, either benign or malignant.

Salivary Gland Endoscopy (Sialoendoscopy)

Minimally invasive modalities of diagnosis and treatment have recently been applied to the major salivary glands. Salivary gland endoscopy (sialoendoscopy) is a specialized procedure that uses a small video camera (endoscope) with a light at the end of a flexible cannula, which is introduced into the ductal orifice. The endoscope can be used diagnostically and therapeuticaUy. Salivary gland endoscopy has demonstrated strictures and kinks in the ductal system, as well as mucous plugs and calcifications. The endoscope may be used to dilate small strictures and flush clear small mucous plugs in the salivary gland ducts. Specialized devices such as small balloon catheters (similar to those used for coronary angioplasty procedures) may be used to dilate sites of ductal constriction, and small metal baskets may be used to retrieve stones in the ductal system

Sialochemistry

An examination of the electrolyte composition of the saliva (see Table 20-2) of each gland may indicate a variety of salivary gland disorders. Principally the concentrations of sodium and potassium, which normally change with salivary flow rate, are measured. Certain changes in the relative concentrations of these electrolytes are seen in specific salivary gland diseases. For example, an elevated sodium concentration with a decreased potassium concentration may indicate an inflammatory sialadenitis.

Fine-Needle Aspiration Biopsy

The use of fine-needle aspiration biopsy in the diagnosis of salivary gland tumors has been well documented. This procedure has a high accuracy rate for distinguishing between benign and malignant lesions in superficial locations. Fine-needle aspiration biopsy is performed using syringe with a 20-gauge or smaller needle. After loc anesthesia the needle is advanced into the mass lesion the plunger is activated to create a vacuum in the syringe and the needle is moved back and forth throughout the mass, with pressure maintained on the plunger. The pressure is then released, the needle is withdrawn, and fluid cellular material and fluid is expelled onto a slide and fixed for histologic examination. This allows an immediate determination of benign versus malignant disease also offers the possibility of providing a tissue diagnosis especially if the oral surgeon and oral pathologist are experienced in performing and interpreting this examination and its results.

Salivary Gland Biopsy

A salivary gland biopsy, either incisional or excision, can be used to diagnose a tumor of one of the major Sal- ivary glands, but it is usually performed as an aid in the diagnosis of SS. The lower lip labial salivary gland biopsy has been shown to demonstrate certain characteristic histopathologic changes that are seen in the major glands in SS. The procedure is performed using local anesthesia and approximately 10 minor salivary glands are removed for histologic examination (Fig. 20-13).

OBSTRUCTIVE SALIVARY GLAND DISEASE

Sialolithiasis

The formation of stones, or calculi, may occur through out the body, including the gallbladder, urinary tract, salivary glands. The occurrence of salivary gland stones is twice as common in men, with a peak incidence between ages 30 and 50. Multiple stone formation occurs approximately 25% of patients. The pathogenesis salivary calculi progresses through a series of stages beginning with an abnormality in calcium metabolism an *t* precipitation, with formation of a nidus that subsequently becomes layered with organic and inorganic material to form a calcified mass.

The incidence of stone formation varies, depending the specific gland involved (Box 20-3). The submadi- bular gland is involved in 85% of cases, which is more common than all other glands combined. A variety of contribute to the higher incidence of submandibular calculi. Salivary gland secretions contain water, electrolytes, urea, ammonia, glucose, fats, proteins, and other stances; in general, parotid secretions are more concentrated than those of the other salivary glands. T exception is the concentration of calcium, which is about twice as abundant in submandibular saliva as in parotid

BOX 20-3

Incidence of Sialolithiasis

Submandibular gland 85%

saliva (see Table 20-2). In addition, the alkaline pH of sub-mandibular saliva may further support stone formation. In addition to salivary composition, several anatomic factors of the submandibular gland and duct are important. Wharton's duct is the longest salivary duct; therefore saliva has a greater distance to travel before being emptied into the oral cavity. In addition, the duct of the submandibular gland has two sharp curves in its course: The first occurs at the posterior border of the mylohyoid muscle, and the second is near the ductal opening in the anterior floor of the mouth. Finally, the punctum of the submandibular duct is smaller than the opening of Stensen's duct. These features contribute to a slowed salivary flow and provide potential areas of stasis of salivary flow, or obstruction, that is not found in the parotid or sublingual ductal systems. Precipitated material, mucus, and cellular debris are more easily trapped in the tortuous and lengthy submandibular duct, especially when its small orifice is its most elevated location, and its flow therefore occurs against the force of gravity. The precipitated material forms the nidus of mucous plugs and either radiopaque or radiolucent sialoliths that may eventually enlarge to the point of obstructing the flow of saliva from the gland to the oral cavity.

The clinical manifestations of the presence of submandibular stones become apparent when acute ductal

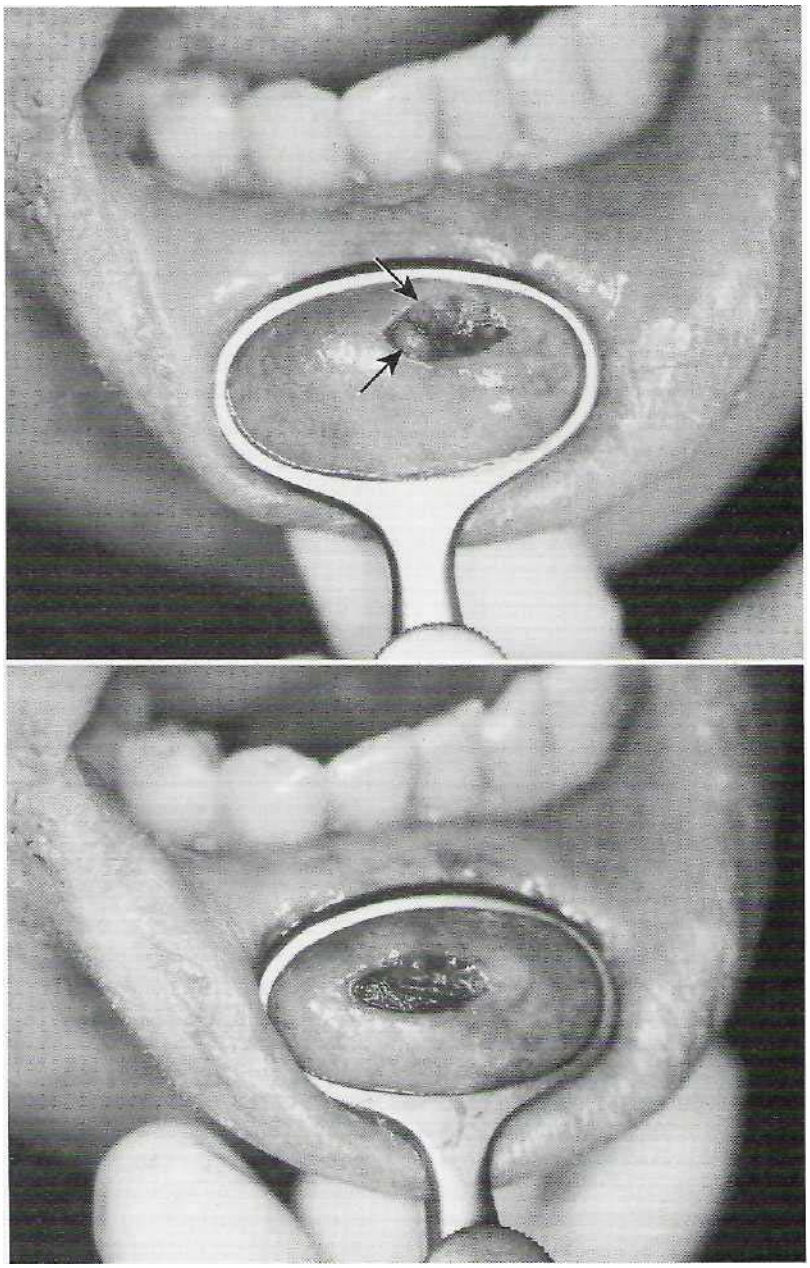


FIG. 20 – 13 **A**, Labial salivary gland biopsy. The lower lip is everted and controlled with a Chalazion clamp. An incision through mucosa permits visualization of the minor salivary glands *(arrows)*. **B**, The minor salivary glands are removed and submitted for histopathological assessment

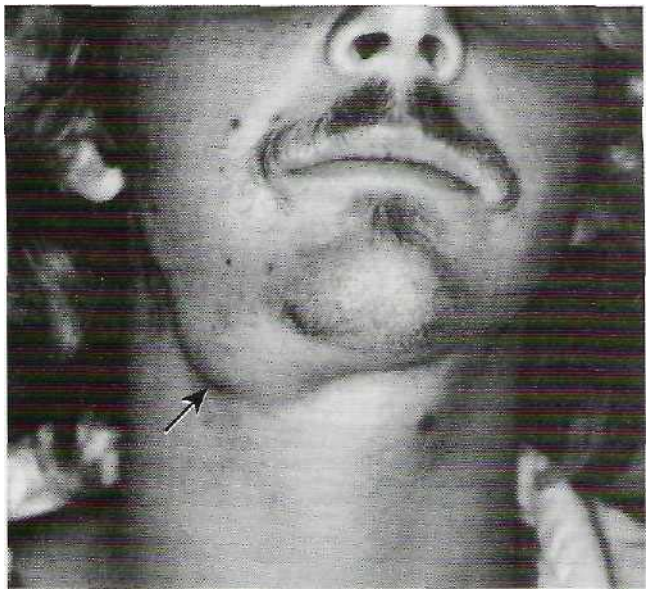


FIG. 20'14 Clinical photograph demonstrates a right submandibular swelling *(arrow)* secondary to obstruction from a submandibular sialolith.

BOX 20-4

Sialolithiasis for the General Dentist

Classic** signs **and** symptoms **of sialolithiasis
I Exacerbation of pain and swelling at mealtimes
I Check for flow from Wharton's duct
J Check for tenderness of submandibular gland
I Palpate for stone in floor of mouth
£ Check mandibular occlusal radiograph
Treatment
Anterior stone
Attempt to dilate Wharton's duct with lacrimal probes
Careful to not dislodge stone posteriorly "Milk" the gland to express stone
I If successful, prescribe salivary stimulants
Posterior stone or no stone visualized
■ Refer to oral surgeon

obstruction occurs at mealtime, when saliva production is at its maximum and salivary flow is stimulated against a fixed obstruction. The resultant swelling is sudden and is usually very painful (Box 20-4; Fig. 20-14). Gradual reduction of the swelling follows, but swelling reoccurs repeatedly when salivary flow is stimulated. This process may continue until complete obstruction, infection, or both occurs. Obstruction, with or without infection, causes atrophy of the secretory cells of the involved gland. Infection of the gland manifests itself by swelling in the floor of the mouth, erythema, and an associated lymphadenopathy. Palpation of the gland and simultaneous examination of the duct and its opening may reveal the total absence of salivary flow or the presence of purulent material.

Sialolithiasis in children is rare. Boys are more commonly affected than girls, and the submandibular gland is most commonly affected. The diagnosis can be made clinically and confirmed radiographically by plain films ultrasound, sialography, or sialoendoscopy.

The management of submandibular gland calculi depends on the duration of symptoms, the number repeated episodes, the size of the stone, and, per" most importantly, the location of the stone. I mandibular stones are classified as either *anterior* or *poste rior* stones, in relation to a transverse line between mandibular first molars. Stones that occur anterior to this line are generally well visualized on a mandibular occlusal radiograph and may be amenable to intraoral removal. Small anteriorly located stones may be rein- : through the ductal opening after dilation of the orifice._

Occasionally, it becomes necessary to remove submandibular stones via an incision made in the floor of the mouth to expose the duct and the stone. A longitudinal incision is then made in the duct, the stone is retrieved, and the ductal lining is sutured to the mucosa of the floor of the mouth. Saliva will then flow out the revised duct. This procedure, known as a *sialodochoplasty* (i.e., revision of the salivary duct), eliminates many of the factors that contributed to formation of the stone entire length of the duct is decreased, the opening created is now larger, and gravity contributes less to salivary stasis. Regardless of the procedure performed, patient are encouraged to maintain ample salivary flow by using salivary stimulants, such as citrus fruits, flavored candies or glycerin swabs. Posterior stones occur in up to 50% of cases and may be located at the hilum of the gland or within the substance of the gland itself. A routine occlusal film will likely not demonstrate the stone, a panoramic radiograph (see Fig. 20-5) or a CT scan (see Fig 20-12) may be necessary to localize the stone. In cases of posterior stones that cannot be palpated intraorally and in many instances of repeated stone formation, the submandibular gland and the stone should be removed by an extraoral approach (Fig. 20-15).

Recent clinical trials using extracorporeal shock I lithotripsy (ECSWL) have been successful in small salivary gland stones. This technology uses trarnscutaneous electromagnetic waves to break the calculus apart into smaller calcified debris particles, which can be flushed from the ductal system by the normal flow of the saliva. This procedure has few reported complications, but is limited by the size of the salivary gland stone (usually than 3 mm), the number of stones (usually less three), and the location of the stone (intraglanldular stones may be less amenable to ECSWL).

Salivary gland calculi occur much less commonly parotid gland. In general, parotid gland infection leads to stone formation; the opposite, however, is the case for the submandibular gland. The parotid gland is examined by inspection and palpation of the gland extraorally over the ascending mandibular ramus. Stensen's duct and its orifice can be examined intraorally. Palpation of the gland and simultaneous observation of the duct observation of salivary flow or the production of other material, such as purulence, from the punctum of the duct Parotid sialoliths found in the distal third of Stensen' s duct that can be palpated intraorally may be removed after

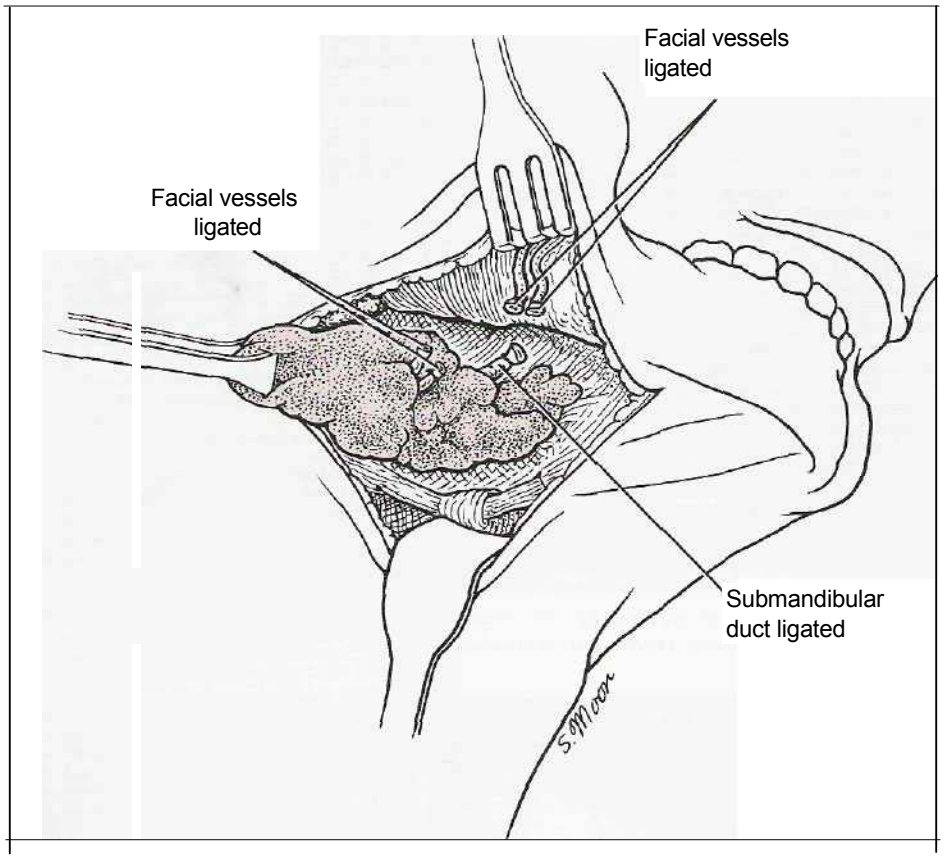


FIG. 20-15 Extraoral technique for removal of the submandibular gland.

dilatation of the duct orifice, or, if slightly more proximal, may require surgical exposure to gain access to the stone. On rare occasion the presence of a parotid stone at the hilum of the gland, or in the gland itself may necessitate an extraoral approach to remove the stone and the superficial lobe of the parotid gland.

The sublingual gland is examined by observation and bimanual palpation of the anterior third of the floor of the mouth. The minor salivary glands are examined by observation and palpation of the mucosal surfaces of the lips, buccal mucosa, palate, and floor of the mouth. Obstruction of the sublingual gland is unusual, but if it occurs, it is usually secondary to obstruction of Wharton's duct on the same side of the oral cavity. Although stone formation is rare in the sublingual and minor salivary glands, the treatment is simple excision of the stone and associated gland.

MUCOUS RETENTION AND EXTRAVASATION PHENOMENA

Mucocele

Salivary ducts, especially those of the minor salivary glands, are occasionally traumatized, commonly by lip biting, and severed beneath the surface mucosa. Subsequent saliva production may then extravasate beneath the surface mucosa into the soft tissues. Over time, secretions accumulate within the tissues and produce a pseudocyst (without a true epithelial lining) that contains thick, viscous saliva. These lesions are most common in the mucosa of the lower lip and are known as *mucoceles* (Fig. 20-16). The second most common site of mucocele formation is the buccal mucosa. Mucocele formation results in an elevated, thinned, stretched overlying mucosa that appears as a vesicle filled with a clear or blue-gray mucus. The patient frequently relates a history of the lesion filling with fluid, rupture of the fluid collection, and refilling of these lesions. Many instances of mucocele formation regress spontaneously without surgery. For persistent or recurrent lesions, the preferred treatment consists of excision of the mucocele and the associated minor salivary glands that contributed to its formation (Fig. 20-17). Usually, local anesthesia is administered via a mental nerve block, and an incision is made through the mucosa. Careful dissection around the mucocele may permit its complete removal; however, in many cases the thin lining ruptures and decompresses the mucocele before removal. The regional associated minor salivary glands are removed as well and sent for histopathologic evaluation. The recurrence rates of mucoceles may be as high as 15% to 30% after surgical removal, possibly caused by incomplete removal or repeat trauma to the minor salivary glands.

Ranula

The most common lesion of the sublingual gland is the ranula, which may be considered a mucocele of the sub-

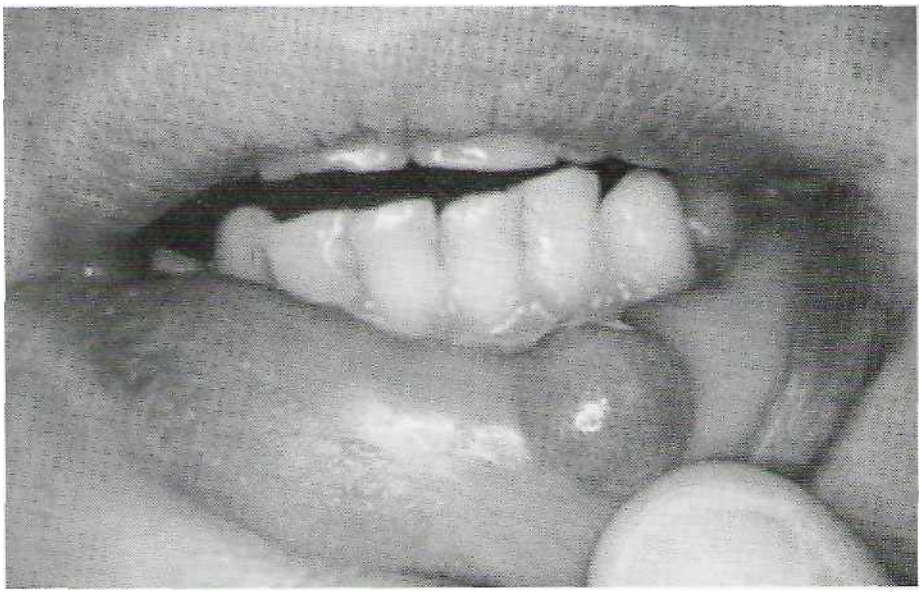


FIG.20-16 Mucocele of left lower lip. Accumulation of minor salivary gland secretions in soft tissues from ruptured minor salivary duct is visualized.

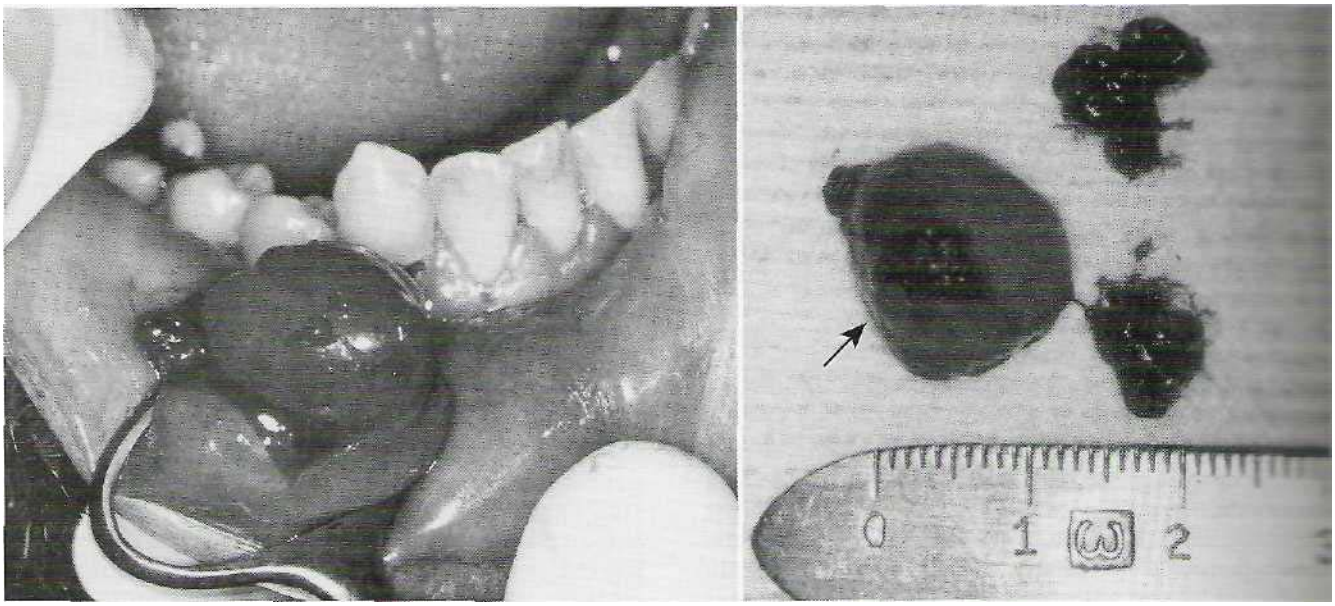


FIG.20-17 A, Excision of mucocele of right lower lip. B, Cross specimen of intact mucocele *(arrow)* is demonstrated.

lingual salivary gland. The two types of ranulas are the simple ranula and the plunging ranula. Ranulas result from either mucous retention in the sublingual gland ductal system or mucous extravasation as a result of ductal disruption. The simple ranula is confined to the area occupied by the sublingual gland in the sublingual space, superior to the mylohyoid muscle (Fig. 20-18, *A).* The progression to a plunging ranula occurs when the lesion extends beyond the level of the mylohyoid muscle into the submandibular space. Ranulas may reach a larger size than mucoceles, because their overlying mucosa is thicker and because trauma that would cause their rupture is less likely in the floor of the mouth. As a result a plunging ranula has the potential to extend into the neck and compromise the airway, resulting in a medical emergency. The differential diagnosis of a floor of mouth swelling includes ranula, lymphoepithelial cyst, epidermoid or dermoid cyst, salivary gland tumors mucoepidermoid carcinoma), and mesenchymal tumors. (e.g., lipoma, neurofibroma, hemangioma). The dirf tial diagnosis of a midline neck mass includes thyroid enlargement (i.e., goiter or tumor), thyroglossal duct cyst, dermoid cyst, and plunging ranula. The differentia diagnosis of a lateral neck mass includes lymphadenopathy, epidermoid cyst, lipoma, infectious mononucleosis, metastatic carcinoma, lymphoma, salivary gland tumors

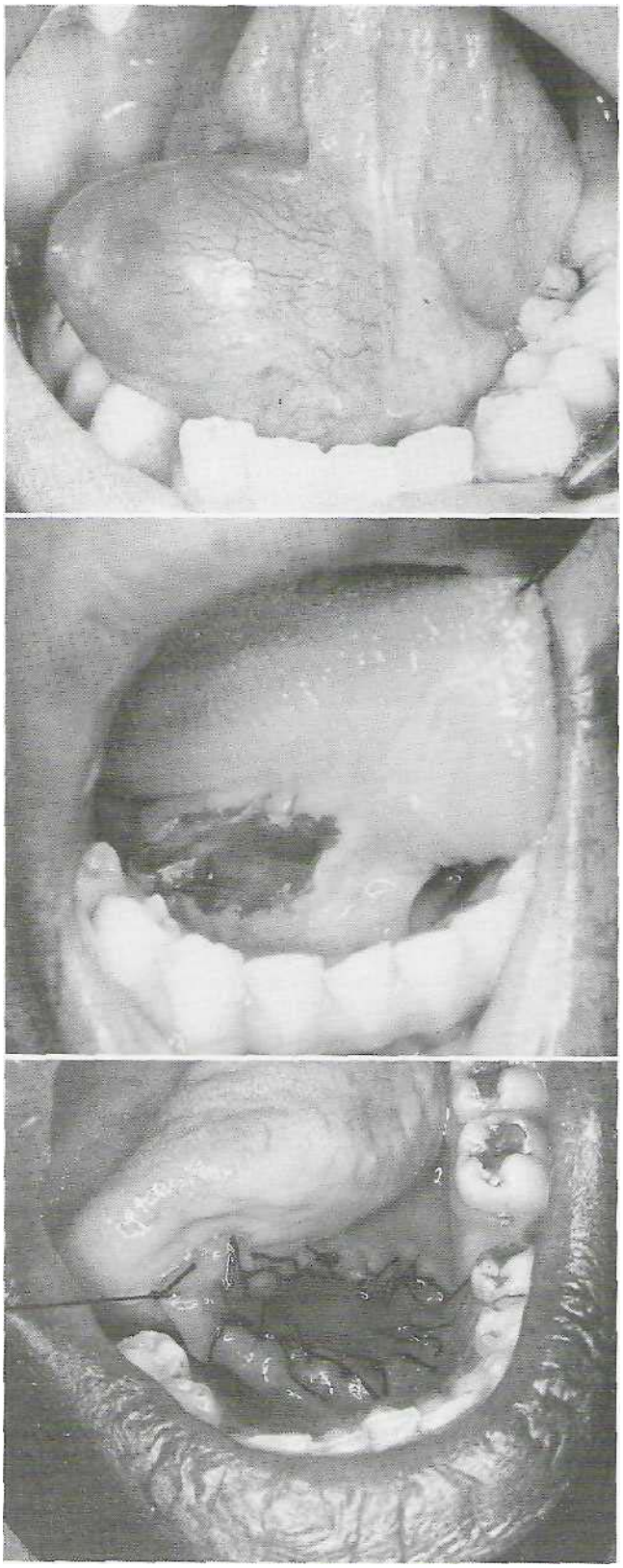


FIG. 20-18 A, Ranula in the right floor of mouth caused by accumulation of sublingual gland secretions in soft tissues secondary to rupture of salivary duct. B, Marsupialization of ranula, with excision of oral mucosa and superior wall of ranula. C, Completion of marsupialization of left floor of mouth ranula with placement of circumferential sutures.

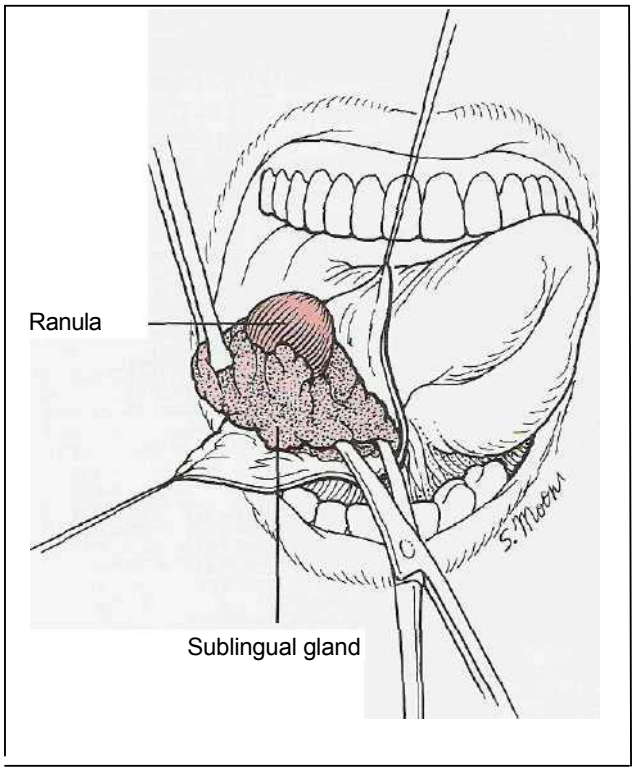


FIG. 20-19 Intraoral sublingual gland and ranula removal.

(e.g. submandibular gland or tail of the parotid gland), submandibular gland sialadenitis, lymphoepithelial cyst, sarcoidosis, tuberculosis, cat-scratch disease, cystic hygroma, carotid body tumor, or plunging ranula. The usual treatment of the ranula is marsupialization, in which a portion of the oral mucosa of the floor of the mouth is excised, along with the superior wall of the ranula (Fig. 20-18, *B).*

Subsequently, the ranula wall is sutured to the oral mucosa of the floor of the mouth and allowed to heal by secondary intention (Fig. 20-18, C). The preferred treatment for recurrent or persistent ranulas is excision of the ranula and sublingual gland via an intraoral approach (Fig. 20-19); several recent studies have indicated that this might be appropriate for initial therapy.

SALIVARY GLAND INFECTIONS

Infections of the major salivary glands can be acute or chronic and are commonly, but not always, related to obstructive disease, especially in the submandibular gland (obstruction leads to infection). The cause of acute suppurativc sialadenitis of the parotid gland usually involves a change in fluid balance that is likely to occur in patients who are elderly, debilitated, malnourished, dehydrated, or plagued with chronic illness. In these cases, gland infections are usually bilateral. The mean age of occurrence of infections is *60* years, with a slight male predilection. Salivary gland infections may be caused by a variety of organisms, including aerobic and anaerobic bacteria, viruses, fungal organisms, and mycobacteria. In most cases, mixed bacterial flora is responsible for

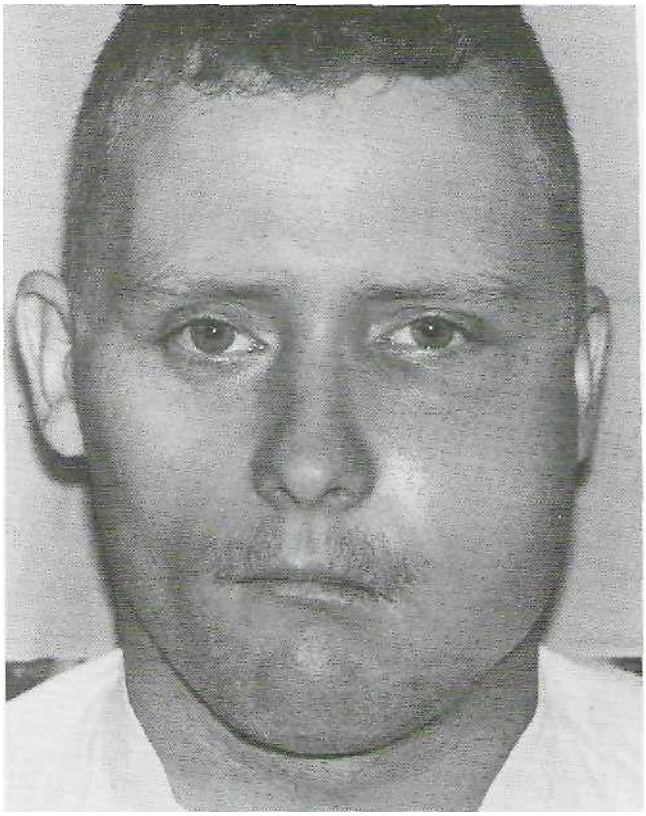


FIG. 20-20 Left parotid gland infection. This infection is extremely painful and may indicate another serious illness. Treatment requires hospitalization, intravenous antibiotics, and possibly surgical drainage.

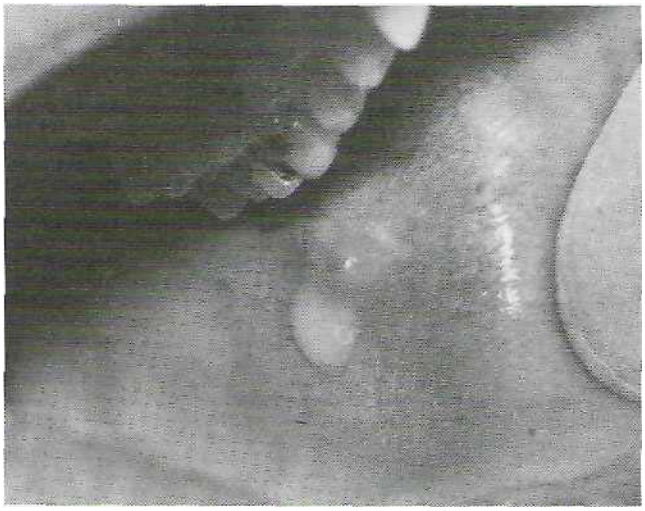


FIG. 20-21 Purulent discharge from left parotid duct is demonstrated in patient with an infection involving parotid gland.

sialadenitis. The single most common organism implicated in salivary gland infection is *Staphylococcus aureus,* because this organism normally colonizes around ductal orifices. In addition, during instances of decreased or slowed salivary flow (i.e., obstruction or dehydration), retrograde influx of *S. aureus* into the ductal system and gland occurs and results in infection.

The clinical characteristics of acute bacterial **s** gland infections include rapid onset of swelling in the preauricular (parotid gland) or submandibular regions with associated erythema and pain (Fig. 20-20). Palpation of the involved gland will reveal no flow or elicit a thick, purulent discharge from the orifice of the duct(Fig 20-21).

Treatment of bacterial salivary gland infections includes symptomatic and supportive care, including IV fluid hydration, antibiotics, and analgesics. Initial empiric antibiotics should be aimed at the most likely causing organism, S. *aureus,* and should include a cephalosporin (first generation) or antistaphylococcal semisynthetic penicillin (oxacillin or dicloxacillin). Culture and sensitivity studies of purulent material should be obtained to aid in selecting the most appropriate antibiotic for each patient.

Antibiotics should be administered IV in high doses for the majority of these patients, who ordinarily require hospitalization. On most occasions, surgery consisting of incision and drainage (I&D) becomes necessary in the management of salivary gland infections. Untreated infect: - may progress rapidly and can cause respiratory obstruction, septicemia, and, eventually, death. In some instances of recurrent salivary gland infection, the repeated insults result in irreversible functional impairment of gland ration and excision of the gland may be indicated.

Viral parotitis, or mumps, is an acute, nonsuppuppurative communicable disease. Before routine vaccination (eg., measles, mumps, rubella vaccine) against the disease began, viral parotitis occurred in epidemics during the winter and spring. It is important to differentiate viral from bacterial salivary gland infection, because viral infections are not the result of obstructive diseaseand require different treatment, not including antibiotics.

Mumps is characterized by a painful, nonerythematous. swelling of one or both parotid glands that begins 2 to 3 weeks after exposure to the virus (incubation period). This disease occurs most commonly in children between ages 6 and 8. The signs and symptoms of mumps include preauricular pain and swelling, fever, chills, and headache.

Viral parotitis usually resolves in 5 to 12 days after onset. Providing supportive and symptomatic care for fever, headache, and malaise with antipyretics, analgesics, and adequate hydration treats viral parotitis. Complications of the disease include meningitis, pancreatitis, nephritis, orchitis, testicular atrophy, and sterility in approximately 20% of young males affected.

NECROTIZING SIALOMETAPLASIA

Necrotizing sialometaplasia is a reactive, nonneoplastic inflammatory process that usually affects the minor salivary glands of the palate. However, it may involve minor salivary glands in any location. Necrotizing sialometaplasia is of unclear origin but is thought to be secondary to vascular infarction of the salivary gland lobules. Potential causes of diminished blood flow to the affected area include trauma, local anesthetic injection, smoking, diabetes mellitus, vascular disease, and pressure from a denture prosthesis. The usual age range of affected patients is between 23 and 66 years.

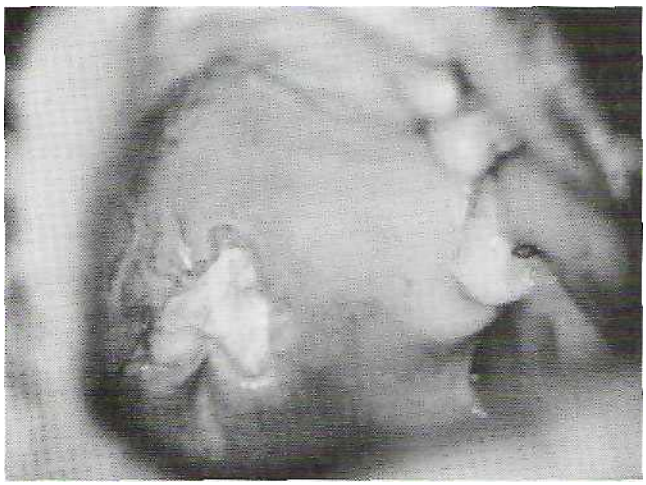


FIG. 20-22 Necrotizing sialometaplasia of posterior palate with ulceration.

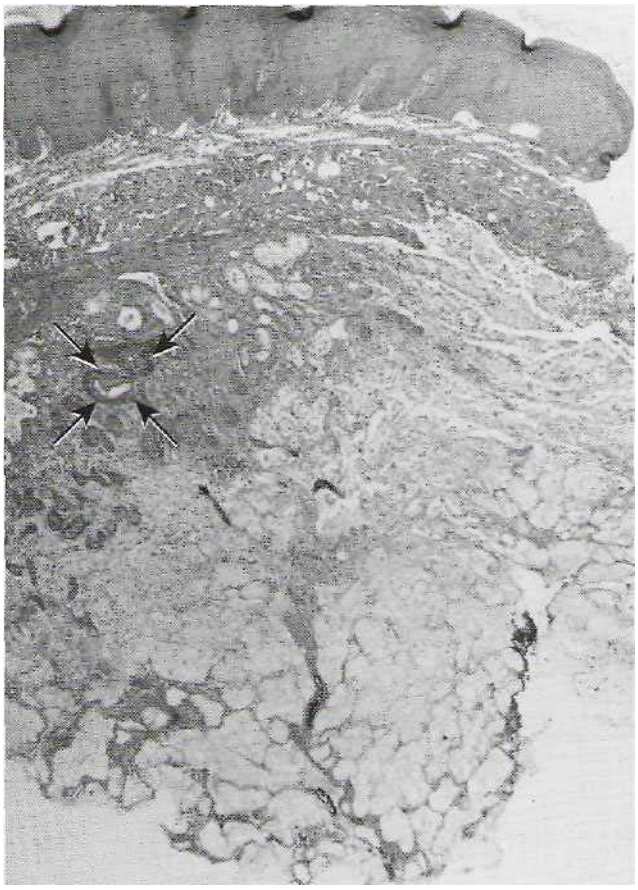


FIG. 20-23 Histopathology of necrotizing sialometaplasia shows pseudoepitheliomatous hyperplasia *(arrows)* that appears similar to infiltration of a squamous cell carcinoma.

Lesions usually appear as large (1 to 4 cm), painless or painful, deeply ulcerated areas lateral to the palatal midline and near the junction of the hard and soft palate (Fig. 20-22). Although lesions are usually unilateral, bilateral involvement may occur. Some patients may report a prodromal flulike illness before the onset of the ulceration. clinically and histologically (Fig. 20-23), it resembles a malignant carcinoma (squamous cell or mucoepider-moid carcinoma). The appropriate diagnosis and management of this disease relies on evaluation by an oral surgeon and pathologist who are familiar with this entity, because the result of a misdiagnosis may be extensive, unwarranted surgical resection. Helpful histologic criteria for distinguishing necrotizing sialometaplasia from a malignant process include the maintenance of the overall salivary lobular morphology, the generally nondys-plastic appearance of the squamous islands or nests, and evidence of residual ductal lumina within the epithelial nests. The ulcerations of necrotizing sialometaplasia usually heal spontaneously within 6 to 10 weeks after their onset and require no surgical management.

SJOGREN'S SYNDROME

SS is a multisystem disease process with a variable presentation. The two types of SS are (1) primary SS, or sicca syndrome, characterized by xerostomia (dry mouth) and keratoconjunctivitis sicca (dry eyes); and (2) secondary SS, which is composed of primary SS and an associated connective tissue disorder, most commonly rheumatoid arthritis. Although the cause of SS is unknown, there appears to be a strong autoimmune influence. SS shows a female predilection of 9:1, with over 80% of affected individuals being females with a mean age of 50 years.

Generally, the first symptoms to appear are arthritic complaints, followed by ocular symptoms, and, late in the disease process, salivary gland symptoms. The involvement of the salivary and lacrimal glands results from a lymphocytic replacement of the normal glandular elements. The xerostomia results from a decreased function of both the major and minor salivary glands, with the parotid gland being the most sensitive. The diagnosis of SS is suggested by the patient's complaints and by a variety of abnormal immunologic laboratory tests. The oral component of SS may be diagnosed using salivary flow rate studies and sialography, but the use of a labial minor salivary gland biopsy (see Fig. 20-13) currently is considered to be highly accurate in aiding the diagnosis. The histopathologic changes seen in the minor glands are similar to those in the major glands (parotid). Keratoconjunctivitis sicca is suggested by the patient's complaints and a Schirmer's test for lacrimal flow (Fig. 20-24). The treatment for SS includes symptomatic care with artificial tears for the dry eyes and salivary substitutes for the dry mouth. Additionally, the medication pilocarpine (Sala-gen) or the Biotene products may be useful to stimulate salivary flow from the remaining functional salivary gland tissue.

TRAUMATIC SALIVARY GLAND INJURIES

Traumatic injuries, particularly lacerations, involving the salivary glands and their ducts may accompany a variety of facial injuries, including fractures. Injuries that occur in close proximity to one of the major salivary glands or ducts require careful evaluation.

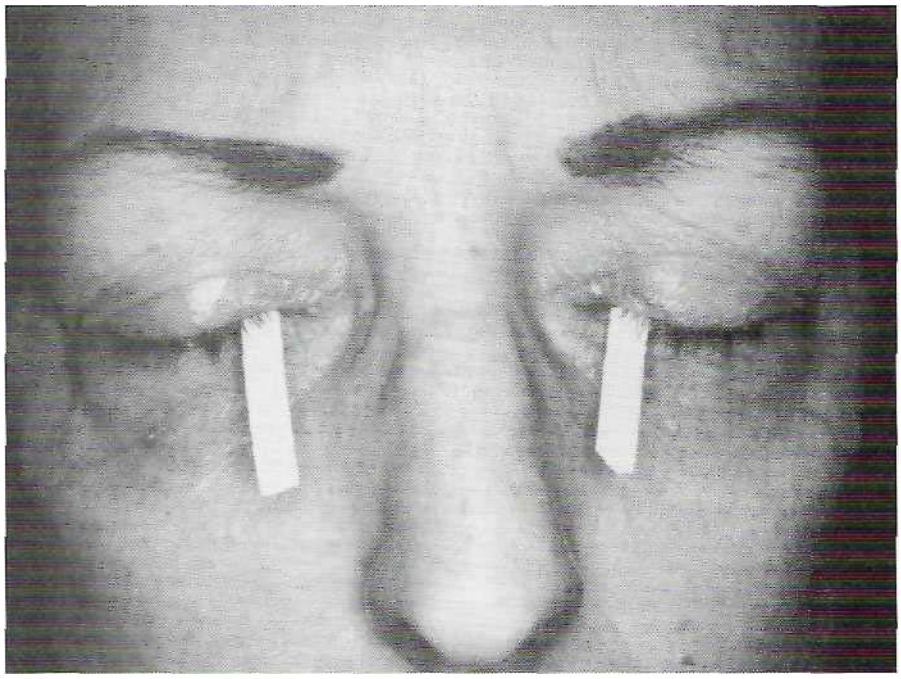


FIG. 20-24 Schirmer's test for dry eyes in a patient with Sjogren's syndrome. Filter paper is placed in the ocular fornix and observed for time limit.

Facial lacerations may involve not only the gland and its ductal system, but also branches of the facial nerve and branches of major facial vessels. These structures require meticulous attention for appropriate diagnosis and prompt repair. Repair may include ductal anastomoses, in which the proximal and distal portions of the duct are identified, a plastic or metal catheter is placed as a stent, and the duct is sutured over the stent (Fig. 20-25). The catheter usually remains in place for 10 to 14 days for epithelialization of the duct to occur. Additionally, nerve anastomoses may he required and performed by placing epineurial sutures, using magnification, to reapproximate the nerve stumps. The lacerations are closed in a usual layered fashion, after debridement of the soft tissue wounds to cleanse the site of entrapped particles, such as glass or dirt. Potential sequelae of trauma involving the major salivary glands include infection, facial paralysis, cutaneous salivary gland fistula, sialocele formation, and duct obstruction as a result of scar formation, with eventual glandular atrophy and decreased function. The involved gland may eventually require surgical removal.

NEOPLASTtC SALIVARY GLAND DISORDERS

Although a comprehensive discussion of salivary gland neoplasms is beyond the scope of this chapter and many other sources are available for this information, a brief review of several important aspects of the more common lesions is warranted. Salivary gland tumors occur much more commonly in the major glands (80% to 85%), as opposed to the minor glands {15% to 20%) (Table 20-3). Additionally, between 75% and 80% of major gland tumors are benign, whereas 50% to 55% of minor gland tumors are benign. The overwhelming majority of salivary tumors occur in the parotid gland, and the majority of those are benign (mostly pleomorphic adenomas).

TABLE 20-3

Salivary Gland Tumor Distribution

Location of Tumor	Occurance
Major glands	80%-85%
Parotid gland	85%-90%
Submandibular gland	5%-10%
Sublingual giand	Rare
Minor glands	15%-20%
Palate	55%
Lips	15%
Remainder	Rare

Benign Salivary Gland Tumors

The *pleomorphic adenoma,* or *benign mixed tumor,* is the most common salivary gland tumor. The mean age of occurrence is 45 years, with a male-to-female ratio of 3:2. In the major glands, the parotid gland is involved in over 80% of cases; in the minor glands, the most common intraoral site is the palate (Fig. 20-26). Pleomorphic adenomas are usually slow-growing, painless masses. The histopathology shows two cell types: (1) the ductal epithelial cell and (2) the myoepithelial cell, which may differentiate along a variety of cell lines *(pleomorphic* means many forms). A connective tissue capsule exists, which may be incomplete. The treatment involves complete sur-

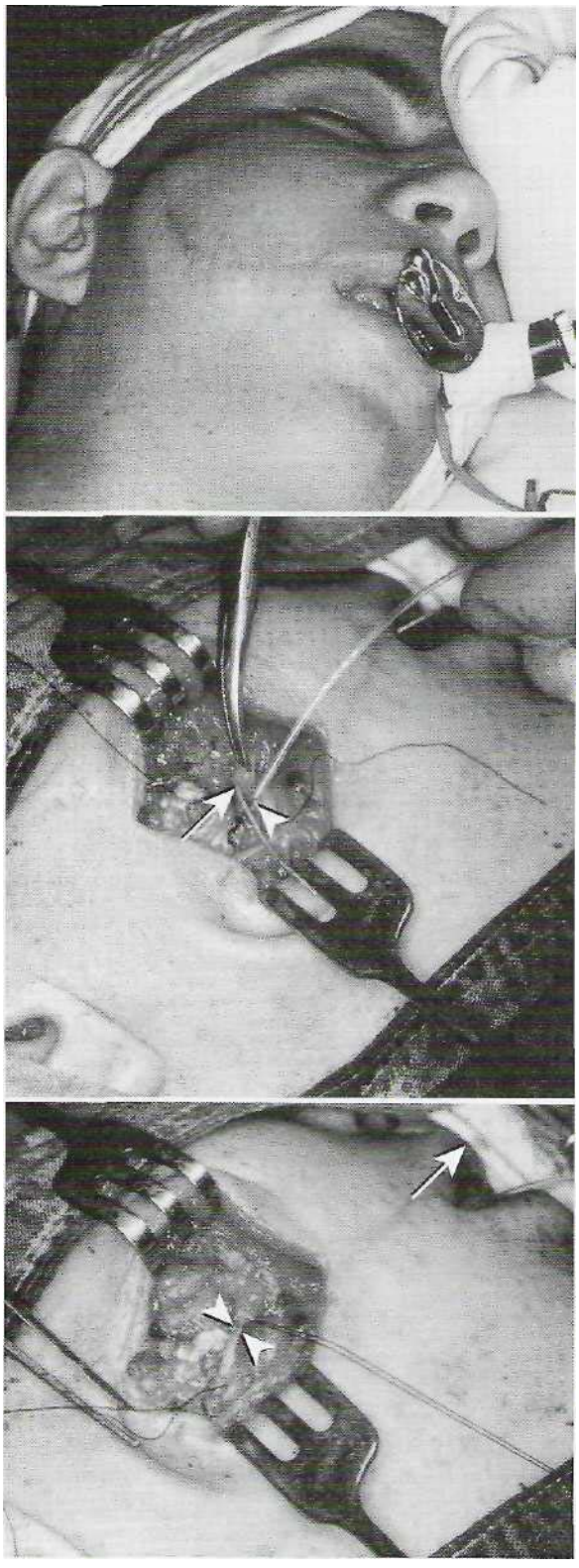


FIG. 20-25 A, Patient who had cheek laceration repaired with failure to appreciate a Stensen's duct laceration and subsequently developed a sialocele (localized collection of saliva). B, Operative repair of Stensen's duct laceration with a metal probe in the distal duct *(arrow)* and a plastic catheter *(arrowhead)* placed into the proximal portion of the duct. C, Repair of Stensen's duct laceration via suturing over a pfastic stent *(arrowheads)* placed via intraoral cannulation of Stensen's duct *(arrow)*.

gical excision with a margin of normal uninvolved tissue. Parotid lesions are treated with removal of the involved lobe along with the tumor. Recurrence is possible in rare occasions, as well as a small risk (5%) of malignant transformation to a *carcinoma ex pleomorphic adenoma.*

Warthin's tumor, or *papillary cystadenoma lymphoma tosum,* almost exclusively affects the parotid gland, specifically the tail of the parotid gland (Fig. 20-27). The peak incidence is in the sixth decade of life, with a male-to-female ratio of 7:1. This lesion presents as a slow-growing, soft, painless mass. Warthin's tumor is believed to be caused by entrapped salivary epithelial rests within developing lymph nodes. The histopathology shows an epithelial component in a papillary pattern and a lymphoid component with germinal centers. The treatment of this lesion is simple surgical excision, and recurrence is rare.

The *monomorphic adenoma* is an uncommon solitary lesion composed of one cell type, affecting predominantly the upper lip minor glands *(canalicular adenoma)* (Fig. 20-28) and the parotid gland *(basal cell adenoma).* The mean age of occurrence is 61 years, and the lesion usually presents as an asymptomatic, freely movable mass. The histopathology reveals an encapsulated lesion composed of one type (monomorphic) of salivary ductal epithelial cell. The treatment is simple surgical excision.

Malignant Salivary Gland Tumors

The *mucoepidermoid carcinoma* is the most common malignant salivary gland tumor. It comprises 10% of major gland tumors (mostly parotid) (Fig. 20-29) and 20% of minor gland tumors (mostly palate) (Fig. 20-30). This lesion may occur at any age, but the mean age is 45 years. The male-to-female ratio is 3:2. The clinical presentation is a submucosal mass that may be painful or ulcerated. The mass may appear to have a bluish tinge because of the mucous content contained within the lesion. An intraosseous form of mucoepidermoid carcinoma may present as a multilocular radiolucency of the posterior mandible. The histopathology shows three cell types: (1) mucous cells, (2) epidermoid cells, and (3) intermediate (clear) cells. The proportion of each cell type helps to grade the mucoepidermoid carcinoma as high-, intermediate-, or low-grade lesions. The higher the grade, the more predominance of epidermoid cells and pleo-morphism, lack of mucous cells and cystic areas, and overall more aggressive behavior. The treatment of low-grade lesions is wide surgical excision with a margin of uninvolved normal tissue; high-grade lesions require more aggressive surgical removal with margins, and, possibly, local radiation therapy. The low-grade lesions have a 95% 5-year survival rate, whereas the high-grade lesions have less than a 40% 5-year survival rate.

The *polymorphous low-grade adenocarcinoma* is the second most common intraoral salivary gland malignancy. This lesion was first described in 1983; before its identification, many cases were probably misdiagnosed as adenoid cystic carcinoma. The most common site is the junction of the hard and soft palates (Fig. 20-31). The male-to-female ratio is 3:1, with a mean age of 56 years. These tumors present as slow-growing, asymptomatic masses

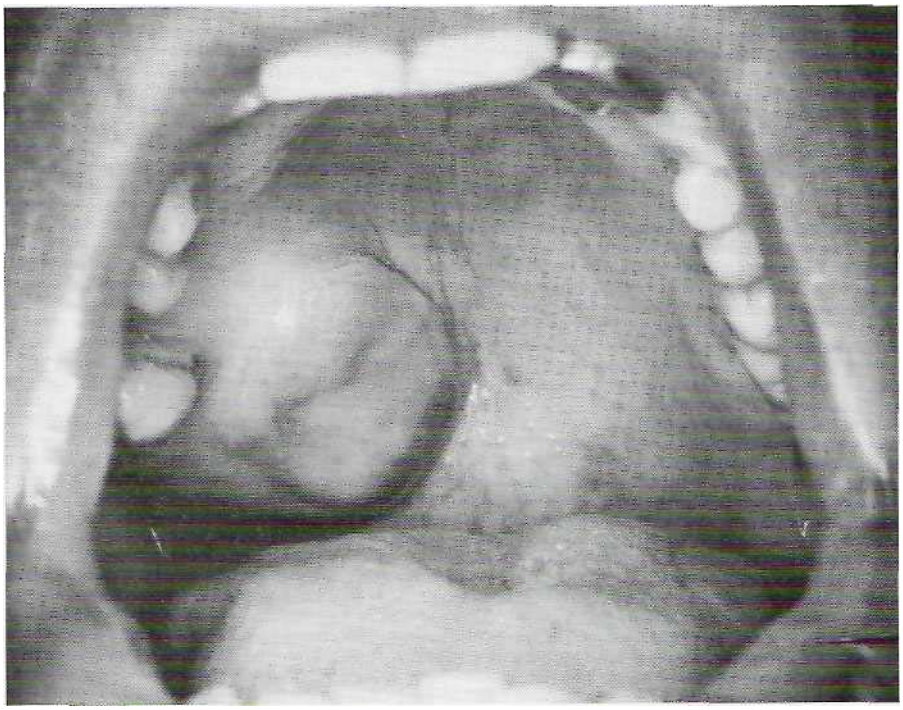


FIG. 20-26 Pleomorphic adenoma of the right palate.

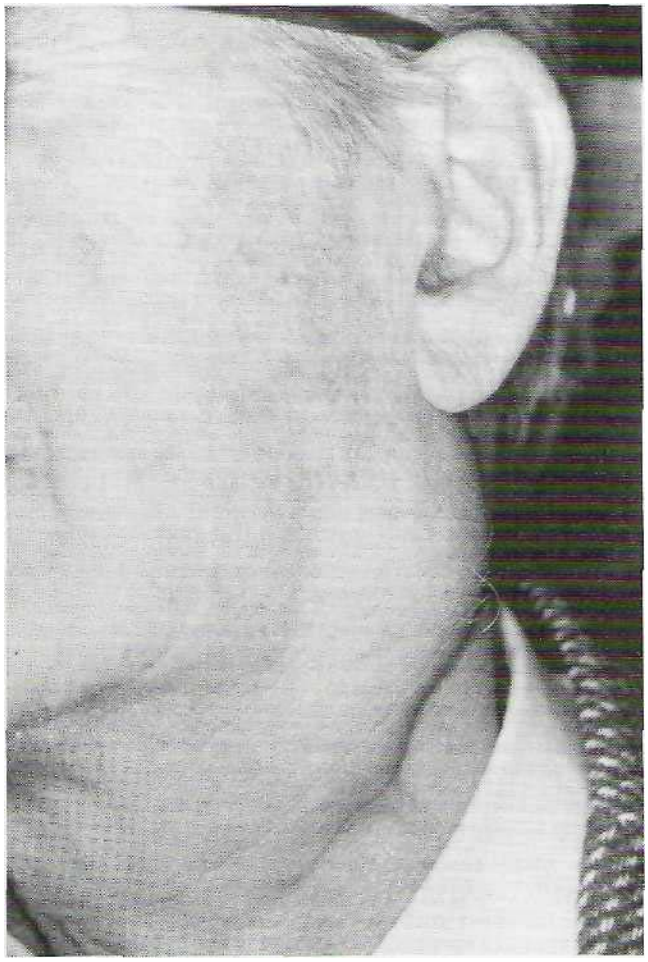


FIG. 20-27 Warthin's tumor of the tail of the left parotid gland.

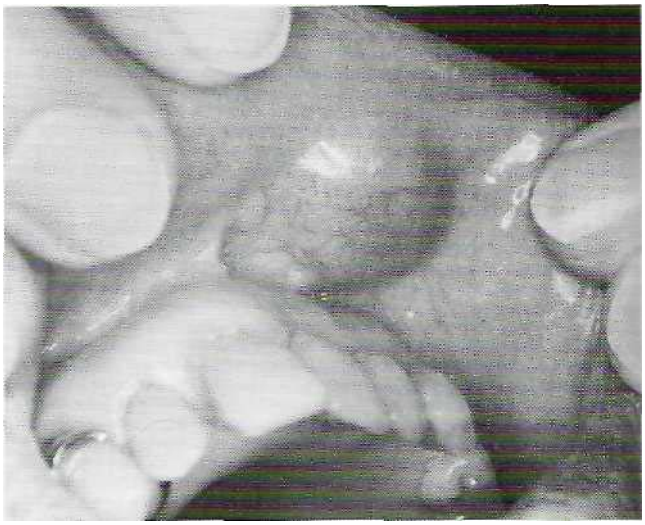


FIG. 20-28 Monomorphic (canalicular) adenoma of the left upper lip.

that may be ulcerated. The histopathology shows many cell shapes and patterns (polymorphous). Patients experience an infiltrative proliferation of ductal epithelial cells in an "Indian file" pattern. This lesion shows a predilection for invasion of surrounding nerves. The treatment of this tumor is wide surgical excision, with a relatively high recurrence rate of 14%.

The *adenoid cystic carcinoma* is the third most common intraoral salivary gland malignancy, with a mean age of 53 years and a male-to-female ratio of 3:2.

Approximately 50% of these tumors occur in the parotid gland, whereas the other 50% occur in the minor

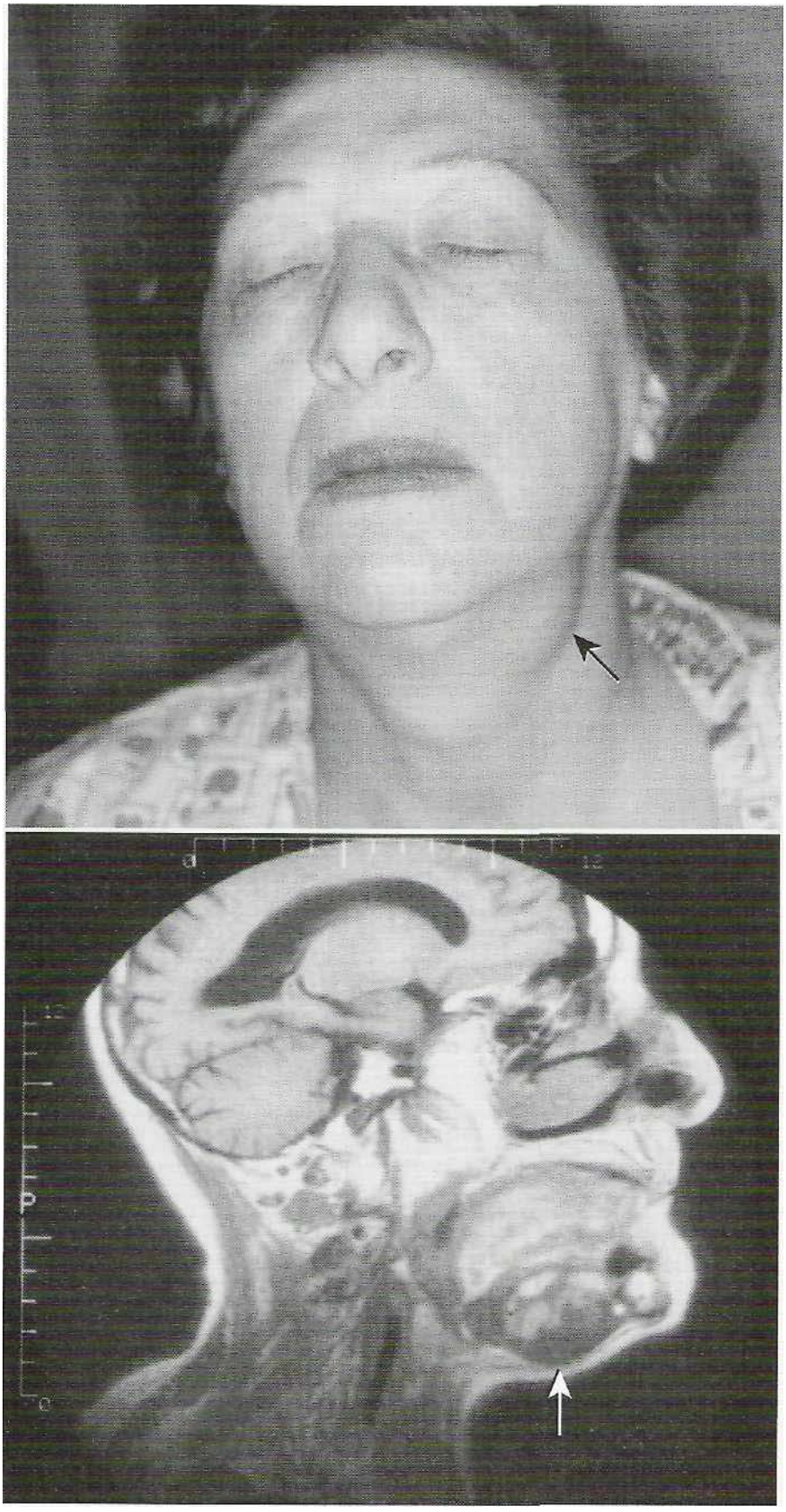


FIG. 20-29 **A**, Mucoepidermoid carcinoma of the left submandibular gland *(arrow)*. **B**, Magnetic resonance image shows the mucoepidermoid carcinoma of the left submandibular gland *(arrow)*.

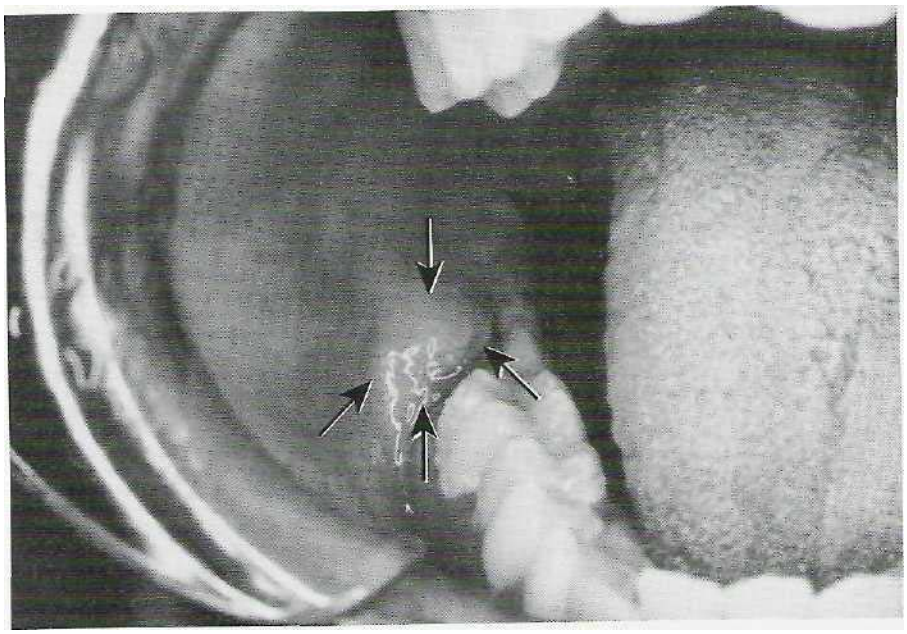


FIG. 20-30 Mucoepidermoid carcinoma of the right retromolar pad minor salivary glands *(arrows).*

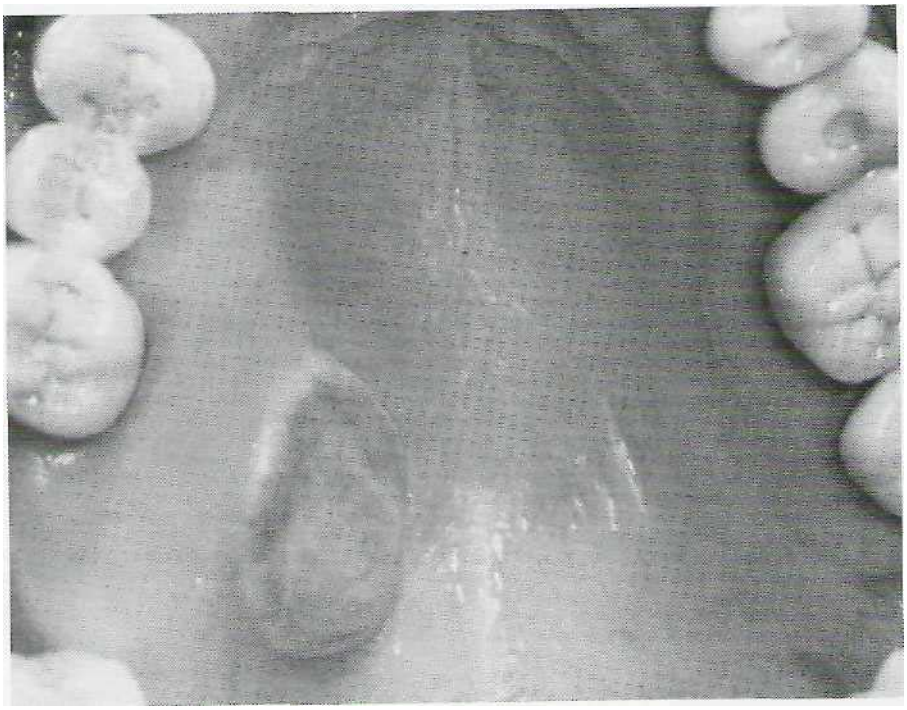


FIG. 20-31 Polymorphous low-grade adenocarcinoma of the right palate.

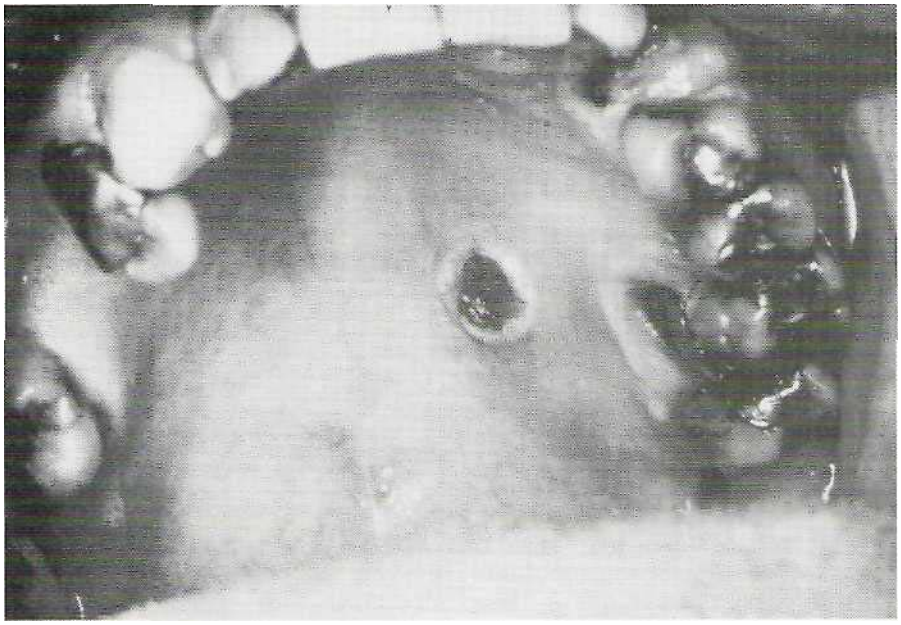


FIG. 20-32 Adenoid cystic carcinoma of the left palate.

glands of the palate (Fig. 20-32). These present as slow-growing, nonulcerated masses, with an associated chronic dull pain. Occasionally, parotid lesions may result in facial paralysis as a result of facial nerve involvement. The histopathology demonstrates an infiltrative proliferation of basaloid cells arranged in a cribriform ("Swiss cheese") pattern. As seen in the polymorphous low-grade adenocarcinoma, there may be perineural invasion. The treatment is wide surgical excision, followed in some cases by radiation therapy. The prognosis is poor despite aggressive therapy.

BIBLIOGRAPHY

Abaza NA, Miloro M: The role of fine-needle aspiration in oral and maxillofacial diagnosis. In Gold L, editor: Diagnosis and management of oral pathologic lesions, *Oral Maxillofac Surg Clin North Am* 6(3):401, 1994.

Abaza N et al: The role of labial salivary gland biopsy in the diagnosis of Sjogren's syndrome: report of three cases, / *Oral Maxillofac Surg* 51(5):574, 1993.

Baurmash HD: Marsupialization for treatment of oral ranula: a second look at the procedure, / *Oral Maxillofac Surg* 50:1274, 1992.

Berry RL: Sialadenitis and .sialolithiasis: diagnosis and management, *Oral Maxillofac Surg Clin North Am* 7:479, 1995.

Carlson ER: Salivary gland tumors: classification, histogenesis, and general considerations, *Oral Maxillofac Surg Clin North Am* 7:519, 1995.

Curtin HD: Assessment of salivary gland pathology, *Otolaryngol Clin North Am* 21:547, 1988.

Delbalso A: Salivary imaging, *Oral Maxillofac Surg Clin North Am* 7:387, 1995.

Goldberg MH, Bevilacqua RG: Infections of the salivary glands, *Oral Maxillofac Surg Clin North Am* 7:423, 1995.

Lustmann J, Regev E, Melamed Y: Sialolithiasis: a survey on 245 patients and a review of the literature, *Int] Oral Maxillofac Surg* 19:135, 1990.

Mandel ID: Sialochemistry in diseases and clinical situations affecting salivary glands, *Crit Rev Clin Lab Sci* 12:321, 1980.

Nahlieli O et al: Diagnosis and treatment of strictures and kinks in salivary gland ducts, / *Oral Maxillofac Surg* 59:484, 2001.

Nahlieli O et al: Pediatric sialolithiasis, *Oral Surg Oral Med Oral Pathoi Oral Radiol Endod* 90:709, 2000.

Regezzi JA, Sciubba JJ: Salivary gland diseases. In Regezzi JA, Sciubba JJ, editors: *Oral pathology: clinical-pathologic correlations,* ed 4, Philadelphia, 2003, WB Saunders.

Topazian RG, Goldberg MH, Hupp JR: *Oral and maxillofaciai infections,* ed 4, Philadelphia, 2002, WB Saunders.

Van der Akker HP: Diagnostic imaging in salivary gland disease, *Oral Surg Oral Med Oral Pathoi* 66:625, 1988.

Van Sickels JE, Alexander JM: Parotid duct injuries, *Oral Surg* 52:364, 1981.

Yoshimura Y et al: A comparison of three methods used for treatment of ranula, / *Oral Maxillofac Surg* 53:280, 1995.

aring GP: Trauma to the parotid region, / *LaryngolOtol* 101:475, 1987.