

## **Merck &20: Dental and Oral Disorders**

### **Chapter 245: Dentistry in Medicine**

#### **Medical-Dental Consultation**

A dentist should consult a physician when he suspects systemic disease, when he needs to evaluate a person's ability to withstand general anesthesia or extensive oral surgery, or when an emergency occurs in a dental office.

A physician should consult a dentist on behalf of a child with abnormal growth manifested by peculiar facies, delayed tooth eruption, or gross malformation or malalignment of teeth; or for a patient with cleft lip or palate, jaw fracture, oral neoplasm, or newly discovered lump in the neck. Dental consultation is also indicated for obscure facial pain, for unexplained swelling or cellulitis of the neck that might have originated in an infected tooth, or for infection of the parapharyngeal space that might have followed an abscess of a lower posterior tooth. Physicians should be familiar with dental procedures and the amount of trauma and risk involved to patients with systemic disease. Dentists who search for oral cancer and who refuse to extract teeth unless valid dental reasons exist are consultants to be prized.

Clarification of the cause of face, head, and neck symptoms often necessitates the pooled knowledge of both physician and dentist. Obscure causes of **face, head, and neck pain** include malocclusion, poorly fitting dental prostheses, disease of the temporomandibular joints, unilateral mastication, spasm of the muscles of mastication, and occult cavities in the jawbones. Referred pain may make diagnosis difficult; for example, pain referred to the ear may arise from an inflammation of the gingival flap about a partly erupted mandibular 3rd molar, or from the back of the tongue in glossopharyngeal neuralgia. Paresthesias of the lower lip may follow damage to the inferior alveolar nerve during extraction of a mandibular molar, but may also be a rare sign of an oral neoplasm compressing that nerve. Conversely, percussion tenderness in several maxillary teeth may indicate nasal or antral disease adjacent to the root tips.

Patients undergoing oral surgery warrant the same preoperative medical workups and postoperative care as those undergoing procedures of comparable scope and severity elsewhere in the body. Oral surgical procedures include simple extractions; removal of impacted teeth; alveoloplasty (cartilage grafts or resection of mucosa with reattachment to deepen the mucobuccal fold so that dentures can be retained better); repair of jaw fractures; correction of protruding mandibles or retruded or small jaws; orthognathic surgery (repositioning an area of alveolar bone and its contained teeth) for severe malocclusion; excision of soft tissues, bone cysts, and neoplasms; and surgery on the temporomandibular joint to remedy arthritis, developmental anomalies, or the results of trauma.

#### **Dental Care of Patients with Systemic Disorders**

Dental care is occasionally hazardous. Its risks can be minimized by (1) doing elective dental procedures when the medical patient is best able to withstand the inherent trauma and (2) encouraging healthy individuals to practice oral hygiene so that, should systemic disease

develop, massive dental treatment in a belated attempt to remedy prolonged neglect is unnecessary and will not delay medical therapy or cause additional complications.

Routine oral surgery or periodontal treatment puts medical patients at risk when the normal inflammation required for healing is inhibited by such drugs as corticosteroids, immunosuppressive agents used in organ transplantation, and cytotoxic drugs given for cancer. Hemorrhage, delayed healing, local infection, and even septicemia may occur. Dental care should be carried out and time for healing allowed before using such systemic drugs.

People with **bleeding disorders** should have teeth filled to avoid subsequent extractions. Filling a tooth is almost always a bloodless procedure. Exceptions occur: (1) Deep decay that has entered or has almost entered the pulp mandates complete removing the decayed tooth structure, to prevent recurrent caries; the minimal bleeding from the pulp that follows the removal is easily controlled by pressure. (2) Minimal gingival lacerations may occur from instrumentation during preparation for treating interproximal caries. Cavity preparation involving the occlusal surface of a tooth that is not deeply decayed should be completely bloodless.

Suspected **bleeding or coagulation disorders** should be assessed prior to oral surgery. In patients with acute forms of **leukemia, thrombocytopenia, or hepatitis**, extractions should be delayed until the condition improves or stabilizes. Prolonged bleeding following extraction or periodontal procedures may occur in patients with **polycythemia vera** or **macroglobulinemia, disorders of platelet number and function**, and severe **liver disease** with diminished vitamin K-associated plasma coagulation factors or increased fibrinolytic activity. In coagulation disorders, extractions and regional block anesthesia often require pretreatment with the appropriate coagulation factor and perhaps aminocaproic acid immediately before, during, and after the oral surgical procedure.

In **leukemia**, oral hygiene is vital because periodontal disease favors occurrence of gingival bleeding and probably local tissue infiltration. Infection often follows extraction in disorders with **granulocytopenia**.

**Cardiovascular patients** may be adversely affected by dental procedures. Following a myocardial infarction, dental procedures should be delayed for 3 mo, if possible, and anticoagulant dosage may need temporary reduction (prothrombin time to about 1.5 times the control value) to avoid undue postextraction bleeding. Individuals with pulmonary or cardiac disease who require inhalation anesthesia for dental procedures should be treated in a hospital. Tooth extraction, scaling (removal of calculus), or other periodontal procedures are followed by bacteremia; patients with mitral valve prolapse, congenital or rheumatic heart disease, or a prosthetic cardiac valve are predisposed to bacterial endocarditis and should receive antibiotics before and after such dental procedures. It is particularly important for them, as for patients with coagulation disorders, that teeth be filled to avoid extractions and that preventive oral hygiene be practiced to minimize tooth decay and gingivitis.

Epinephrine, used as a vasoconstrictor to potentiate the duration of local anesthetics, may cause arrhythmias or exacerbate hypertension as the exogenous hormone adds to the anxiety or fear-induced endogenous level; cardiac ischemia may result. Sedation or tranquilization is advantageous in fearful cardiovascular patients and facilitates treatment.

Electrical equipment such as a cautery, a pulp tester, and even the dental handpiece (drill) can interfere with pacemakers. Such patients and their dentists should be forewarned so that appropriate measures may be taken. The horizontal position of a dental chair may be intolerable to patients with congestive heart failure, and those on antihypertensive drugs may develop orthostatic hypotension on arising.

Infections may follow dental procedures such as tooth extraction, particularly of abscessed or periodontally involved teeth. **Bacteremia** may result in endocarditis, mediastinitis, thrombophlebitis of the jugular veins, pneumonia, empyema, meningitis, brain abscess, and cavernous sinus thrombophlebitis. Mediastinitis, thrombophlebitis, a parapharyngeal abscess, or a cellulitis of the floor of the mouth may develop from contiguity with an abscessed tooth, whether or not endodontic treatment (root canal) was performed. Infectious material, such as fragments of teeth or fillings or pus from periodontal infection, may be aspirated and cause a lung abscess.

**Chemotherapy for neoplasms** includes drugs (i.e. doxorubicin, 5-fluorouracil, bleomycin, dactinomycin, and methotrexate) that cause stomatitis; the severity is often related to the degree of periodontal disease present. Before instituting such drugs, it is advisable to remove calculus (tartar) and improve the health of periodontal tissue to minimize gingival hemorrhage, tissue sloughing, oral pain, and consequent poor food intake. Subsequent proper use of the toothbrush and dental floss will minimize the likelihood of stomatitis.

**Prior to radiotherapy** of the oral region, patients should have required oral surgery, periodontal treatment, restoration of salvageable teeth, and fluoride treatment (to minimize caries following xerostomia secondary to irradiation and destruction of the salivary glands). Dental work should be completed and healing permitted before radiotherapy begins. *Extraction of teeth from previously irradiated tissues is commonly followed by osteoradionecrosis of the jaws, a catastrophic complication* (see Osteoradionecrosis in Ch. 267). It is best to avoid extraction, if possible, by using dental restorations, dental splints, or endodontic treatment (root canal). Careful life-long attention to oral hygiene is necessary to avoid oral or periodontal surgery. Frequent fluoride applications are also indicated for an indefinite period following radiotherapy. Tissue breakdown and persistent ulceration is likely beneath a partial or complete denture because of scarring and inelasticity of irradiated tissue. The prostheses should be checked and adjusted whenever discomfort is noted.

**Extraction of a tooth adjacent to a carcinoma** of the gingiva, palate, or antrum favors invasion of the alveolus (tooth socket) by the neoplasm. Extraction should be done only in the course of definitive treatment.

**Subcutaneous and mediastinal emphysema** may rarely follow use of a high-speed air turbine dental drill or compressed air during a root-canal procedure, or to section a tooth or the alveolar bone during an extraction, as air is forced into the alveolus of the bone and then dissects along fascial planes. Acute onset of jaw and cervical swelling with characteristic crepitus on palpation of the swollen skin is diagnostic.

In **endocrine disorders**, tooth extraction is never routine. Dental treatment should usually be postponed until the systemic disease is well controlled. For example, tachycardia may occur in hypertrophied individuals, accompanying the anxiety often present in dental

patients. One exception to postponement is that poorly controlled diabetics may be more easily managed after they receive necessary dental care to improve poor oral hygiene. **Diabetics** are prone to periodontal disease and following periodontal surgery or extractions may require adjustment of insulin dosage and parenteral fluids or diet during the time when food intake is limited because of postoperative pain. Poorly controlled diabetics, often dehydrated, also have a decreased salivary flow that contributes to caries. To avoid undue interference with food intake, extensive extractions, restorative dentistry, or periodontal surgery should not be done in one visit. Patients with **adrenocortical insufficiency** may require supplemental corticosteroids during the stress of major dental procedures. Individuals who have **Cushing's syndrome** or are receiving corticosteroids for a systemic disease may have delayed wound healing and increased capillary fragility.

An **allergic** individual might, despite previous interrogation, receive an offending antibiotic, local anesthetic, or other medication given in conjunction with dental treatment.

In **Bell's palsy** the natural cleansing action of the lip and cheek on the tooth surface of the involved side is lost; without scrupulous oral hygiene and repeated fluoride treatments, unilateral decay will increase.

People with **convulsive disorders** should have fixed (not removable) small dental appliances, to prevent swallowing or aspirating them and causing airways obstruction or possibly esophagitis or enteritis due to the foreign body. Tracheoesophageal or enterocolonic fistulas may develop subsequently. **Quadriplegics** and individuals with severe upper extremity incoordination or tremor cannot have good oral hygiene unless those who care for them are very conscientious. Unexplained fever in them may have an oral basis that should be sought.

**Hepatitis B** virus rarely may be transmitted by dentists with antigenemia who are carriers of the disease to patients via open lesions on the dentist's fingers into the alveolus of an extracted tooth or into the patient's mouth and therefore the GI tract. The hazard is diminished if dentists with digital lesions, particularly with a history of hepatitis, wear rubber gloves. Unfortunately, autoclaving is not the usual means of sterilizing dental instruments, so the use of dental instruments or the probably rare reuse of hypodermic needles may infect a subsequent patient. If a patient with viral hepatitis visits a dentist during the incubation period, additional individuals may be placed at risk.

## **Dental Restorations and Appliances**

**Fillings** are inserted after removal of decay. A temporary filling is kept in place for weeks in the hope that the tooth will retain its vitality and deposit secondary dentin to seal a pulp exposure. Silicate, a type of porcelain cement, is used to fill cavities in anterior because it resembles enamel. Recently, plastic resins have been used for the same purpose. The occlusal surfaces of posterior teeth, which bear the brunt of mastication, require stronger materials. The commonest ones used, amalgam and gold inlay, can be identified by color. A small, less common filling is gold foil.

If decay is extensive, placing several fillings in one tooth might undermine its structure. To avoid fracture of the natural crown, the dentist removes the decay and fills the sites with cement. He grinds and tapers the outer surfaces so that an artificial **crown**, usually

of gold, may be placed. A porcelain jacket crown is used on anterior teeth for its natural appearance. During laryngoscopy, care must be exercised not to dislodge any artificial crowns fixed on anterior teeth.

When teeth are missing, a bridge or partial denture can be made. A **bridge** is usually smaller than a partial denture, but it is possible to make 1 or 2 bridges to cover an entire maxillary or mandibular dental arch. Stress in a bridge is largely borne by abutment teeth (usually on either side of the missing tooth or teeth). Abutment teeth have crowns cemented to them; thus, the bridge is a fixed appliance that is not easily removed. False teeth are soldered to the crowns and to each other. A **partial denture**, typically a removable appliance with clasps that snap over the abutment teeth, may be removed for cleaning. Part of the load of occlusion is borne by the soft tissues underlying artificial teeth, which often are on both sides of the jaw. This appliance is often used when there are no more natural teeth beyond the tooth or teeth to be replaced.

**Complete dentures** are removable appliances that help a patient chew solid foods and improve his speech and appearance, but they cannot achieve the efficiency or tactile sensations of natural dentition.

All **removable dental appliances** are generally removed before throat surgery, general anesthesia, or convulsive shock therapy to avoid loss, breakage, aspiration, or swallowing during the procedure. They should be stored in water to prevent dimensional changes that may occur with drying. However, some anesthesiologists believe that an appliance aids the passage of an airway tube, keeps the face in a more normal shape so the mask fits better, does not interfere with laryngoscopy, and prevents natural teeth from injuring the opposing gingiva of a completely edentulous jaw.

## Chapter 246: Examination of the Oral Region

Knowing the details and frequency of dental visits may alert the physician to a particular dental problem or to a lack of attention to dental care. **Inability to chew food well** suggests insufficient teeth for proper mastication, poorly fitting dental appliances, loose or painful teeth, or disorders affecting the TMJ or the muscles of mastication. **Slight, occasional bleeding** after brushing suggests bristle damage or mild gingivitis; **frequent, spontaneous, or profuse bleeding** may indicate severe gingivitis or a blood dyscrasia. A history of a **single, mild infection** after oral surgery does not necessarily have systemic implications; but **recurring oral and other infections** may indicate agranulocytosis, neutropenia, leukemia, immunoglobulin defects, or disorders of leukocyte function. **Root canal treatment** - a common dental procedure when decay has reached the pulp of for devitalization after trauma - occasionally is associated with osteitis at the tip of the root. **Sores, lumps, or pain** may originate from both teeth and soft tissues. **Medication history** may reveal incompatibilities and duplications. **Abnormal taste sensations** may be due to psychiatric disorders. However, a search for local causes should always be made. A bitter taste may indicate bleeding or seepage of tissue fluid from beneath poorly fitting dentures or from inflamed periodontal tissues into the normally sodium-poor oral environment. Correction of such underlying dental problems results in disappearance of the symptom.

## The Face

The face and mouth are inspected for marked asymmetry, lesions, or disproportions. An **unusual facial appearance** characterizes many head and neck syndromes of genetic or developmental origin and often occurs with atypical positioning or malformations of the pinnae or an unusual skull shape, with or without dental abnormalities. An underweight patient may lack teeth or have severe periodontal disease, caries, or poorly fitting dental appliances that interfere with chewing. Not all involuntary weight loss indicates systemic disease in such individuals.

**Slight facial asymmetry** is universal. It may be due to preferential chewing on one side causing unilateral enlargement of the masticatory muscles, differences in the contour of the dental arches, angulation of the teeth on one side compared to the other, or combinations of these. However, **marked facial asymmetry** occurs in individuals with lipodystrophy, hemiatrophy, hemihypertrophy of the face, or congenital absence of the condyle of the mandible. Awareness of the psychological trauma of facial malformation should lead to referral for possible facial surgery.

Cheek **contours** depend mainly on the posterior teeth. **Swellings** of the face may be cutaneous (i.e. neurofibromas or sebaceous cysts) or may arise from deeper tissues. If one or both cheeks appear swollen, the distention may be in the skin, in the parotid glands (which may be enlarged because of mumps, Sjögren's syndrome, or a tumor), or inside the mouth. An excessively thick denture flange gives the wearer a puffy appearance that disappears on removing the denture and can be remedied by a dentist's grinding down its outer thickness. An abscessed tooth may cause soft-tissue swelling as pus drains from the tooth toward the outer surface of the face or neck. Endodontic (root canal) treatment or extraction promotes drainage and mitigates against further spread of infection. Salivary gland and lymph node enlargement is evident on inspection of the preauricular and submandibular regions.

**Fistulas** may represent malformations of the embryologic branchial pouches or draining sinuses from abscessed teeth. An abscessed lower molar may drain below the angle of the mandible and eventually leave a depressed scar.

## Breath Odor

(see also Halitosis, Real and Imagined in Ch. 52)

The physician should wonder why a patient who is reeking from mouthwash is self-conscious about his breath. Is it only recent ingestion of alcohol, onions, or garlic, or use of tobacco, or is it for another reason? Extensive dental caries, periodontal disease, or tonsillitis causes a fetid odor often accompanied by a bad taste. Rhinitis, ozena, or sinusitis also causes halitosis. There are **systemic causes** of bad breath: in liver failure, a mousy odor is present; in uremia, a uriferous smell; and in a lung abscess or bronchiectasis, a putrid odor. In diabetic ketosis, acetone is present in the expired air.

## Lips

**Lip movements** normally betray emotion as the patient speaks; in scleroderma or Parkinson's disease, the lips are rigid. With a facial nerve paralysis (Bell's palsy), marked asymmetry occurs when the patient talks or smiles. The **vermillion border** (between the mucosa of the lips and the skin of the face) is the site of recurrent infections (cold sores) and carcinoma. **Generalized thickening** of the lips occurs in myxedema, cretinism, and acromegaly. **Localized swellings** may indicate a lymphangioma or hemangioma, the latter causing purplish discoloration as well. Besides cosmetics to camouflage the deformity, more definitive treatment is available (see Ch. 243). In the absence of anterior teeth, the lips become shorter and more concave. They are attached to the jaws by prominent midline and smaller lateral frena. Other abnormalities are discussed in Ch. 251.

## Temporomandibular Joint

The TMJ is palpated laterally and intrameatally for tenderness, range, smoothness of motion, and condylar deformity. As the patient opens his mouth, one notes any limitation or **deviation of jaw movement** indicating abnormality of the 5th cranial nerve or weakness of jaw muscles. Congenital malformation or absence of the TMJ can cause similar signs, and characteristic abnormalities appear on x-ray. Normally the jaw should move symmetrically, and with mouth open, 3 fingers should fit comfortably between the upper and lower incisors (40 to 50 mm). If less space exists, both articular and nonarticular conditions should be considered in the differential diagnosis, since they can produce similar types of dysfunction. The patient may have scleroderma, parotitis, malformation or arthritis of the TMJ, a peritonsillar abscess, or pericoronitis (infection of the gingiva about a partially erupted 3rd molar). Tetanus or a depressed fracture of the zygomatic arch impinging on the coronoid process of the mandible can also impair opening the mouth. Muscular problems (trismus, myofascial pain-dysfunction (MPD) syndrome) for which the cervical muscles and muscles of mastication should be palpated are more common causes of limited jaw movement than are joint problems (ankylosis, disk derangement). An unusually wide opening may indicate a subluxation of the mandible.

By placing his or her little fingers deeply into the patient's auditory canals, the physician can test the range and smoothness of mandibular condylar motion as the mouth opens and closes.

For complaints of ear or facial pain on chewing, the examiner can use a stethoscope in front of each ear as the patient opens and closes his mouth. Clicking or popping sounds indicate an internal disk derangement. A grating noise or crepitus suggests degenerative joint disease.

## Teeth

(see Growth and Development from Birth Through Childhood in Ch. 182)

A useful shorthand representation for recording observations is shown in Tables 246-1 and 246-2. The horizontal line represents the space between the jaws and the vertical line denotes the midline of the face. Another method of identifying teeth is using the letter A to

T for the deciduous and numbers 1 to 32 for the permanent teeth; i.e. in the permanent dentition, numbering begins with the 3rd maxillary right molar and goes to the left across the maxillary arch, then to the right across the mandibular arch to the 3rd mandibular right molar.

**Oral hygiene** reflects the patient's general attitude toward himself or his physical, psychologic, or economic ability to care for himself. Are teeth decayed or missing? Pain experienced when teeth are tapped with a tongue depressor suggests extensive dental caries or periodontal disease. Severe periodontal disease causes most instances of viable mobility of teeth, but rarely, erosion of the alveolar bone by an underlying tumor (i.e. ameloblastoma) will loosen them. A deeply carious tooth may have an infected or necrotic pulp. A dentist can test the patient's reaction of pain to a weak electrical stimulus to determine whether a tooth is alive. A tooth with decay involving the pulp is a potential source of infection into surrounding alveolar bone. **Calculus (tartar)**, if present, is deposited particularly near the orifices of the salivary ducts on the buccal surfaces of the maxillary molars and the lingual surfaces of the mandibular anterior teeth.

The commonest motor abnormality of the oral region is probably not Bell's palsy (see Ch. 131) but **bruxism** - the clenching or grinding of teeth that erodes and eventually diminishes the height of dental crowns. Such attrition is common with advancing years; in youth, it often indicates bruxism. The teeth may loosen. Although the patient may be oblivious of his habit, other family members are aware. The treatment requires that the patient consciously try to overcome the habit, perhaps with the help of sedatives or tranquilizers and abstinence from alcohol. Alcohol often aggravates bruxism. A dentist can make a splint to be worn over the teeth to prevent the grinding movements. Biofeedback may be tried.

Maxillary incisor **fractures** are common in children with neurologic disorders who fall often. Malocclusion can result in a front tooth contacting its opponent at an angle so that a corner of an incisor may be chipped.

**Defects in tooth form:** Once formed, teeth are never remodeled by systemic influences, only by local ones. Thus, the examiner may find evidence of developmental disorders or endocrinopathies. In **congenital syphilis** the contours of the incisors and 1st molars undergo characteristic changes: The incisors show a constriction at the incisal 3rd, which produces a pegged or screwdriver shape, and a characteristic notch in the central portion of the incisal edge. The 1st molar is dwarfed, with constriction of the occlusal surface and roughening and hypoplasia of the enamel. With hereditary opalescent dentin (**dentinogenesis imperfecta**), an autosomal dominant trait, the dentin is abnormally formed and teeth are a dull bluish-brown. Such teeth cannot withstand occlusal stresses and rapidly becomes worn. "**Peg**" **lateral incisors** are congenitally narrow but are unassociated with systemic disease.

**Defects in enamel or tooth color:** A **dead tooth** appears gray. Darkening of the teeth and enamel hypoplasia follow **chronic administration of tetracyclines** during the second half of pregnancy or during tooth development in the child; the affected teeth, rather than fluorescing white in ultraviolet light, have a colored fluorescence characteristic of the type of tetracycline given. A dark band may be visible in ordinary light after only 5 days of therapy. The abnormal calcium metabolism associated with **rickets** results in enamel hypoplasia. A rough irregular band appears around each tooth. The location of thus provides



an estimate of the age when the disease occurred and its duration. Such teeth are not unduly susceptible to dental caries. A **high fever** during odontogenesis can also interfere with enamel formation, and a narrow zone of chalky pitted enamel is visible after the tooth erupts. **Amelogenesis imperfecta**, an autosomal dominant hereditary disease, causes severe enamel hypoplasia. Children who drink water containing > 1 ppm of fluoride during the period of tooth development are likely to develop mottled enamel (**fluorosis**). The enamel changes can range, depending on the amount of fluoride ingested, from irregular whitish opaque areas to severe brown discoloration of the entire crown, with a roughened surface. Such teeth have a high resistance to dental caries. In **congenital porphyria**, teeth fluoresce a reddish color due to deposition of pigment in the dentin. Periapical x-rays show that hypopituitary dwarfs have small dental roots and people with gigantism have large roots. In acromegaly, one sees not only enlargement of the jaws but also hypercementosis of the roots.

The patient should be asked to remove dentures so that the underlying soft tissues may be seen. The likelihood of dropping or distorting a dental appliance is greater if the physician attempts to remove it.

With age, teeth darken and biting surfaces become worn (**attrition**) so that chewing is less effective. **Gingivae** may **recede** and thus expose more of the crowns and often even part of the roots. Since a small zone of exposed dentin is sensitive to touch or temperature alterations, noncarious teeth can become painful when enamel and cementum do not quite contact each other. The dentist can desensitize such teeth. Proper keratinization of the oral mucosa requires sex hormones. Hormonal therapy can ameliorate the poorly keratinized, inflamed mucosa commonly found after menopause.

In edentulous jaws of the aged, **atrophy of the alveolar process (senile atrophy)** is common. This diminishes the stability of artificial full dentures, particularly the lower one. Oral surgical procedures can improve the situation.

### **Normal Occlusion**

Occlusion should be checked on both sides of the mouth; this can be done by retracting each cheek with a tongue depressor while the patient bites normally. In normal occlusion, the maxillary anterior teeth overlies the mandibular anterior teeth. The outer (buccal) cusps of the maxillary posteriors are external to the corresponding cusps of the mandibular posterior teeth. Since the outer parts of all the maxillary teeth are superficial to the mandibular teeth, the lips and cheeks are displaced from between the teeth so they are not bitten. Furthermore, the lingual (inner) surfaces of the lower teeth are in a smaller arc than that of the upper teeth, confining the tongue and minimizing the likelihood of its being bitten as the teeth occlude. All the teeth should contact their opponents, so that the powerful masticatory forces (which may be > 100 lb in the molar region) will be widely distributed. If these forces are applied to only a few teeth, the latter are likely to loosen.

### **Malocclusion**

The commonest **classification** of deviation from the normal contact of the maxillary and mandibular teeth identifies 3 major forms of malocclusion: **Class I:** The upper and lower molars occlude normally, but the anterior teeth are crowded or malpositioned. **Class II:** The

lower jaw is retruded and the facial profile is convex. **Class III:** The mandible protrudes.

**Etiology:** Malocclusion often reflects a disproportion between jaw and tooth size - i.e. a jaw so small or teeth so large that the jaw cannot accommodate them all in proper alignment. Other causes are supernumerary, malformed, or missing teeth; delayed eruption or impaction of permanent teeth; ankylosis of the mandible; cleft lip or palate; and rarely, cleidocranial dysostosis or Hurler's syndrome. A frequent cause of acquired malocclusion is loss of teeth with shifting of adjacent teeth and extrusion of opposing teeth unless a bridge or partial denture is made. Premature loss of deciduous teeth in children often results in approximation of adjacent teeth causing insufficient space for eruption of the permanent successors. This shift can be prevented by a dental appliance termed a space maintainer. Less frequently, malocclusion is from habits like thumb- and finger-sucking or tongue-thrusting. Iatrogenic malocclusion can occur from improper dental restorations and appliances, improper fixation of jaw fractures, or a Milwaukee orthopedic brace, which places constant pressure on the mandible. Rarely, teeth are displaced by an underlying jaw tumor.

**Diagnosis and Treatment:** Malocclusions are corrected primarily for oral health reasons, although the patient often needs help for cosmetic or psychologic reasons. Because early interceptive **orthodontics** usually eliminates a later need for more expensive and difficult technics, a child should have a dental consultation as soon as malocclusion is suspected. The evaluation includes x-rays of the skull, facial bones, and teeth, and study casts of the teeth. Malocclusion following facial trauma may suggest tooth displacement, often accompanied by a jaw fracture.

**Therapy** increases resistance to dental decay, anterior tooth-edge fracture, and periodontal disease, and it improves speaking and mastication. Occlusion can be improved by selective grinding where teeth or restorations contact prematurely, by aligning teeth properly, or by inserting crowns or onlays to build up teeth that are below the plane of occlusion. Applying a continuous mild force to the teeth by means of orthodontic appliances (braces) moves the teeth by gradually remodelling the alveolar bone. Some patients initially require extraction of 1 or more permanent teeth to obtain enough space for stable alignment. When the final relationship has been achieved, the patient wears a plastic retainer at night until the teeth stabilize in new positions. Where it is anticipated that orthodontic treatment may not suffice, surgical correction of jaw abnormalities contributing to malocclusion (orthognathic surgery) is often indicated. Orthodontic treatment of adults is now commonplace.

### Oral Mucosa

With a finger cor for protection against infectious lesions, the cheek is palpated bimanually (with one finger inside and one outside) to delimit lesions. Most people have yellowish pinhead-size macules in the buccal mucosa. These are harmless **ectopic sebaceous glands** (Fordyce granules). Many persons have a thin white line (linea alba) on the buccal mucosa along the occlusal plane. It represents surface keratinization due to accidental, repeated cheek biting over the years.

The **color** of soft tissues can reflect anemia, polycythemia, cyanosis, or jaundice. One looks for generalized inflammation (stomatitis) as well as localized areas of inflammation, ulceration, petechiae, or thickening. Darkly pigmented areas may indicate a racial

characteristic, Addison's disease, or, very rarely, melanoma.

**Dryness** of the mouth may be from dehydration, mouth breathing, or use of diuretics, or may reflect salivary gland dysfunction or disease (see Ch. 247). The orifices of the parotid ducts open in the cheek beside the maxillary molars. The sublingual and submandibular salivary gland ducts open on the floor of the mouth behind the lower incisors. In case of pain or swelling in those regions, if saliva does not emanate from the appropriate duct, it may be obstructed by a calculus (sialolith).

The **distribution of keratinized and nonkeratinized oral mucosa** can be significant. Keratinized epithelium is on the facial aspect of the lips, the dorsum of the tongue, the gingiva around the base of the dental crowns and adjacent part of the roots of the teeth, and on the hard palate. It is less likely to be damaged by hard food particles than nonkeratinized mucosa that is mobile, as in the cheek, sides of the tongue, soft palate, and floor of the mouth. Keratinized mucosa in these areas is abnormal and a definite diagnosis must be made. White areas may be seen in the mucobuccal fold in adolescents or others who retain snuff or chewing tobacco there. Such leukoplakia are often precancerous (see Ch. 251).

### **Gingiva**

The gum should be firm and nicely contoured about and adapted to the crowns of the teeth. Pink, stippled tissue should fill the entire interdental space. Keratinized gingiva is present near the crowns. More distant gingiva is nonkeratinized, highly vascular, and continuous with the buccal mucosa. A tongue depressor should express no blood or pus from the gingiva. At the gingival margin, a dark line suggests exposure to lead or a heavy metal. Gingivitis (see in Ch. 249) is common.

### **The Palate**

The anterior portion of the hard palate is the site of the incisive papilla adjacent to the central incisors. Behind it are the rugae, firm ridges that keep food from slipping as the tongue crushes the food against them. A person with a cleft palate (see also under Musculoskeletal Defects in Ch. 187) has a very nasal voice. Normal vocal resonance and articulation involve the anterior teeth, lips, tongue, and palate, as well as the lungs, vocal cords, and pharynx. A very **high palatal vault** is seen in Marfan syndrome (see under Inherited Disorders of Connective Tissue in Ch. 197). Punctate areas of inflammation about the ducts of the numerous minor salivary glands of the palate are common, especially in pipe smokers. The boneless soft palate should rise symmetrically when the patient says "ah". The uvula at the far end of the soft palate's midline varies greatly in length among individuals.

### **Tongue and Floor of the Mouth**

As the patient touches the tip of his tongue to his soft palate, one can examine the floor of the mouth and the under surface of the tongue, where cancer often starts. The tongue, having a wide range of **movement**, should be able to twist its tip around the sides of the molar teeth. Normal tongue movement indicates good hypoglossal nerve function, but neuromuscular weakness may prevent its holding a midline position or moving rapidly. For a neurologic evaluation of taste, it is possible to use 0.1 M solution of NaCl, HCl, sucrose,

and urea. The tongue may be **enlarged** in myxedema, amyloidosis, or acromegaly, or if a rhabdomyoma is present. In edentulous individuals without complete dentures, the tongue tends to broaden.

The **papillae** may be normal or atrophied. The tongue is smooth and pale in pernicious anemia or smooth and fiery red in deficiencies of niacin or riboflavin (see Glossitis in Ch. 247). Oral cancer often occurs on the lateral surface of the tongue (see Neoplasms in Ch. 251).

### Salivary Glands

**Saliva** promotes retention of artificial dentures because of its niacin content. Thus, conditions characterized by diminished saliva flow often adversely affect the ease with which dentures may be worn. If natural teeth are present, salivary flow washes away bacteria and food debris and favors oral cleanliness. A reduction in flow facilitates development of tooth decay. This may be caused by an abnormality of the salivary glands (see in Ch. 247).

## Chapter 247: Disorders of the Lips, Mouth, and Tongue

Most diseases affecting the mucous membrane can occur anywhere in the oral mucosa, but they will be mentioned under the most frequent sites. Also see Chs. 246 and 251.

### Lips

An acute swelling of the lip may be **angioedema** (see Urticaria; Angioedema in Ch. 21). Brownish-black melanin pigmentation spots in association with GI polyposis occur in the **Peutz-Jeghers syndrome**. **Exfoliative cheilitis** is a chronic desquamation of the superficial mucosal cells; if it persists despite abstention from very hot or irritating foods and alcoholic beverages and despite changes of lipstick or dentifrices, triamcinolone acetonide ointment should be tried. A **chancre** has a serosanguineous crust (see Syphilis in Ch. 14).

Mucocele (mucous retention cyst), most commonly found on the lower lip, is due to trauma severing the excretory ducts of the accessory salivary glands and permitting the mucin-containing saliva to escape into the tissues. A soft nodule forms. If the nodule is superficial, the overlying epithelium is thinned and assumes a bluish tinge. Treatment is excision.

Tiny painful vesicles characterize two common disorders: herpes labialis ("**cold sore**"), found on the vermilion border, and recurrent aphthous ulcers ("**canker sores**"), on the inner aspects of the lips (see also Recurrent Aphthous Stomatitis and Oral Herpetic Manifestations in this chapter, below).

**Cheilosis** (angular cheilitis) is characterized by fissuring and dry scaling of the skin and vermilion surface of the lips and angles of the mouth. The condition is associated with deficiency of some of the B vitamins, particularly riboflavin and pyridoxine, and frequently accompanies other clinical signs of avitaminosis. One must also consider herpetic involvement, the split papule of syphilis, and the pseudocheilosis or wrinkling at the corners of the mouth accompanying loss of vertical dimension of the face (the distance between the

jaws). In edentulous patients, wrinkling can be diminished by inserting dentures that separate the jaws. Loss of skin elasticity with aging normally results in overlapping of the skin at the corners of the mouth, so that saliva pools there and contributes to angular inflammation. Persistent lesions should be cultured to rule out a mycotic infection, particularly candidiasis (**Perlèche**). Mixed infections respond best to a combination of antifungal, antibacterial, and anti-inflammatory agents. High doses of vitamin B complex are beneficial if blood tests indicate a deficiency.

## **Buccal Mucosa**

(See also Stomatitis, below)

**Mucosal lesions** may cause dentures to hurt or fit poorly. **Koplik's spots** are tiny grayish-white macules with red margins occurring during the late prodromal and early eruptive stages of measles. **Aspirin burn** is a painful white area of coagulated tissue caused by the local action of aspirin placed against the mucosa to relieve a toothache. Wiping off the white film reveals a reddened area. **Irritation "fibroma"**, composed of fibrous tissue, is not a true neoplasm because its growth is limited. It is usually present opposite the occlusal plane where the patient chronically bites or sucks his cheek. Treatment is excision and breaking the habit or reducing the cusps of offending teeth. **Hereditary hemorrhagic teleangiectasia** (see in Ch. 99) is characterized by localized dilated blood vessels in the oral cavity, nasal mucosa, and elsewhere. Should bleeding occur, applying local pressure and an absorbable gelatin sponge ordinarily suffices. If not, a cautery may be used. **Lichen planus** often occurs with cutaneous lesions (see Ch. 236). The mucosa is characteristically netlike and hardened, with papules or erosive areas. Violet atrophic or white hyperkeratotic variations may appear on the dorsum of the tongue. **Bullae** of short duration occur in various mucocutaneous disorders (i.e. pemphigus and erythema multiforme), including the severe Stevens-Johnson syndrome. Behçet syndrome has oral as well as ocular and genital mucosal ulcers. Herpangina, characterized by vesicles in the posterior part of the mouth, and hand-foot-and-mouth disease, with small ulcerated vesicles, are both due to coxsackieviruses (see in Enteroviral Diseases under Childhood Infections in Ch. 191).

## **Floor of the Mouth**

This area may be the site of a **cellulitis** following extraction of, or root canal treatment on, an abscessed mandibular tooth. Incision, drainage, and aggressive antibiotic therapy (i.e. two 500-mg tablets penicillin V, then 1 tablet q 6 h to a total of 40 tablets) are indicated. An extensive sublingual infection, termed **Ludwig's angina**, is very hard to palpation (see Submandibular Space Infection in Ch. 5).

An **epidermoid cyst** is an inclusion cyst in the floor of the mouth (usually in the midline) or the mucobuccal fold, which is the junction of the buccal mucosa and the alveolar mucosa. The midline of the floor of the mouth is the most common location for a **dermoid cyst**, which has a doughy feel. All cysts should be excised and carefully examined for evidence of malignancy.

## Salivary Glands

**Painless swelling** of the parotid glands is often noted in hepatic cirrhosis, in sarcoidosis, in mumps, following abdominal surgery, or associated with neoplasms or infections. The common factors may be dehydration and inattention to oral hygiene. The latter promotes the growth of large numbers of bacteria that, in the absence of sufficient salivary flow, ascend from the mouth into the duct of a gland. Another cause of a painful salivary gland is **sialolithiasis** (salivary duct stone). The submandibular glands are most commonly affected. Pain and swelling associated with eating are characteristic. Calcium phosphate stones tend to form because of the high pH and viscosity of the submandibular gland saliva, which has a high mucin content. Stones are removed by manipulation or excision.

Autoimmune sialosis is the Mikulicz-Sjögren syndrome, a unilateral or bilateral enlargement of the parotid and/or submandibular glands, and often the lacrimal glands. Occasionally painful, it is associated with xerostomia (dry mouth) due to impaired saliva formation, which is most common in older women. (See Sjögren's Syndrome in Ch. 108).

## The Palate

**Torus palatinus** is a common benign overgrowth of bone (osteoma) in the midline of the hard palate where the maxilla fuse. No treatment except reassurance is required unless a dentist wishes to cover the hard palate completely with a denture. If so, surgery is required.

A hole in the palate may be a **congenital cleft** involving either or both the hard and soft palates. Clefts of the palate or lip occur once in 700 to 800 births. The cleft may vary from involvement of the soft palate only to a complete cleft of the soft and hard palates, the alveolar process of the maxilla, and the lip. The infant should be referred to a cleft palate team (pediatrician, orthodontist, speech pathologist, plastic surgeon, and psychologist). There are several etiologies of **perforating lesions** other than the congenital cleft; these include salivary gland tumors, the gumma of tertiary syphilis, carcinoma of the palate, and rarely, TB.

In infectious mononucleosis, **petechiae** may occur at the junction of the hard and soft palate. In neutropenia or agranulocytosis, there may be **ulcerations** with little inflammation. Clumps of asymptomatic red **papules** are seen in inflammatory hyperplasia of the palate (pseudopapillomatosis). A denture may irritate such lesions.

The palate may be the site of **Wegener's granulomatosis** (lethal midline granuloma - see in Ch. 110) in which there is destruction of bone with sequestration.

## Stomatitis

*An inflammation of the mouth, often a symptom of systemic disease.* Fetid breath odor and blood-tinged saliva may accompany any ulcerative lesions of the oral mucosa.

## Etiology

Stomatitis may be caused by infection, trauma, dryness, irritants and toxic agents, hypersensitivity, or autoimmune conditions. Infectious agents include streptococci, gonococci,

fusospirochetes, *Candida albicans*, *Corynebacterium diphtheriae*, *Treponema pallidum*, *Mycobacterium tuberculosis*, and the viruses of herpes simplex, coxsackie, measles, and infectious mononucleosis. Stomatitis may be also result from avitaminosis particularly lack of the B vitamins or vitamin C (as in pellagra, sprue, pernicious anemia, or scurvy); or from iron deficiency anemia with dysphagia (**Plummer-Vinson syndrome**), agranulocytosis, or leukemia. Lichen planus, erythema multiforme, SLE, Behçet's syndrome, and pemphigus vulgaris frequently present oral mucosa signs. Mechanical trauma from cheek biting, mouth breathing, jagged teeth, orthodontic appliances, ill-fitting dentures, or nursing bottles with hard or too-long nipples may produce characteristic lesions. Xerostomia resulting from drugs, the aging process, or radiation therapy predisposes the mouth to sensitivity and infection. Generalized stomatitis may follow excessive use of alcohol, tobacco, hot foods, or spices; or sensitization to toothpaste, mouthwash, candy dyes, lipstick, and, rarely, acrylic dentures. Phenytoin, iodides, bismuth, mercury, barbiturates, lead, and many other drugs may produce stomatitis. Chemical stomatitis of occupational origin may be due to dyes, heavy metals, acid fumes, or metal or mineral dust. The latter 3 may also cause abrasion of the hard tissues. Mercury causes marked salivation.

### Symptoms and Signs

Clinical signs vary widely according to the type of stomatitis present. **Allergic stomatitis** is characterized by an intense, shiny erythema with slight swelling. Itching, dryness, or burning, often present, may be due to sensitivity to foods or to lipstick.

**Vincent's infection** (necrotizing ulcerative gingivitis - see Vincent's infection in Ch. 249) causes ulceronecrotic lesions of the interdental papillae that may extend to the marginal gingivae or produce painful ulcers of the mucous membranes.

**Thrush** (candidiasis), caused by *C. albicans*, is characterized by white, slightly raised patches resembling milk curds that, when removed, expose a hyperemic area that may bleed slightly. The infection usually begins on the tongue and buccal mucosa and may spread to the palate, gums, tonsils, pharynx, larynx, GI tract, respiratory system, and skin. The mouth usually appears dry. Thrush is common in infants; the debilitated; the immunosuppressed; individuals on long-term antibiotic, corticosteroid, and antineoplastic therapy; and those with xerostomia. (See also Systemic Candidiasis in Ch. 9.)

**Pseudomembranous** (or **membranous**) **stomatitis**, an inflammatory reaction that produces a membrane-like exudate, may be caused by chemical irritants (i.e. gold, iodides) as well as bacteria (streptococci, staphylococci, gonococci, *C. diphtheriae*). Fever, lymphadenopathy, and malaise may occur or the infection may be localized.

**Mucosal lesions accompanying systemic disease** include the mucous patches of syphilis; the strawberry, then raspberry, tongue of scarlet fever; Koplik's spots of measles; the ulcers of erythema multiforme; and the smooth, fiery red tongue and painful mouth of pellagra. Hemorrhagic lesions may occur in scurvy and disorders of platelet number and function. Unprovoked bleeding, decreased salivation, and an ammonia odor accompany uremic stomatitis.

The **mucocutaneous lymph node syndrome (Kawasaki syndrome)** affects children, causing erythema of the lips and oral mucosa. See Kawasaki Syndrome in Ch. 191.

**Acrodynia** occurs in children and is characterized by oral ulcerations, profuse salivation, and bruxism (see in Ch. 246) with loss of teeth. It is caused by a mercurial toxicity reaction.

### **Diagnosis**

Establishing the etiology may be difficult. The history may disclose a systemic disease, a dietary deficiency, or contact with irritants or allergens. Physical examination is obligatory, since it may reveal lesions of other mucous membranes, as in erythema multiforme, candidiasis, or syphilis; lesions of the skin, as in pellagra, pemphigus, lichen planus, or SLE; signs of pulmonary TB, sprue, anemia, or another contributory disease; or a general decrease in exocrine secretions.

Direct smears and cultures from the lesions may disclose a pathogen. Any diphtheria-like membrane should be so examined *promptly*. Vincent's infection usually limits itself to the gingival tissue, differentiating it from primary herpetic gingivostomatitis. Darkfield examination of scrapings from the lesions and STS are indicated in an attempt to rule out syphilis before penicillin is given. In thrush, a history of recent antibiotic therapy is common. To identify *C. albicans*, scrapings from suspect lesions should be cultured and examined microscopically in 10% potassium hydroxide hanging drop preparations or methylene blue stained smears. Blood count, bone marrow examination, gastric analysis, or other laboratory procedures may be indicated.

A solitary, undiagnosed oral lesion of > 1 wk duration that does not respond to treatment must be considered malignant until biopsy proves otherwise.

### **Treatment**

Underlying systemic disorders should be treated specifically. Oral hygiene is always necessary. Candidiasis usually responds to nystatin oral suspension 400,000 u. (4 mL) qid for 10 days as an oral rinse and then swallowed. Clotrimazole 10-mg oral lozenges 5 times a day for 14 days are effective in persistent overgrowths. When compliance is a problem, ketoconazole, one 200-mg tablet (for children, 1/4 tablet) orally once a day is effective. All antifungals should be continued for at least 3 days after signs and symptoms disappear. For oral bacterial infections, oral penicillin V is the drug of choice (see under Penicillins in Antimicrobial Chemotherapy in Ch. 3). Large, painful ulcers that prevent eating may be relieved temporarily by rinsing the mouth with 2% lidocaine viscous, 15 mL (1 tbsp) before each meal and q 3 h as needed for relief. A mouthwash of 1/2 tsp sodium bicarbonate in 250 mL (8 oz) warm water qid is soothing and cleansing. Rinsing (and spitting out) after each meal with elixir of dexamethasone 0.5 mg/5 mL (1 tsp) relieves discomfort and promotes healing of nonviral and nonbacterial oral lesions.



## **Recurrent Aphthous Stomatitis**

*Acute painful ulcers on the movable oral mucosa, occurring singly or in groups ("canker sores").* Minor ulcers, the most common form, are < 1 cm in diameter, last 10 to 14 days, and heal without scarring; major ulcers, > 1 cm in diameter, last weeks to months and heal with scarring. Recurrent attacks are common, with 2 or 3 ulcers during each attack; however, 10 to 15 ulcers are common in some individuals. Women are affected more often than men.

**Etiology** is unknown, but several factors point toward a localized immune reaction. Deficiencies of iron, vitamin B12, and folic acid increase susceptibility. Stress and local trauma are usually the predominant precipitating factors.

### **Symptoms and Signs**

Beginning as a shallow, ovoid erosion with a slightly raised, yellowish border surrounded by a narrow, red, hyperemic zone, the ulcer is covered within 5 to 7 days with a yellowish opaque material composed of coagulated tissue fluids, oral bacteria, and WBCs. The acutely painful phase lasts 3 to 4 days; symptoms then diminish until the lesions heal spontaneously, usually without scarring, in 7 to 10 days. Malaise, fever, and lymphadenopathy may accompany severe attacks. Recurrent attacks vary from one lesion 2 to 3 times/yr to an uninterrupted succession of multiple lesions.

### **Diagnosis**

The mucosal lesion looks distinctive enough to differentiate aphthous stomatitis from primary or recurrent herpetic oral lesions (which may appear concurrently) and from the lesions of erythema multiforme, oral pemphigus, or benign mucosal pemphigoid. Aphthae rarely appear on the immovable mucosa (hard palate, attached gingiva), the prime areas for recurrent intraoral herpetic ulcers. The history and clinical examination herpangina.

### **Treatment**

A topical anesthetic such as 2% lidocain viscus, 15 mL (1 tbsp) as an oral rinse q 3 h or before meals, provides short-term relief and facilitates eating. A dental protective paste (Orabase) applied qid prevents irritation of the ulcers by the teeth, dental appliances, and oral fluids. An application of triamcinolone acetonide in emollient dental paste reduces discomfort and promotes healing.

For multiple lesions, tetracycline oral suspension (250 mg qid for 10 days) is held in the mouth for 2 to 5 min before swallowing, to coat the ulcers. If treatment is started early after onset, symptomatic relief occurs within a day and new lesions are aborted. Treatment must be repeated for each new attack. Occasionally this therapy results in oral (and vaginal) candidiasis. For severe episodes of minor aphthae or for major aphthae, corticosteroid therapy is indicated, both topical and systemic (elixir of dexamethasone 0.5 mg/5 mL (1 tsp) to rinse with (and spit out) after meals and at bedtime for 5 days).

## Oral Erythema Multiforme

(See also Erythema Multiforme in Ch. 237)

*Acutely painful stomatitis characterized by diffuse hemorrhagic lesions of the lips and oral mucosa and usually associated with constitutional symptoms.* Oral, ocular, genital, and dermal lesions can occur concurrently.

### Symptoms, Signs, and Diagnosis

Prodromal symptoms may include rhinitis and sinusitis. Multiple vesicles form in the earliest stage. Typical lesions consist of diffuse hemorrhagic eroded areas throughout the mouth; the lips are commonly bloody and crusted, but the gingivae are rarely involved. Extensive oral, conjunctival, and genital lesions may be present, even without dermal eruption.

The patient may have fever as high as 40 to 40.6 °C (104 or 105 °F) during the early stages. Severe constitutional symptoms (fever, malaise, arthralgia) usually persist for 4 or 5 days; as they regress, the typical lesions develop. The constitutional symptoms may be similar to those in allergic stomatitis, pemphigus, and herpetic stomatitis, which must be differentiated. The lesions are a deeper red than the mucous patches of secondary syphilis.

### Treatment

Oral lesions in the acute phase may be treated with systemic corticosteroids (prednisone 10 mg orally tid for 5 days) or elixir of dexamethasone 0.5 mg/5 mL (1 tsp) to rinse with and swallow qid for 5 days. Without corticosteroids, the lesions may persist from 3 to 8 wk or longer. When intraoral lesions cause difficulty in eating, a liquid diet is helpful. Dehydration may necessitate IV fluid therapy. A warm mouthwash of 10% sodium bicarbonate solution and anesthetic troches, ointments, or solutions (i.e. 2% lidocaine viscous) can be used 5 or 6 times/day. Petrolatum ointment may soothe lip lesions. With treatment, improvement is rapid and lesions usually heal without scarring. Recurrence is not usual.

## Oral Herpetic Manifestations

(See also Herpes Simplex under Respiratory Viral Disease in Ch. 12)

*Acute, painful vesicular eruptions of the oral mucosa or vermilion borders, caused by the herpes simplex virus.* Primary acute herpetic infection is common in infants and young children, and may occur in teenagers and young adults (see also Herpes Simplex in Ch. 12). A history of recent contact with an adult having a herpes simplex eruption is frequent.

In childhood, the initial viral infection usually goes unrecognized unless the stomatitis causes feeding difficulties. After infection, antibody titer remains high throughout life and limits future response to the virus to an occasional recurrent lesion intraorally on the hard palate, or extraorally on the vermilion border, often extending to the skin surfaces of the lips. The latter are often called **fever blisters** or **cold sores**. Mild trauma such as that associated with dental treatment, abrasion of the vermilion border, sunburn, food allergy, anxiety, onset of menstruation, or any disease that produces a fever or an increased metabolic rate may

precipitate lesions.

## Symptoms and Signs

In **primary acute herpetic gingivostomatitis**, multiple shallow ulcers of varying size occur throughout the mouth. The oral ulcerations are preceded by inflamed gingivae resembling acute necrotizing ulcerative gingivitis and a 2- to 3-day prodromal period of malaise, fever, and cervical lymphadenopathy. During the first 4 or 5 days, pain may be severe enough to discourage a child from eating and drinking. Although the disease is usually self-limited and symptoms subside in 7 to 10 days, extensive systemic involvement and fatal viremia have occurred in infants and occasionally in older children.

In **recurrent herpes labialis**, patients usually experience a sensation of fullness, burning, and itching before the typical vesicle develops on slightly elevated, erythematous tissue at or near the junction of the vermilion and skin. The greatest extension of the lesion is usually toward the skin. A vesicular lesion may exist for hours before the vesicle breaks and a yellowish, crusted lesion forms. Underlying tissues are not indurated, though varying degrees of edema may be present. Lesions seldom last > 10 days.

Recurrent intraoral herpetic palatal and gingival lesions begin with multiple small vesicles that rupture quickly and unite to form large, superficial ulcerations with irregular margins. A large zone of erythema usually surrounds the ulcers.

## Diagnosis

**Primary acute herpetic stomatitis** must be differentiated from drug eruptions and erythema multiforme, and, more rarely in adults, from pemphigus. Allergic forms of stomatitis can usually be suspected from the history. In both erythema multiforme and pemphigus, accompanying skin lesions are common. **Erythema multiforme** (see in Ch. 237) may be discerned by more marked constitutional symptoms and widespread hemorrhagic lesions. In **pemphigus** (see also in Ch. 238), constitutional symptoms usually persist for several weeks, and the patient often recalls a prior episode of large painless bullae without accompanying prodromal symptoms.

Diagnosing the solitary lesion of **herpes labialis** is usually not difficult because of its characteristic appearance and location.

## Treatment

In **primary herpetic stomatitis**, a topical analgesic relieves temporary pain; 2% lidocaine viscous, 5 mL (1 tsp) as an oral rinse q 3 h, or diphenhydramine elixir, 5 mL (1 tsp) as an oral rinse q 2 h is used as needed. A sodium bicarbonate mouthwash of 2.5 mL (0.5 tsp) in 250 mL (8 oz) warm water qid soothes and cleanses. Since children tend to decrease fluid intake, they must be watched for dehydration. Supportive therapy consists of increasing fluids and giving diet supplements (see Table 79-5 in Ch. 79). Systemic antibiotics may be used to guard against secondary infection.

**Recurrent herpes labialis** (lip lesions) can be reduced in frequency by using a sun-screen containing amino benzoic acid during sun exposure. *All proposed treatment is more effective if started in the prodromal stage, i.e., at the first symptoms of local change in sensations.*

**Topically**, antiviral agents such as vidarabine or acyclovir ointments will discourage spreading and act as lubricants. Applying ice locally will reduce swelling. Petrolatum will tend to prevent cracking, bleeding, and self-inoculation from manual spread.

**Systemic treatment** is directed toward improving resistance to the virus. A combination of equal parts of citrus bioflavonoids and ascorbic acid tablets 400 mg tid for 3 days may abort or greatly reduce the duration of the lesions. Where frequent recurrences interfere with daily function or nutrition, acyclovir 200-mg capsules 5 times/day will give relief.

Desiccating agents such as alcohol, ether, and chloroform are thought to fractionate the virus, thereby inviting resistant and mutagenic strains. Corticosteroids can spread the virus, especially on mucosal tissue.

There is no cure for herpes simplex.

## Tongue

Infants may have **muco-cutaneous lymph node syndrome (Kawasaki syndrome** - see in Ch. 191) in which there is a bright red tongue and face, edema of the extremities, and thrombocytosis. **Ankyloglossia** (tongue-tie) may be diagnosed if the tip of the tongue cannot contact the alveolar ridge or the tips of the teeth or sweep from one corner of the mouth to the other. To increase mobility of the tongue, the lingual frenum may need cutting. Untreated tongue-tie may affect speech and interfere with mastication and passive cleansing of the teeth. A **burning sensation** is a frequent postmenopausal symptom in association with poor keratinization, or may be a symptom of diabetic neuropathy; both require treatment of the underlying endocrine disorder. In the absence of physiologic or anatomic abnormalities, depression or an anxiety state should be considered. Amitriptyline 25 mg at bedtime with a weekly increase of dosage is usually effective in 3 to 4 wk.

## Glossitis

*An acute or chronic inflammation of the tongue.*

## Etiology

Glossitis may be either a primary disease or a symptom of disease elsewhere. The many and varied causes include the following:

**Local:** Infectious agents commonly found in the mouth; mechanical trauma (jagged teeth, ill-fitting dentures, oral habits, repeated biting during convulsive seizures); primary irritants (excessive use of alcohol, tobacco, hot foods, spices); or sensitization (by toothpaste, mouthwashes, breath fresheners, candy dyes, and, rarely, plastic dentures or restorative

materials).

**Systemic:** Avitaminosis (particularly of the B group, as in pellagra), anemia (pernicious anemia, iron deficiency anemia), certain generalized skin diseases (lichen planus, erythema multiforme, aphthous lesions, Behçet's syndrome, pemphigus vulgaris, syphilis).

### Symptoms, Signs, and Diagnosis

Clinical manifestations vary widely without strong correlation between the appearance of lesions and the severity of symptoms. Reddened tip and edges of the tongue may indicate incipient pellagra, pernicious anemia, irritation from excessive smoking, or a tooth with a rough surface. In later stages of pellagra, the entire tongue is fiery red, swollen, and often ulcerated. In iron deficiency and particularly in pernicious anemia, the tongue is pale and smooth. Painful ulcers may indicate primary herpetic or aphthous lesions, pulmonary TB with positive sputum, streptococcal infection, erythema multiforme, or pemphigus vulgaris. Whitish patches suggest candidiasis, the mucous patch of syphilis, lichen planus, leukoplakia, or mouth breathing. Denuded smooth areas, if not painful, may indicate **geographic tongue** (benign migratory glossitis), or if moderately painful, anemia or pellagra; if they are very distressing and persistent, they may be the lesions of **atrophic lichen planus** (slick, glossy, or glazed tongue). **Median rhomboid glossitis**, a developmental lesion, consists of a rhomboid-shaped smooth, reddish, nodular area on the dorsal surface of the back portion of the middle third of the tongue. **Hairy tongue**, due to a profuse overgrowth of the filiform papillae, is usually asymptomatic and often follows antibiotic therapy, fever, excessive use of O<sub>2</sub> - liberating mouthwashes, or a reduction in salivary flow. Brown papillae are usually from tobacco staining or the overgrowth of chromogenic bacteria. Treatment is rectifying the underlying cause and brushing the tongue with a toothbrush.

**Severe acute glossitis** occasionally results from local infection, burns, or trauma. It may develop rapidly, producing marked tenderness or pain with swelling sufficient to cause protrusion of the tongue and the danger of airway obstruction and suffocation. Mastication, swallowing, and speaking are painful and sometimes impossible. Cervical and submandibular adenitis with evidence of systemic toxicity may be present. Immediate treatment with steroids may be indicated to reduce the edema.

Patients may complain of a painful burning tongue (**glossodynia** and **glossopyrosis**) without obvious clinical evidence of inflammation. Many patients are postmenopausal. Incipient candidiasis, anemias, diabetes mellitus, latent nutritional deficiencies, or malignancies should be excluded.

Each case of glossitis deserves study, since the tongue often mirrors disease. History may disclose an irritant, contact allergen, sensitizing drug, deficient diet, or other symptoms of disease. Other mucosal surfaces and the skin should be inspected for evidence of pellagra, erythema multiforme, syphilis, or lichen planus. Studies for an anemia, mild diabetes mellitus, sprue, and syphilis should be performed.

## Prognosis

When the cause can be determined and corrected, response is usually prompt, but may be delayed in nonspecific or chronic involvement. The patient should be reassured that persistent lesions such as median rhomboid glossitis and geographic tongue are innocuous. Aphthous lesions, erythema multiforme, and hairy tongue often recur periodically. Solitary ulcerations that do not respond to treatment after 1 wk should be biopsied.

## Treatment

**General:** Specific causative disorders are treated as indicated. Irritants and sensitizing agents are to be *avoided*. A bland or liquid diet, preferably cooled, is given. Meticulous oral hygiene is imperative.

**Local:** Oral infections call for specific therapy (see Stomatitis, above). The pain of large lesions that interfere with eating may be relieved temporarily by rinsing with an obtundent mouthwash before each meal; topical anesthetics (lidocaine 5% ointment or 10% spray; benzocaine 2% ointment; dyclonine 0.5% liquid) applied to discrete lesions also give relief and encourage eating. Occasionally, systemic analgesics (aspirin or acetaminophen 650 mg q 4 h) are required. Topical application of triamcinolone acetonide 0.1% in emollient dental paste to specific lesions (except those of viral etiology) tid or qid will relieve symptoms and may promote healing.

The patient with symptoms of painful burning but a clinically normal tongue requires special management. Tests for vitamin B12 deficiency should be conducted, especially after menopause. For therapy of B12 deficiency, see Ch. 96. After systemic causes (anemia, diabetes mellitus) have been ruled out, an emotional basis may be presumed. Reassurance (especially regarding neoplasm) and encouragement are important.

## Chapter 248: Dental Caries and Its Complications

### Tooth Decay

*A gradual pathologic disintegration and dissolution of tooth enamel and dentin, with eventual involvement of the pulp.* Except for the common cold, this is the most prevalent human disorder.

### Etiology

The interaction of 3 factors results in dental caries: a susceptible tooth surface, the proper microflora, and a suitable substrate for the microflora. Although several oral acidogenic microorganisms can initiate the carious lesion, laboratory and clinical evidence points to *Streptococcus mutans* as the primary pathogen. Its virulence stems from its ability to synthesize extracellular polysaccharide. Lactic acid, a by-product of this synthesis, contributes to tooth demineralization. Mono- and disaccharide sugars serve as the principal substrates for the process. The gummy extracellular polysaccharides increase the bulk of dental plaque and favor bacterial proliferation and attachment to the tooth surface. **Dental plaque** - a combination of these polysaccharides, salivary glycoproteins, and desquamated

mucosal cells - serves as a localized site of acid production.

Dietary carbohydrates play a significant role. The types of carbohydrates and frequency of their ingestion are more important than the amounts consumed. Frequent between-meal snacks, especially of sucrose-containing foods, enhance the carious process; sticky foods that linger are potentially more harmful than nonsticky foods.

### **Pathogenesis**

Dental caries begins on the external crown or exposed root surface of the tooth. Bacterial plaque, not food debris, causes caries. Plaque is not flushed away by the action of oral musculature or saliva. The role of saliva in preventing caries is in its buffering capacity and remineralization effect. Acid action first demineralizes enamel with its high inorganic content; proteolysis of its organic matrix follows. When the carious process reaches the dentin or begins on the root surface, the tooth becomes sensitive to temperature or osmotic changes engendered by foods or by touch. Caries spreads rapidly because of the lower mineral content of dentin and cementum. As demineralization and necrosis of the dentin progress, microorganisms may invade the dentinal tubules. Microbial products preceding the organisms in the dentinal tubules may cause inflammation of the dental pulp before destruction of the surrounding dentin is evident.

### **Symptoms and Signs**

The patient is often unaware of the presence of caries until the lesion is well advanced. Common early symptoms are sensitivity to heat and cold and discomfort after eating sugar-containing foods. A darkened area between anterior teeth or cavitation when the carious process has progressed sufficiently may be noticed. Caries is clinically diagnosed by the dentist when softened enamel or dentin is detected with a sharp instrument. Radiographically, caries appears as a radiolucent area, as do most resin filling materials and bases under metallic restorations. Consequently, radiographic diagnosis must be coupled with a visual examination.

### **Prophylaxis**

Teeth are less susceptible to caries if optimum amounts of fluoride (approximately 1 mg/day) are ingested while the teeth are developing. Fluoride combines with some of the apatite crystals in the tooth structure to form the less soluble fluoroapatite. Maximum benefit accrues when water containing 1 ppm of fluoride is consumed from birth until the permanent dentition completes eruption (age 11 to 13). Nearly 1/2 the US population still does not have access to optimally fluoridated water. There is no proof that ingesting fluoridated water or fluoride supplements during pregnancy will significantly protect a child's deciduous teeth and permanent 1st molars, although these calcify in utero.

Ingesting excessive fluoride before eruption, while the enamel is forming, may cause permanent mottling of the enamel. Once erupted, teeth cannot develop mottling when exposed to excessive fluoride. During pregnancy, the placenta acts as a barrier against marked increases in fluoride concentration and thus protects the calcifying fetal teeth against mottling.

If the water supply contains less than the optimum amount of fluoride for the local mean maximum air temperature (as prescribed by the Public Health Service Drinking Water Standards), children should take daily supplements during their tooth-forming years, by using bottled fluoridated water for drinking and cooking, or by taking a sodium fluoride tablet.

Applying fluoride compounds to erupted teeth enhances benefits from systemic fluorides in both children and adults; it is not a substitute, since the modes of action differ. Periodic applications should be supplemented by daily use of a fluoride-containing dentifrice. Daily use of a mouth rinse containing low fluoride concentrations is also effective for children as prophylaxis against caries. For highly caries-prone patients, custom trays for self-application of fluoride gel for 5 min/day are recommended.

Food particles and dental plaque should be removed from all accessible tooth surfaces at least once daily. Mechanical removal is the only effective method currently available. Proper use of a soft-bristled toothbrush removes plaque adequately from all areas except interproximal tooth surfaces and deep pits and fissures of the enamel. Interproximal surfaces, highly susceptible to dental caries, should be cleaned daily with dental floss or tape. Plaque-disclosing tablets or liquids composed of food coloring may be used to check the efficacy of plaque removal. Since fluorides are relatively less effective for preventing pit and fissure caries, sealing enamel pits and fissures with a BIS-GMA-type resin is highly effective in preventing caries and is performed increasingly by dentists. The sealed teeth should be checked annually and the sealant replaced when lost.

### **Treatment**

Although caries may be arrested, destroyed tooth structure cannot regenerate. All affected tooth structure should be removed and replaced with a restorative material. For details of filling teeth, see Dental Restorations and Appliances in Ch. 245.

### **Pulpitis**

*Inflammation of the dental pulp (containing vascular, connective, and nervous tissue) and of the adjacent periodontal tissues, resulting in **toothache**.*

### **Etiology and Pathology**

Pulpitis may result from thermal, chemical, traumatic, or bacterial inflammation of the pulp. Pulpal inflammation and infection secondary to caries is the most frequent cause. Since hard dentinal walls surround the pulp, an inflammatory reaction usually results in necrosis. The inflammation extends through the apex of the tooth and involves the periapical tissues (connective tissue of the periodontal membrane and bone).

### **Diagnosis**

In acute suppurative pulpitis, a sharp, throbbing, shooting pain may be intermittent; it is less intense in pulpitis secondary to mechanical debridement of a cavity. A distinction must be made between caries-induced pulpitis and pulpitis secondary to traumatic occlusion. Intense pain may be difficult to localize. It may be referred to the opposite mandible or



maxilla or to areas supplied by common branches of the 5th nerve. Radiographs, pulp testers, percussion, thermal tests, and palpation aid in diagnosis.

### **Treatment**

In early pulpitis, cleansing the cavity to remove food debris and softened dentin followed by packing the cavity with zinc oxide-eugenol cement (clove oil mixed with zinc oxide powder to form a thick paste), usually affords relief and prevents food debris from accumulating. Infected pulpal tissue should be removed and root canal therapy instituted, or the tooth should be extracted.

### **Periapical Abscess**

#### **(Dentoalveolar Abscess)**

*An acute or chronic suppurative process of the periapical region.*

#### **Etiology and Pathogenesis**

The abscess is secondary to an infection of the dental pulp usually due to caries. However, it may occur after trauma to the teeth or from periapical localization of organisms, usually alpha-hemolytic streptococci or staphylococci.

#### **Symptoms and Signs**

Pain is gnawing and continuous. The involved tooth is painful when percussed, and often the teeth cannot close without added discomfort. Hot or cold foods may increase the pain. If treatment is delayed, the infection may spread through adjacent tissues, causing **cellulitis**, varying degrees of facial edema, and fever. The infection may extend into osseous tissues or into the soft tissues of the floor of the mouth. Local swelling and **gingival fistulas** may develop opposite the apex of the tooth, especially with deciduous teeth. Drainage into the mouth causes a bitter taste. **Abscesses** from lower molars may drain at the angle of the jaw.

A *chronic* periapical abscess usually presents few clinical signs, since it is essentially a circumscribed area of mild infection that spreads slowly. In time, the infection may become granulomatous. As the granuloma enlarges, the lesion may progress to an epithelium-lined cavity and a **periapical cyst** results. Persistent periapical cysts and granulomas may become infected. All are radiolucent on x-ray examination. Bacteria may spread via the blood and by contiguity to involve the brain, cervical, and thoracic structures (see Ch. 245).

### **Treatment**

Extraction or root canal therapy is usually indicated. If high fever persists, antibiotics (i.e. penicillin G or V 250 to 500 mg q 6 h, or erythromycin or a tetracycline 250 mg q 6 h) should be given. Hot saline mouth rinses may encourage pointing. If the swelling becomes fluctuant, it should be drained, usually by intraoral incision. An analgesic (i.e. aspirin or acetaminophen 650 mg alone or with codeine 30 to 60 mg orally q 3 to 4 h) is usually

needed. Bed rest, a soft diet, and forced fluids may be necessary.

## Chapter 249: Periodontal Disease

### (Pyorrhea)

*Inflammation or degeneration of tissues that surround and support the teeth: gingiva, alveolar bone, periodontal ligament, and cementum.* Periodontal disease most commonly begins as gingivitis and progresses to periodontitis. If the severity of the disease is disproportionate to the amount of plaque and calculus, **systemic disease** may be present; however, in widespread periodontal disease, local factors are also present. For example, diabetes mellitus, scurvy, leukemia and other disorders of leukocyte number or function, hyperparathyroidism, and osteoporosis aggravate local factors.

### Gingivitis

*Inflammation of the gingivae, characterized by swelling, redness, change of normal contours, watery exudate, and bleeding.* Swelling deepens the crevice between the gingiva and the teeth, and gingival pockets form. Gingivitis is common, and may be acute, chronic, or recurrent.

### Etiology

The greatest single cause is poor hygiene, characterized by bacterial plaque (microbial colonies growing in carbohydrate residues tenaciously attached to the tooth surfaces). Other local factors such as malocclusion, dental calculus (calcified plaque, called tartar), food impaction, faulty dental restorations, and mouth breathing play important secondary roles.

Gingivitis is commonly noted at puberty and during pregnancy and is presumably due to endocrine factors. Gingivitis may be the first sign of a systemic disorder with lowered tissue resistance, i.e. hypovitaminosis, leukopenic disorders, allergic reaction, endocrine disturbance (i.e. diabetes mellitus), or a debilitating disease. Prolonged ingestion of phenytoin may cause enlargement of the gingiva; the use of birth control pills may increase inflammatory changes; heavy metals (i.e. lead and bismuth) may also cause gingivitis. Correcting the factors causing the gingivitis would prevent most periodontal disease.

### Symptoms, Signs, and Diagnosis

**Simple gingivitis:** The outstanding signs are a band of red, inflamed gum tissue surrounding the necks of teeth, edematous swelling of the interdental papillae, and bleeding on minimal injury. Pain is usually absent. The inflammation, sometimes acute in onset, may subside or persist.

**Gingivitis of diabetes mellitus:** Uncontrolled diabetics have an exaggerated response to gingival irritants; secondary infections and acute gingival abscesses are common. Rapid, progressive periodontal bone loss is a common radiographic finding.

**Gingivitis of pregnancy:** Mild inflammation of the gingiva may develop in pregnancy; hyperplasia, especially of the interdental papillae, is likely to occur. Pedunculated gingival growths (**pregnancy tumors**) often arise from the papillae in the first trimester, may persist throughout pregnancy, and may or may not subside after delivery. A similar lesion, **pyogenic granuloma**, is a soft, reddish mass in the interdental gingiva that develops rapidly and then remains static. Treatment is excision. A similar gingivitis may accompany dysmenorrhea.

**Desquamative gingivitis** is characterized by deep red, painful, easily bleeding gingival tissue. Vesicles may precede the stage of desquamation. Desquamative gingivitis often occurs during menopause. The gingivae are soft because the cornified cells that would resist masticatory trauma are absent. Sequential administration of estrogens and progestins is often beneficial. A similar gingival lesion may be associated with bullous pemphigoid or benign mucosal pemphigoid, which responds to corticosteroid therapy (see Ch. 238).

**Gingivitis in leukemia:** Engorged, edematous, painful, enlarged, livid gums that bleed readily suggest leukemia. They result from reduced tissue resistance, the presence of leukemic infiltrates in the periodontal tissues, and characteristic bleeding abnormalities. The gingiva may become secondarily infected with fusospirochetal organisms, resulting in necrotizing ulcerative gingivitis (see Vincent's infection, below).

**Phenytoin gingivitis:** Prolonged intake of phenytoin may cause fibrotic gingival hyperplasia. The interdental papillae enlarge initially. The process may progress until the gums are entirely involved and the teeth partially obscured. The hyperplastic tissue is firm and less prone to bleed than in other forms of gingivitis. Excision may afford temporary benefit. A folic acid supplement (3 mg/day) helps to control the hyperplasia. The drugs nifedipine and cyclosporine can produce a similar hyperplasia of the gingiva.

**Gingivitis in hypovitaminosis:** The gingiva in **scurvy** is inflamed, hyperplasia, engorged with blood, and bleeds easily. It may appear as "bags of blood". Petechial and ecchymotic areas may be on the gums and elsewhere in the mouth. Destruction of periosteum and periodontal tissue, resulting in loosened teeth, is common. Gum changes are not seen in edentulous patients. In **pellagra**, the gingiva is inflamed, bleeds easily, and is subject to secondary infection. The lips are reddened and cracked, the mouth feels scalded, the tongue is smooth and bright red, and tongue and mucosa may show ulcerations.

**Pericoronitis:** Recurrent episodes of acute inflammation of the gingival flap overlying a partially erupted tooth are common - most often around the 3rd molar; extraction may be considered after the acute process subsides. The gingival flap disappears when the tooth is fully erupted. Treatment is local aqueous irrigation. If the infection is severe, antibiotics may be required; i.e. penicillin V 500 mg orally q 6 h for 10 days.

**Gingival abscess** (parulis) develops from a periapical abscess at the tip of the root of a nonvital tooth. Pus escapes from a sinus that opens in the mucosal surface. A periodontal abscess may drain similarly.

## **Prophylaxis**

Daily removal of plaque with dental floss and a toothbrush, and routine cleaning by a dentist every 4 to 6 mo are essential preventive procedures, especially when systemic conditions predispose to gingivitis.

## **Treatment**

The treatment is to control or correct both plaque and local and systemic factors. Some cases require extensive treatment such as scaling, selective grinding of teeth to eliminate traumatic occlusion, and replacement of overhanging fillings and poorly contoured restorations. Otherwise, food trapped against the gingiva may become impacted into the gingival margin. Excision of excess gingiva is required in specific situations as noted above.

## **Vincent's Infection**

### **(Trench Mouth; Necrotizing Ulcerative Gingivitis; Fusospirochetosis)**

*A noncontagious infection, associated with a fusiform bacillus and a spirochete, that begins on the interdental papillae and can affect the marginal and attached gingiva by direct extension.* Lack of oral hygiene, physical or emotional stress, nutritional deficiencies, blood dyscrasias, debilitating diseases, insufficient rest, and heavy smoking predispose to this disease, which is seen most frequently in young adults.

## **Symptoms and Signs**

Onset, usually abrupt, may be accompanied by malaise. Without a secondary infection, there is usually no fever. The chief symptoms are acutely painful bleeding gums, salivation, and fetid breath. The ulcerations, usually limited to the marginal gingivae and interdental papillae, have a characteristic punched-out appearance, are covered by a grayish membrane, and bleed on slight pressure or irritation. Swallowing and talking may be painful. Regional lymphadenopathy is often present. Lesions on the buccal mucosa are rare but may appear as diffuse ulcerations covered with an easily removed pseudomembrane. Rarely, lesions may occur on the tonsils, pharynx, bronchi, rectum, or vagina.

## **Diagnosis**

The punched-out appearance of the interdental papillae, the interdental grayish membrane, spontaneous bleeding, and pain are pathognomonic. The presence of overwhelming numbers of fusospirochetal forms in stained smears from the lesions confirms the diagnosis. Early differentiation from diphtheria or agranulocytosis is essential when tonsillar or pharyngeal tissues are involved. The differential diagnosis must consider streptococcal or staphylococcal pharyngitis and primary herpetic stomatitis.

## **Treatment**

Gentle but thorough local debridement, oral hygiene, adequate nutrition, high fluid intake, and rest are essential. Using a soft brush and irrigating under low pressure or rinsing

the mouth with warm normal saline or 1.5% peroxide solution may be helpful for the first few days. Analgesics may be required during the first 24 h after initial debridement. The patient should avoid irritation (i.e. from smoking or from hot or spicy foods) and should have appropriate rest, good nutrition, and high fluid intake. Marked improvement usually occurs within 24 h, and then debridement can be completed. Although the acute stage responds quickly to antibiotic therapy (i.e. penicillin G or V, erythromycin, or a tetracycline, 250 mg q 6 h), antibiotics are seldom necessary and should be avoided unless high fever is present. Poor tissue topography, often produced during the acute phase, may need surgical correction to reduce the possibility of recurrence.

### **Other Gingival Disorders**

**Enlargement of the gingivae** occurs frequently during hormonal changes, i.e. pregnancy and puberty, particularly where local irritation exists, as with malocclusion or calculus. **Idiopathic fibromatosis** is characterized by diffuse enlargement of the gingivae, either smooth or nodular (see also Phenytoin Gingivitis, above). The hypertrophied tissue is often removed after the etiologic factors have been controlled.

Gingival **fibromas** often occur near sites of chronic irritation. A **giant cell epulis** which looks similar, may arise from the periodontal ligament. If the tissue contains such cells, blood chemistry should be investigated for the possibility of hyperparathyroidism.

**Denture sore mouth** is chronic inflammation due to frictional trauma of the mucosa under a poorly fitting denture. Treatment is replacement with a proper fit. Frequently this condition is accompanied by an oral candidiasis that requires concomitant treatment with nystatin oral rinses or ointment applied to the tissue surface of the denture or clotrimazole troches, 10 mg, 5 times/day. Ketoconazole, one 200-mg tablet/day, may be required.

In thrombocytopenic purpura or disorders of platelet function, gingival petechiae and **hemorrhage** may occur. Localization is predominantly at site of periodontal disease.

### **Periodontitis**

*Progression of gingivitis to the point that loss of supporting bone has begun.* It is the primary cause of tooth loss in adults.

### **Etiology**

Periodontitis results from the same local and systemic factors that cause gingivitis. The duration and severity of these factors as well as the resistance and repair potential of the patient influence the rate of osseous resorption. Faulty occlusion causing an excessive functional load on the teeth may contribute to the progress of the disease.

### **Symptoms, Signs, and Diagnosis**

The early symptoms and signs of periodontitis are similar to those of gingivitis. The gingival pockets between the gingivae and the teeth deepen, calculus deposits often enlarge, the gums lose their attachment to the teeth, and bone loss begins. The pockets collect debris

and allow microbes to proliferate, thus promoting the disease. Destruction of the supporting osseous tissue in varying degree is the earliest evidence seen on x-ray. Loosening of teeth and possible recession of the gums follow progressive bone loss; tooth migration is common in later stages. Pain is usually absent unless an acute infection (i.e. abscess formation in one or more periodontal pockets) is superimposed on the chronic process.

### **Treatment (see also Gingivitis, above)**

Systemic disorders require correction. Astringent agents, mouthwashes, and antibiotics are of little value in long-term treatment. Dental referral is indicated to correct or eliminate local irritative factors and to instruct in home care that will limit further destruction. If abnormal gum shape and pockets go uncorrected, surgery will be required. Advanced periodontitis with deep pocket formation and tooth mobility is likely to require extensive periodontal surgery. Selective grinding of tooth surfaces to eliminate traumatic occlusion, and splinting of loose teeth may be necessary. Extractions are often imperative in advanced disease.

### **Juvenile Periodontitis**

#### **(Periodontosis)**

*An uncommon widespread degeneration of the periodontal tissues with loss of alveolar bone so that teeth may be lost at an early age.* It differs from periodontitis in its lack of associated inflammation and pus formation. Typically, there is significant bone loss localized to the first molars and incisors during late childhood or the early teens. Specific microorganisms and a hereditary predisposition may be linked to the disease. By early adulthood, typical chronic inflammatory periodontitis may be superimposed on the original juvenile condition. While its cause and treatment are unknown, improving the occlusion and splinting the teeth may be of value. Periodontal surgery may stabilize the condition.

## **Chapter 250: Temporomandibular Joint (TMJ) Disorders**

The TMJ is susceptible to common congenital and developmental anomalies, fractures, dislocations, ankylosis, arthritis, and neoplastic diseases. There may also be internal derangements of the intra-articular disc. Nonarticular disorders that affect the area and can mimic true TMJ disease and the muscular disorder known as **myofascial pain-dysfunction (MPD) syndrome** (see below), which may secondarily involve the TMJ, must be considered in differential diagnosis. (See also Temporomandibular Joint in Ch. 246.)

### **Congenital and Developmental Anomalies**

**Agenesis:** Congenital absence of the condyloid process results in severe facial deformity. The coronoid process, the ramus, and parts of the mandibular body may also be absent. Abnormalities of the external, middle, and inner ear, the temporal bone, the parotid gland, the muscles of mastication, and the facial nerve are often associated. Without the condyle, the mandible deviates to the affected side, and the unaffected side is elongated and flattened. Mandibular skewing results in severe malocclusion. X-rays of the mandible and TMJ show the degree of agenesis and distinguish this condition from others that affect the

growing condyle and produce similar facial deformities but are *not* associated with severe structural loss.

**Treatment:** Jaw reconstruction by autogenous bone grafting should be initiated as soon as possible to limit progression of the facial deformity. Mentoplasty and onlay grafts of bone, cartilage, or soft tissue are also frequently used to improve facial symmetry. Orthodontic treatment helps correct malocclusion.

**Condylar hypoplasia** may be developmental in origin but usually results from local injury by trauma, infection, or irradiation during the growth period. Hypoplasia produces facial deformity characterized on the affected side by a short mandibular body, fullness of the face, and deviation of the chin. On the unaffected side, the body of the mandible is elongated and the face appears flat. Malocclusion results from the mandibular deviation. Diagnosis is based on a history of progressive facial asymmetry during the growth period, x-ray evidence of condylar deformity and antegonial notching, and, frequently, a history of trauma. **Treatment** by surgically shortening the normal side of the mandible and lengthening the affected side is usually functionally and aesthetically corrective. Presurgical orthodontic therapy helps to achieve an optimal result.

**Condylar hyperplasia**, a disorder of unknown etiology, is characterized by persistent or accelerated growth at a time when growth should be diminishing or ended. Slowly progressive unilateral enlargement of the mandible causes cross-bite malocclusion, facial asymmetry, and shifting of the midpoint of the chin to the unaffected side. The patient may appear prognathic. The lower border of the mandible is often convex on the affected side. On x-ray, the TMJ may appear normal or the condyle may be symmetrically enlarged and the mandibular neck elongated. The condition is self-limiting. Since chondroma and osteochondroma may produce similar symptoms and signs, they must be ruled out. These grow more rapidly and usually cause asymmetric condylar enlargement.

**Treatment:** Condylectomy is recommended during the period of active growth, as determined by serial cephalometric x-rays and bone scan. If growth has already stopped, orthodontics and surgical mandibular repositioning are indicated. If the height of the mandibular body is greatly increased, facial symmetry can be further improved by reducing the inferior border.

## **Trauma and Dislocation**

(See Ch. 252)

### **Ankylosis**

Ankylosis of the TMJ is most often a sequel to trauma or infection, though it may accompany RA or be congenital. Chronic, painless limitation of movement occurs. When associated with condylar growth arrest or tissue loss, facial asymmetry is usual (see **condylar hypoplasia**, above). True (intra-articular) ankylosis must be distinguished from false (extra-articular) ankylosis. The latter may be caused by such things as enlargement of the coronoid process, depressed fracture of the zygomatic arch, or scarring from surgery or irradiation. In most cases of true ankylosis, x-rays of the TMJ show loss of normal bony architecture.

**Treatment:** Forced opening of the jaws is generally ineffective because of bony fusion. Condylectomy can be used if the ankylosis is intra-articular. An ostectomy in the ramus may be needed if the coronoid process and zygomatic arch are also involved. Prolonged use of jaw-opening exercises is essential to maintain the surgical correction.

### **Internal Disk Derangement**

Internal derangement of the articular disk can be caused by chronic spasm of the lateral pterygoid muscle, trauma, or arthritic changes in the articulating surfaces. Such derangements take 2 forms: anterior disk displacement with reduction during function, which is accompanied by clicking or popping sounds on opening the mouth, and anterior disk displacement without reduction, which is characterized by painful limitation of jaw movement. This clinical condition is confirmed by arthrography or CT scan.

### **Treatment**

Nonsurgical treatment is effective in approximately 25% of the patients who have anterior disk displacement with reduction. It employs jaw repositioning appliances, nonsteroidal anti-inflammatory drugs, and, when there is lateral pterygoid spasm, muscle relaxants. For patients with persistent pain and clicking unresponsive to such modalities, the disk can be repositioned surgically.

For anterior displacement of the disk that is not self-reducing, surgical correction is indicated. If the disk is in satisfactory condition, it is merely repositioned. Otherwise, it is removed (meniscectomy) and replaced with an alloplastic implant.

### **Arthritis**

**(See also Ch. 108)**

Most forms of arthritis can involve the TMJ; the most common ones are infectious, traumatic, rheumatoid, and degenerative (osteoarthritis).

**Infectious arthritis** may be part of a generalized systemic disease, may arise from direct extension of adjacent infection, or may result from localization of blood-borne organisms from a distant infection. Inflammation and limited jaw movement are present. Early x-rays are negative but bone destruction becomes evident later. Local signs of inflammation, associated with evidence of a systemic disease or an adjacent infection, suggest the diagnosis. In suppurative arthritis, joint aspiration may confirm the diagnosis and identify the causative organism.

**Treatment** includes antibiotics, proper hydration, control of pain, and restriction of motion. Suppurative infections should be aspirated or incised. Once the infection is controlled, jaw-opening exercises are important to prevent scarring and limitation of function.

**Traumatic arthritis** may be caused by acute injury or excessive opening - i.e. during yawning, tooth extraction, or endotracheal intubation. Pain, tenderness, and limitation of motion occur. X-rays are negative except for occasional widening of the joint space due to



intra-articular edema or hemorrhage. The diagnosis includes a history of trauma and x-rays negative for fracture. **Treatment** includes analgesics, heat application, a soft diet, and restriction of jaw movement.

In **RA**, the TMJ is involved in > 50% of cases in both adults and children. Pain, swelling, and limited movement are the most common findings. In children, destruction of the condyle results in growth disturbance and facial deformity. Ankylosis may follow in all patients. X-rays of the TMJ are usually negative in early stages, but bone destruction is seen later and may result in an anterior open-bite deformity. The **diagnosis** is suggested by TMJ inflammation associated with polyarthritis and is confirmed by positive laboratory findings.

**Treatment** is similar to that of RA of other joints. In the acute stage, anti-inflammatory drugs are given and jaw function should be limited. When symptoms subside, mild jaw exercises help prevent excessive loss of motion. Surgical correction is necessary if ankylosis develops, but should not be undertaken until the condition is quiescent.

**Primary degenerative arthritis (osteoarthritis)** may involve the TMJ as well as other joints and usually occurs in persons over age 50. Relatively asymptomatic, patients complain only occasionally of stiffness, crepitation, or mild pain. Joint involvement is generally bilateral. X-rays may show flattening and lipping of the condyle. **Treatment** is symptomatic.

**Secondary degenerative arthritis** usually occurs in persons aged 20 to 40 after trauma or persistent MPD syndrome (see below), and is characterized by limitation of opening and unilateral pain on motion, joint tenderness, and crepitation. When associated with MPD syndrome, the symptoms intermittently become more severe and some muscles of mastication are tender. X-rays generally show condylar flattening, lipping, spurring, or erosion. The unilateral joint involvement helps to distinguish secondary from primary degenerative arthritis. **Treatment** is conservative, as for MPD syndrome, though arthroplasty or high condylectomy may be necessary. Intra-articular injection of corticosteroids or of 2% lidocaine may bring symptomatic relief but may harm the joint if repeated often.

### **Nonarticular Conditions Mimicking TMJ Disorders**

Conditions unrelated to the TMJ that can produce preauricular pain, limitation of jaw movement, or a combination of both include pulpitis, pericoronitis, otitis, parotitis, trigeminal neuralgia, atypical (vascular) neuralgia, temporal arteritis, nasopharyngeal carcinoma, myositis and myositis ossificans, tetanus, scleroderma, depressed fracture of the zygomatic arch, and osteochondroma of the coronoid process. Pain due to these, unlike intrinsic TMJ disorders, is usually not exacerbated by finger pressure on the TMJ as the patient opens the mouth.

### **Myofascial Pain-Dysfunction (MPD) Syndrome**

The most common disorder involving the TMJ area, MPD syndrome occurs in women more frequently than in men. Most cases are psychophysiologic in origin and result from tension-relieving jaw-clenching or tooth-grinding habits, or a centrally generated increase in masticatory muscle tonus in response to stress. The ensuing muscle fatigue in turn induces spasm of the masticatory muscles, the immediate cause of MPD. Poorly aligned teeth or ill-fitting dentures occasionally contribute to the condition. Secondary degenerative arthritis may

involve the joint in the late stages.

**Diagnosis** is based on clinical findings. Characteristically, the patient complains of unilateral, dull, aching preauricular pain that radiates to the temporal region, the angle of the jaw, and the occiput; tenderness in one or more of the muscles of mastication; jaw limitation; and occasional "clicking" or "popping" sounds in the joint. Joint pain on awakening may indicate bruxism during sleep. X-rays are usually normal, although secondary degenerative changes are seen occasionally in very late stages. Degenerative arthritis, which causes similar symptoms, must be excluded. Primary degenerative arthritis is usually bilateral. Both primary and secondary degenerative arthritis usually produce x-ray changes in the joint, and secondary changes in the joint, and secondary degenerative arthritis causes tenderness of the TMJ on lateral or intrameatal palpation; these findings help to distinguish the arthritides from MPD syndrome. However, secondary degenerative arthritis can be a sequel to MPD syndrome and therefore both conditions can be present simultaneously.

**Treatment** includes a soft, non-chewy diet; limited use of the jaw; hot, moist applications or diathermy; diazepam 2 to 5 mg qid (the last dose taken at bedtime) as a muscle relaxant and tranquilizer; analgesics (i.e. aspirin 650 mg qid) for pain; and use of a biteplate. Clenching or grinding the teeth should be avoided. Possible causative life stresses should be discussed with the patient. Psychological counseling may be helpful in persistent cases.

## Chapter 251: Preneoplastic and Neoplastic Lesions

### Oral Preneoplastic and Early Lesions

Approximately 20,000 new cases of oral cancer (mainly squamous) are reported each year in the USA and account for about 5% of cancers in men and 2% in women. More than any other factor, the stage of these cancers determines the prognosis (see Staging and Prognosis in Ch. 212). While oral cancers < 1 cm in diameter are easily cured, most lesions are *not* diagnosed before stage III or IV and  $\geq 50\%$  have metastasized to lymph nodes. Therefore, 5-yr survival rates remain at 30 to 40%. This unfortunate situation appears to result from inadequate knowledge of appropriate screening procedures and strongly entrenched misconceptions.

**Persons at risk:** While screening is easy enough to include all patients, careful attention to patients with clearly defined **risk factors** is mandatory; i.e. individuals  $\geq 40$  yr old, those who smoke  $\geq 1$  pack of cigarettes/day, those who use smokeless ("chewing") tobacco, and those who drink alcoholic beverages regularly. Screening these individuals has been reported to increase discovery rates of early cancer in these high risk populations to 1/200 to 1/250.

**Sites at risk:** Next, it should be recognized that 90% of oral cancers are detected in only a few "high risk" sites: the floor of the mouth, the ventrolateral aspect of the tongue, and the soft palate complex (uvula, soft palate proper, anterior pillar, and lingual aspects of the retromolar trigone). Buccal carcinoma should be considered in people who smoke cigars or pipe or use smokeless tobacco.

**Misconceptions:** Most physicians and dentists have been taught that **leukoplakia** (white lesions) are the most common precancerous lesions in the mouth and that early cancers are white lesions. In fact, < 5% of such lesions ultimately prove to be cancerous. Early, asymptomatic oral cancer appears most often as a **red (erythroplastic) lesion**. These lesions are not *precancerous*; they are early carcinomas. These areas look like an inflammation, probably as the result of a submucosal round-cell infiltrate that has arisen below the malignant squamous cells in response to the developing neoplasm. When dry, these red lesions appear more granular or slightly abraded. Therefore, they should be dried gently with a piece of gauze and examined carefully under good light. Two distinct types of erythroplastic lesions have been identified: (1) a red, granular lesion (appearing like worn velvet) speckled with islands of keratin (white) or normal mucosa within or peripheral to the red component, and (2) a smooth, nongranular, red lesion with minimal associated keratin, similar to a nonspecific inflammation. Both types may have irregular, ill-defined borders; generally, palpation is not helpful diagnostically, as few are indurated or raised. *Any erythroplastic lesion that does not respond to treatment and persists > 14 days should be considered carcinoma in situ or invasive carcinoma and requires biopsy.* Unfortunately, **squamous cell carcinoma** is not usually diagnosed in its earliest stages and later appears as a deep ulcer, with smooth, indurated, rolled margins, fixed to deeper tissues.

**Other malignancies of the oral cavity** are epidermoid carcinoma of the lip, cheek, and tongue; lymphoepithelioma; melanoma; and myelocytic and lymphocytic leukemias. **Benign lesions that may be confused with oral cancer include** irritation, fibroma, papilloma, granuloma (including pregnancy tumor), the glossitis of avitaminosis, geographic tongue, median rhomboid glossitis, hemangioma and lymphangioma, fibrous hypertrophy of the gingiva, melanosis, myoblastoma, retention cysts (including ranula), xanthomatosis, torus palatinus, submandibular duct calculus, hypertrophy of the foliate papillae, radiculodental cysts, and ameloblastoma. Syphilis, erosive lichen planus, benign ulcer, TB, leukoplakia, and dental abscess should also be considered. Exfoliative cytology is useful in screening, but biopsy is essential to establish the diagnosis.

**Multiple carcinomas:** *Patients with an oral cancer are at high risk (up to 33%) of developing a second primary neoplasm in the mouth, pharynx, larynx, esophagus, or lung.* Therefore, patients identified as having an oral cancer should be screened for cancer in all these sites and reexamined at yearly intervals (i.e. examination of mouth and throat, indirect laryngoscopy, chest x-ray).

## Treatment

Any recognizable irritation (i.e. faulty restorations and prosthetic devices) should be corrected or removed. Tobacco in any form should be eliminated. Mucosal drying agents such as alcoholic beverages and mouth rinses with alcoholic vehicles should be discontinued.

Treatment of oral neoplasms generally consists of wide local excision for small lesions and en bloc excision of larger lesions in continuity with radical neck dissection if lymph nodes are involved. Radiotherapy alone may be appropriate for certain small or large lesions or may be combined with surgery. Chemotherapy may be used as palliation or as an adjunct to surgery and radiotherapy.

## Neoplasms of Specific Tissues

**Lips, gingiva, and tongue: Smoker's patch** is a firm, brownish, keratotic plaque on the vermilion border of the lower lip and is most common in smokers who hold a cigarette or pipe in one location. *Only a biopsy can rule out squamous cell carcinoma.* Cessation of smoking and careful observation are recommended even if the lesion is not malignant. **Actinic cheilosis** occurs in adults, especially redheads with fair skin, who spend much time out of doors. The lips are dry with many erosive areas. A person with this *precancerous* lesion should be seen every 3 to 6 mo, avoid prolonged exposure to sunlight, wear a broad-brimmed hat or cap, and use an antiactinic cream. **Squamous cell carcinoma** usually appear on the vermilion border of the lower lip as a nonhealing ulcer with a convex, indurated margin, or less commonly, as a keratotic patch. It may be fixed to the underlying tissues. If treated early, prognosis is excellent. In **leukemia**, the gingival tissue may be infiltrated and prone to bleed. **Rhabdomyoma** of the tongue causes a palpable interior mass. It is much rarer than squamous cell carcinoma, which arises in the mucosa.

**Cheek:** Irritation of the mucosa is commonly seen in the mucobuccal fold, when chewing tobacco or snuff may be habitually retained. The irritation may progress to erythroplakia or leukoplakia and squamous cell carcinoma. A firm, nodular, nonpainful swelling in the cheek covered by normal-appearing mucosa is rather common. It is usually the result of a cheek bite and is an "**irritation fibroma**". This is considered benign.

**Palate: Accessory salivary gland tumor** is usually a mixed tumor with both epithelial and mesenchymal components, an adenoid cystic carcinoma, or a mucoepidermoid carcinoma. Typically, it appears as a firm, smooth, painless mass lateral to the midline. Any such swelling that is not bony hard should be considered a salivary gland tumor until a biopsy proves otherwise. A **fullness of the palate** can represent extension of a malignant tumor of the lining of the nose or the antrum rather than a primary lesion of the palate. The soft palate may become immobile if a cancer is in the nasopharynx.

**Jaws:** If not initially detected on x-ray, jaw tumors are diagnosed clinically because their growth causes **swelling** of the face, palate, or alveolar process (the area of the jaw surrounding the teeth). They cause bone tenderness and severe pain originating in the involved bone. **Ameloblastoma**, the most common odontogenic neoplasm, most frequently arises in the posterior mandible and is slowly invasive, but rarely metastatic. On x-ray, it typically appears as a multiloculated or soap-bubble radiolucency. **Odontomas** are tumors of the dental follicle or the dental tissues that usually appear in the mandibles of young people; several types include fibrous odontomas and cementinomas. An absent molar tooth suggests a composite odontoma. **Other neoplasms** include osteogenic sarcoma, giant cell tumor, Ewing's tumor, multiple myeloma, and metastatic tumors.

**Salivary glands:** The two main types of tumors are the **mixed tumor (pleomorphic adenoma)**, 60% of which occur in the parotid glands, and **mucoepidermoid carcinoma**. These tumors occur not only in major salivary glands but about 20% are found in accessory salivary glands located mainly in the palate and the buccal mucosa (see above). One of 6 tumors in the parotid gland, 1/3 of those in the submandibular gland, almost 1/2 of all palatal tumors, and nearly all sublingual gland tumors are malignant.

Slowly developing parotid swellings may be painless and the patient complains because of change in appearance; but acute swelling of the parotid gland is painful because of dense fascia surrounding it. A parotid tumor causes facial paralysis if it compresses or infiltrates the facial nerve, which may also be damaged inadvertently during surgery to remove the tumor. Tumors of the submandibular salivary glands are often painful because of close association with the lingual branch of the trigeminal nerve.

## Chapter 252: Dental Emergencies

Emergency dental treatment by a physician is sometimes required when dentists are unavailable, but dental consultation is desirable as soon as possible.

### Toothache and Infection

Pain localized to a particular tooth and provoked by sweets or cold is usually caused by **caries** that not yet involve the dental pulp (nerve). Because this type of pain is usually fleeting, the patient should avoid provoking stimuli, use mild analgesics, and seek prompt treatment.

Localized pain that is usually intensified by heat most commonly denotes caries that has reached the dental pulp (see Pulpitis in Ch. 248). Associated **periapical inflammation**, often present, may be diagnosed by tenderness to percussion. (If all maxillary posterior teeth on one side are sensitive to percussion, maxillary sinusitis should be suspected.) Initial treatment with an analgesic (i.e. acetaminophen 325 mg with codeine 30 mg orally q 4 h) and an antibiotic (i.e. erythromycin, penicillin V, or a cephalosporin, 250 mg q 6 h) may be indicated until dental therapy can be initiated.

**Periapical infection**, often accompanied by swelling of contiguous soft tissues, will usually develop from untreated pulpitis. Emergency treatment consists of analgesics and antibiotics, as described above in periapical inflammation. A **periapical abscess** that has spread beyond the alveolar bone and is causing swelling and fluctuation in adjacent soft tissue requires incision and drainage; antibiotics alone are inadequate. An intraoral incision is usually appropriate, but a percutaneous incision may be necessary for dependent drainage. To aid in selecting the appropriate antibiotics, a specimen should be cultured. (See also Periapical Abscess in Ch. 248). **Erupting or impacted molar teeth**, particularly 3rd molars, can be painful and may cause adjacent soft tissue inflammation that can progress to serious infection; erythromycin, penicillin V, or a cephalosporin, 250 to 500 mg orally qid, should be started. **Less common causes of acute perioral swelling** include periodontal abscess, infected cysts, antritis, allergy, salivary gland obstruction or infection, peritonsillar infection, or skin infection.

### Postextraction Problems

**Swelling** is normal after intraoral surgical procedures and is somewhat proportional to the degree of manipulation and trauma. If it does not begin to subside by the 3rd postoperative day, infection is likely and an antibiotic (i.e. erythromycin, penicillin V, or a cephalosporin, 250 to 500 mg orally qid) should be started.

**Postoperative pain**, usually moderate, can be controlled with acetaminophen or aspirin 325 mg with codeine 30 mg orally q 4 h.

**Postextraction alveolitis (dry socket)** is usually peculiar to the removal of mandibular posterior teeth. Typically, the pain begins on the 2nd or 3rd postoperative day, is referred to the ear, and lasts from a few days to many weeks. Alveolitis is best treated with topical analgesic medication; 1/4-in. gauze saturated in eugenol and/or guaiacol, placed in the socket and changed daily, usually reduces the need for prolonged systemic analgesics. Infection is uncommon. Rarely, **osteomyelitis** may be confused with alveolitis, but osteomyelitis is characterized by fever, local swelling, and later x-ray changes. If osteomyelitis is suspected, an antibiotic such as a cephalosporin or erythromycin should be instituted and the patient referred for definitive care.

**Postextraction bleeding** usually oozes from small vessels. After removing any superfluous clot with gauze, a pressure dressing (cotton wrapped in gauze, or a tea bag) is applied directly and continuously to the extraction site for 20 to 30 min. This is repeated 2 or 3 times. If the bleeding continues, the site may be anesthetized with 2% lidocaine with 1:100,000 epinephrine by nerve block or infiltration as appropriate, and the socket are sutured under tension, allowing space between sutures for packing if necessary. Local hemostatic agents such as oxidized cellulose or topical thrombin on a gelatin sponge or microfibrillar collagen may be placed in the socket. If these measures fail, a systemic cause should be sought. Rarely, blood loss may require transfusion.

### **Fractured and Avulsed Teeth**

If a small portion of a crown is fractured and the dental pulp is not exposed, analgesic medication (i.e. acetaminophen 650 mg q 4 h) and a topical covering with zinc-eugenol or similar cement are appropriate. If the pulp is exposed or the tooth is mobile, erythromycin, penicillin V, or a cephalosporin 250 mg q 6 h should also be given. Partially avulsed teeth should be repositioned and stabilized. With any partially or completely avulsed tooth, antibiotic therapy should be prescribed for several days.

Permanent retention of a completely avulsed tooth may be possible *if it is immediately replaced into the socket without washing and with minimal handling*. If replacement is delayed a few minutes or if the tooth is washed or handled excessively, it may be retained for a few months to a few years, but the prognosis for indefinite retention is poor. Root resorption usually occurs after delayed replacement. Thus, the patient should be instructed to replace the avulsed tooth immediately and seek professional care to stabilize it. If this is not possible, the tooth should be kept moist in saline or in milk and then, if indicated, replaced and stabilized.

### **Fractures of the Jaw and Contiguous Structures**

Fractures of the jaws and contiguous structures are diagnosed primarily by physical examination and x-ray. Fracture should be suspected if there is malocclusion, mobility of the maxillae, discrepancy in the smooth contour of the orbital rims, diplopia, infraorbital anesthesia, tenderness to palpation (particularly over the condyle or condylar neck of the mandible), and restriction or deviation when opening the mouth. A facial fracture is an

emergency if there is airway obstruction, uncontrollable hemorrhage, or trauma to the eye or CNS. Routine x-rays usually confirm the diagnosis for fractures of the mandible, but CT scanning may be helpful for midface fractures. *Fracture of a cervical vertebra should be considered when a blow has been sufficient to fracture facial bones.* An oral-maxillofacial surgeon should be consulted for a fractured jaw; otherwise, unrecognized malocclusions may result.

**Treatment:** Manually holding the mandible in a protruded position or inserting an orotracheal or oropharyngeal airway may be necessary temporarily to maintain an airway (see Ch. 33). If there is hemorrhage into the oropharynx, an orotracheal airway should be placed or the patient positioned to allow the oropharynx to drain dependently. Until definitive care is available, the jaws usually can be temporarily stabilized and hemorrhage minimized by use of a Barton bandage. If a jaw fracture can be treated within the first few hours after injury, closure of lip lacerations is best delayed until the fracture has been reduced.

**Fractures through a tooth socket** are compound fractures, usually requiring antibiotic prophylaxis (i.e. with penicillin V, erythromycin, or a cephalosporin) 250 to 500 mg orally in liquid form q 6 h).

**Fractures of the mandibular condyle** are usually characterized by preauricular pain, swelling, and limitation of opening. With a unilateral fracture, the jaw deviates to the affected side on attempted opening. Bilateral fracture can produce an anteriorly open bite. Posterior-anterior and Towne's views of the mandible usually show the fracture on x-ray. **Treatment** is usually intermaxillary fixation for varying periods, though severely displaced, bilaterally fractured condyles may require open reduction and fixation. Jaw opening exercises are usually used to aid function after fixation is discontinued. In children, some abnormal growth may ensue.

### **Dislocated Mandible**

A dislocated mandible will be fixed in a markedly open position with only the most posterior teeth contacting. If the midline is deviated, the dislocation is unilateral rather than bilateral. Injecting a local anesthetic agent (i.e. 1% lidocaine 2 to 5 mL) in the joint area and in the area of insertion of the lateral pterygoid muscle may allow the mandible to reduce spontaneously.

Alternatively, or in addition, manual reduction may be necessary. Premedication with diazepam (5 mg IV) and a narcotic (i.e. for the average healthy adult, meperidine 25 mg IV or 50 mg IM) is desirable. The patient should be well below the operator and the patient's head should be stabilized to obtain leverage; the operator places his thumbs on the external oblique line of the mandible (lateral to the 3rd molar area) and his fingers under the tip of the chin. A rotary motion is used, with the thumbs pressing inferiorly and anteriorly and the fingers pressing superiorly, until the mandible is reseat.

The jaw should be stabilized with a Barton bandage to maintain the mandible in the reduced position. The patient should avoid wide opening of the mouth for at least 6 wk. When the patient feels a yawn coming, he should place his fist under his chin to prevent wide opening of the mouth. If this is not the first such dislocation, an oral-maxillofacial surgeon

should be consulted.