Evaluation of the Dental Patient



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The first routine dental examination should take place by age 1 yr or when the first tooth erupts. Subsequent evaluations should take place at 6-mo intervals or whenever symptoms develop. Examination of the mouth is part of every general physical examination. Oral findings in many systemic diseases are unique, sometimes pathognomonic, and may be the first sign of disease. Oral cancer may be detected at an early stage.

History

Important dental symptoms include bleeding, pain, malocclusion, new growths, numbness or paresthesias, and chewing problems; prolonged dental symptoms may decrease oral intake, leading to weight loss. General information includes use of alcohol or tobacco (both major risk factors for head and neck cancer) and systemic symptoms, such as fever and weight loss.

Table 2

Symptom	Causes
Bleeding or pain with brushing (common)	Acute necrotizing ulcerative gingivitis (rare) Bleeding diathesis* Gingivitis (most common) Leukemia*
Ear pain, referred (fairly common)	Inflamed gingival flap around a partly erupted mandibular 3rd molar (pericoronitis) Localized osteitis (dry socket) after lower molar extraction
Face, head, or neck pain (uncommon, except with poorly fitting dental appliances or temporomandibular disorders)	Eagle's syndrome [†] Infection Malocclusion Occult lesions with low- grade anaerobic infections spreading to the bone Poorly fitting dental appliances Spasm of the masticatory muscles Temporomandibular disorders

Facial numbness or paresthesias Antrum or nasopharynx

tumor

Brain stem tumors

(uncommon, except with

stroke)

Extraction of a mandibular molar causing damage to the inferior alveolar nerve[‡]

Multiple sclerosis

Oral tumor (rare)

Stroke

Viral infection

Masticatory fatigue (rare, except Congenital muscular or with poorly fitting dentures)

neuromuscular disorder (in younger people)

Myasthenia gravis (a cardinal symptom)

Poorly occluding artificial dentures (in older people)

Masticatory pain or jaw claudication (rare)

Giant cell (temporal)

arteritis

Polymyalgia rheumatica

Weight loss (fairly common)

Poorly fitting dental appliances

Stomatitis

Temporomandibular

disorder

Too loose, too few, or painful teeth

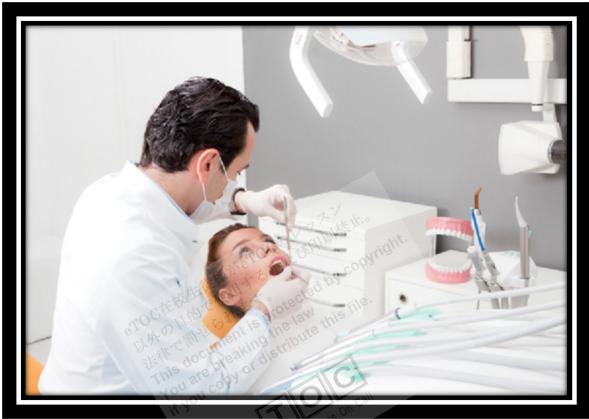
Physical examination

^{*}May be indicated by easily induced gingival hemorrhaging.

[†]Elongation of the styloid process or ossification of the stylohyoid ligament, causing pain when the head is turned.

^{*}May cause paresthesia of the lower lip.

A thorough inspection requires good illumination, a tongue blade, gloves, and a gauze pad. Complete or partial dentures are removed so that underlying soft tissues can be seen.



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Most physicians use a head-mounted light. However, because the light cannot be precisely aligned on the axis of vision, it is difficult to avoid shadowing in narrow areas. Better illumination results with a head-mounted convex mirror; the physician looks through a hole in the center of the mirror, so the illumination is always on-axis. The head mirror reflects light from a source (any incandescent light) placed behind the patient and slightly to one side and requires practice to use effectively.

The examiner initially looks at the face for asymmetry, masses, and skin lesions. Slight facial asymmetry is universal, but more marked asymmetry may indicate an underlying disorder, either congenital or acquired.

Table 3

Some Disorders of the Oral Region by Predominant Site of Involvement

Site	Disorder or Lesion	Description
Lips	Actinic atrophy	Thin atrophic mucosa with erosive areas; predisposes to neoplasia
	Angioedema	Acute swelling
	Angular cheilitis (cheilosis)	Fissuring at corners of mouth, often with maceration
	Cheilitis glandularis	Enlarged, nodular labial glands with inflamed, dilated secretory ducts; sometimes everted, hypertrophic lips
	Cheilitis granulomatosa	Diffusely swollen lips, primarily the lower
	Erythema multiforme	Multiple bullae that rupture quickly, leaving hemorrhagic ulcers; includes Stevens-Johnson syndrome
	Exfoliative cheilitis	Chronic desquamation of superficial mucosal cells
	Keratoacanthoma	A benign, locally destructive epithelial tumor resembling squamous cell carcinoma; regresses spontaneously in about 6 mo
	Peutz-Jeahers	Brownish black melanin

	syndrome	spots, with GI polyposis
	Secondary herpes simplex (cold sore)	Short-lived (< 10 days) vesicle followed by small painful ulcer at the vermillion border (common)
	Verruca vulgaris (wart)	Pebbly surface
Buccal mucosa	Aspirin	Painful white area; when wiped off, exposes an inflamed area
	burn	OCO V 3 X Paright.
	Fordyce's granules	Cream-colored macules about 1 mm in diameter; benign; aberrant sebaceous glands
	Hand-foot-and- mouth disease	Small ulcerated vesicles; coxsackievirus strain infection in young children; mild
	Herpangina	Vesicles in posterior of mouth
	Irritation fibroma	Smooth-surfaced, dome- shaped, sessile
	Koplik's spots	Tiny, grayish white macules with red margins near orifice of parotid duct; measles precursor
	Linea alba	Thin white line, typically bilateral, on the level of the

		occlusal plane; benign
	Smokeless tobacco lesion	White or gray corrugated; usually behind lower lip; tends toward cancer
	Verrucous carcinoma	Slow-growing, exophytic, usually well differentiated; at site of snuff application; metastasis unusual, occurs late
	White sponge nevus	Thick white folds over most of buccal mucosa except gingivae; benign
Palate	Infectious mononucleosis	Petechiae at junction of hard and soft palate
	Kaposi's sarcoma	Red to purple painless macules progressing to painful papules
	Necrotizing sialometaplasia	Large, rapidly developing ulcer, often painless; appears grossly malignant; heals spontaneously in 1–3 mo
	Papillary inflammatory hyperplasia	Red, spongy tissue, succeeded by fibrous tissue folds; velvety texture; benign; occurs under poorly fitting dentures
	Pipe smoker's palate (nicotine stomatitis)	Red punctate areas, are ducts of minor salivary glands, appearance is red spots surrounded by (often

		severe, usually benign) leukoplakia
	Secondary herpes simplex	Small papules quickly coalescing into series of ulcers (uncommon)
	Torus palatinus	Overgrowth of bone in midline; benign
	Wegener's granulomatosis	Lethal midline granuloma, with bone destruction, sequestration, and perforation
Tongue and floor of mouth	Ankyloglossia	Tongue unable to protrude; speech difficulty
	Benign lymphoepithelial cyst	Yellowish nodule on ventral part of tongue or anterior floor of mouth
	Benign migratory glossitis (geographic tongue, erythema migrans)	Changing patterns of hyperkeratosis and erythema on dorsum and edges; desquamated filiform papillae in irregular circinate pattern, often with an inflamed center and a white or yellow border
	Dermoid cyst	Swelling in floor of mouth
	Enlargement of tongue (macroglossia)	Localized or generalized depending on how many teeth are missing; adjacent teeth may indent tongue;

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	posterior enlargement associated with obstructive sleep apnea and snoring
Fissured (scrotal) tongue	Deep furrows in lateral and dorsal areas
Glossitis	Red, painful tongue; often secondary to another condition, allergic, or idiopathic
Hairy tongue	Dark, elongated filiform papillae
Linea alba	Thin white line on edges of tongue, usually bilateral
Lingual thyroid nodule	Smooth-surfaced nodular mass of thyroid tissue follicles, on the far posterior dorsum of tongue, usually at the midline
Ludwig's angina	Painful, tender swelling under the tongue; can compromise the airway by forcing the tongue superiorly and posteriorly
Median rhomboid glossitis	Red (usually) patch in midline of tongue, without papillae; asymptomatic
Neurilemoma	Persistent swelling, sometimes at site of prior trauma; can be painful
Pernicious anemia	Smooth, pale tongue, often with alossodvnia or

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		glossopyrosis
	Ranula	Large mucocele penetrating the mylohyoid muscle; may plunge deep into the neck; swollen floor of mouth
	Thyroglossal duct cyst	Midline swelling that moves upward when tongue protrudes
	ТВ	Ulcers on dorsum (firm), cervical adenopathy
Salivary glands	Benign lymphoepithelial lesion (Mikulicz's disease)	Unilateral or bilateral enlargement of salivary glands; often with dry mouth and eyes
	Sialadenitis	Swelling, often painful; benign
	Sialolithiasis	Swelling (eg, of floor of mouth) that increases at mealtime or when offered a pickle
	Sjögren's syndrome	Systemic disease causing dry mucous membranes
	Xerostomia	Dry mouth; usually secondary to drugs
Various	Acute herpetic gingivostomatitis	Widespread ulcerating vesicular lesions; always present on gingiva; other locations may be involved; usually in young children

Behçet's syndrome	Multiple oral ulcers similar to those of aphthous stomatitis; also includes dry eyes
Cicatricial pemphigoid	Bullae that rupture quickly, leaving ulcers; ocular lesions develop after oral lesions; found on alveolar mucosa and vestibules
Condyloma acuminatum	Venereally transmitted wart forming cauliflower-like clumps
	Occurs with erythroplakia (red), leukoplakia (white patch on mucous membrane that does not rub off), and mixed red and white lesions; precancerous
Hemangioma	Purple to dark-red lesions, similar to port wine stain; benign
Hereditary hemorrhagic telangiectasia	Localized dilated blood vessels
Lichen planus	Lacy pattern (Wickham's striae), sometimes erosive; may become malignant; most common on buccal mucosa, lateral tongue
Lymphangioma	Localized swelling or discoloration; benign; most common on tongue

Mucocele (mucous retention cyst)	Soft nodule; if superficial, covered by thin epithelium; appears bluish; most common on lips and floor of mouth
Noma	Small vesicle or ulcer that rapidly enlarges and becomes necrotic
Pemphigoid	Small yellow or hemorrhagic tense bullae; may last several days before rupture; most common on vestibules and alveolar mucosa
Pemphigus	Bullae that rupture quickly, leaving ulcers; can be fatal without treatment
Canker sores, recurrent aphthous stomatitis	Small painful ulcers or large, painful scarring ulcers (two distinct conditions)
Syphilis	Chancre (red papule rapidly developing into a painless ulcer with a serosanguineous crust), mucous patch, gumma

Teeth are inspected for shape, alignment, defects, mobility, color, and presence of adherent plaque, materia alba (dead bacteria, food debris, desquamated epithelial cells), and calculus (tartar).

Teeth are gently tapped with a tongue depressor or mirror handle to assess tenderness (percussion sensitivity). Tenderness to percussion suggests deep caries that has caused a necrotic pulp with periapical abscess or severe periodontal disease. Percussion sensitivity or pain on biting also can indicate

an incomplete (green stick) fracture of a tooth. Percussion tenderness in multiple adjacent maxillary teeth may result from maxillary sinusitis. Tenderness to palpation around the apices of the teeth also may indicate an abscess.

Loose teeth usually indicate severe periodontal disease but can be caused by bruxism or trauma that damages periodontal tissues. Rarely, teeth become loose when alveolar bone is eroded by an underlying mass (eg, ameloblastoma, eosinophilic granuloma). A tumor or systemic cause of alveolar bone loss (eg, diabetes mellitus, hyperparathyroidism, osteoporosis, Cushing's syndrome) is suspected when teeth are loose and heavy plaque and calculus are absent.



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Calculus is mineralized bacterial plaque—a concretion of bacteria, food residue, saliva, and mucus with Ca and phosphate salts. After a tooth is cleaned, a mucopolysaccharide coating (pellicle) is deposited almost immediately. After about 24 h, bacterial colonization turns the pellicle into plaque. After about 72 h, the plaque starts calcifying, becoming calculus. When present, calculus is deposited most heavily on the lingual (inner, or tongue) surfaces of the mandibular anterior teeth near the submandibular

and sublingual duct orifices (Wharton's ducts) and on the buccal (cheek) surfaces of the maxillary molars near the parotid duct orifices (Stensen's ducts).

Caries first appears as defects in the tooth enamel. Caries then appears as white spots, later becoming brown.

Attrition (wearing of biting surfaces) can result from chewing abrasive foods or tobacco or from the wear that accompanies aging, but it usually indicates bruxism. Another common cause is abrasion of a porcelain crown occluding against opposing enamel, because porcelain is considerably harder than enamel. Attrition makes chewing less effective and causes noncarious teeth to become painful when the eroding enamel exposes the underlying dentin. Dentin is sensitive to touch and to temperature changes. A dentist can desensitize such teeth or restore the dental anatomy by placing crowns or onlays over badly worn teeth. In minor cases of root sensitivity, the exposed root may be desensitized by fluoride application or dentin-bonding agents.

Deformed teeth may indicate a developmental or endocrine disorder. In Down syndrome, teeth are small. In congenital syphilis, the incisors may be small at the incisal third, causing a pegged or screwdriver shape with a notch in the center of the incisal edge (Hutchinson's incisors), and the 1st molar is small, with a small occlusal surface and roughened, lobulated, often hypoplastic enamel (mulberry molar). In ectodermal dysplasia, teeth are absent or conical, so that dentures are needed from childhood. Dentinogenesis imperfecta, an autosomal dominant disorder, causes abnormal dentin that is dull bluish brown and opalescent and does not cushion the overlying enamel adequately. Such teeth cannot withstand occlusal stresses and rapidly become worn. People with pituitary dwarfism or with congenital hypoparathyroidism have small dental roots; people with gigantism have large ones. Acromegaly causes excess cementum in the roots as well as enlargement of the jaws, so teeth may become widely spaced. Acromegaly also can cause an open bite to develop in adulthood. Congenitally narrow lateral incisors occur in the absence of systemic disease. The most commonly congenitally absent teeth are the 3rd molars, followed in frequency by the maxillary lateral incisors and 2nd mandibular premolars.



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Defects in tooth color must be differentiated from the darkening or yellowing that is caused by food pigments, aging, and, most prominently, smoking. A tooth may appear gray because of pulpal necrosis, usually due to extensive caries penetrating the pulp or because of hemosiderin deposited in the dentin after trauma, with or without pulpal necrosis. Children's teeth darken appreciably and permanently after even short-term use of tetracyclines by the mother during the 2nd half of pregnancy or by the child during odontogenesis (tooth development), specifically calcification of the crowns, which lasts until age 9. Tetracyclines rarely cause permanent discoloration of fully formed teeth in adults. However, minocycline darkens bone, which can be seen in the mouth when the overlying gingiva and mucosa are thin. Affected teeth fluoresce with distinctive colors under ultraviolet light corresponding to the specific tetracycline taken. In congenital porphyria, both the deciduous and permanent teeth may have red or brownish discoloration but always fluoresce red from the pigment deposited in the dentin. Congenital hyperbilirubinemia causes a yellowish tooth discoloration. Teeth can be whitened.

Table 4		
Tooth Whi	tening Procedures	
Done By	Ingredients	Comments
Dentist		

In office Concentrated hydrogen Very effective peroxide is applied to teeth, Gingiva, skin, which is exposed to a light or and eyes laser must be protected **Patient** At home 6% carbamide peroxide Very effective (becomes 3% hydrogen peroxide when applied) and a thickening agent containing copolymers of acrylic acid cross-linked with a polyalkenyl polyether are added to a custom-made tray Patient (OTC products) Commercial Composed of carbamide Very effective whitening peroxide strips Usually contain carbamide or Whitening Moderately effective toothpaste hydrogen peroxide Paint-on Usually composed of titanium Not very whitening dioxide effective

Defects in tooth enamel may be caused by rickets, which results in a rough, irregular band in the enamel. Any prolonged febrile illness during odontogenesis can cause a permanent narrow zone of chalky, pitted enamel or simply white discoloration visible after the tooth erupts. Thus, the age at which the disease occurred and its duration can be estimated from the location and height of the band. Amelogenesis imperfecta, an autosomal dominant disease, causes severe enamel hypoplasia. Chronic vomiting and esophageal reflux can decalcify the dental crowns, primarily the lingual surfaces of the maxillary anterior teeth. Chronic snorting of cocaine can result in widespread decalcification of teeth, because the drug dissociates in

saliva into a base and HCI. Chronic use of methamphetamines markedly increases dental caries ("meth mouth").

Swimmers who spend a lot of time in overchlorinated pools may lose enamel from the outer facial/buccal side of the teeth, especially the maxillary incisors, canines, and 1st premolars. If Na carbonate has been added to the pool water to correct pH, then brown calculus develops but can be removed by a dental cleaning.

Fluorosis is mottled enamel that may develop in children who drink water containing > 1 ppm of fluoride during tooth development. Fluorosis depends on the amount of fluoride ingested. Enamel changes range from irregular whitish opaque areas to severe brown discoloration of the entire crown with a roughened surface. Such teeth are highly resistant to dental caries.

The lips are palpated. With the patient's mouth open, the buccal mucosa and vestibules are examined with a tongue blade; then the hard and soft palates, uvula, and oropharynx are viewed. The patient is asked to extend the tongue as far as possible, exposing the dorsum, and to move the extended tongue as far as possible to each side, so that its posterolateral surfaces can be seen. If a patient does not extend the tongue far enough to expose the circumvallate papillae, the examiner grasps the tip of the tongue with a gauze pad and extends it. Then the tongue is raised to view the ventral surface and the floor of the mouth. The teeth and gingiva are viewed. An abnormal distribution of keratinized or nonkeratinized oral mucosa demands attention. Keratinized tissue that occurs in normally nonkeratinized areas appears white. This abnormal condition, called leukoplakia, requires a biopsy because it may be cancerous or precancerous. More ominous, however, are thinned areas of mucosa. These red areas, called erythroplakia, if present for at least 2 wk, especially on the ventral tongue and floor of the mouth, suggest dysplasia, carcinoma in situ, or cancer.

With gloved hands, the examiner palpates the vestibules and the floor of the mouth, including the sublingual and submandibular glands. To make palpation more comfortable, the examiner asks the patient to relax the mouth, keeping it open just wide enough to allow access.

The temporomandibular joint (TMJ) is assessed by looking for jaw deviation on opening and by palpating the head of the condyle anterior to the external

auditory meatus. Examiners then place their little fingers into the external ear canals with the pads of the fingertips lightly pushing anteriorly while patients open widely and close 3 times. Patients also should be able to comfortably open wide enough to fit 3 of their fingers vertically between the incisors (typically 4 to 5 cm). Trismus, the inability to open the mouth, may indicate temporomandibular disease (the most common cause), pericoronitis, scleroderma, arthritis, ankylosis of the TMJ, dislocation of the temporomandibular disk, tetanus, or peritonsillar abscess. Unusually wide opening suggests subluxation or type III Ehlers-Danlos syndrome.

Testing

For a new patient or for someone who requires extensive care, the dentist takes a full mouth x-ray series. This series consists of 14 to 16 periapical films to show the roots and bone plus 4 bite-wing films to detect early caries between posterior teeth. Modern techniques reduce radiation exposure to a near-negligible level. Patients at high risk of caries (ie, those who have had caries detected during the clinical examination, have many restorations, or have recurrent caries on teeth previously restored) should undergo bite-wing x-rays every 12 mo. Otherwise, bite-wings are indicated every 2 to 3 yr. A panoramic x-ray can yield useful information about tooth development, cysts or tumors of the jaws, supernumerary or congenitally absent teeth, 3rd molar impaction, Eagle's syndrome (less frequently), and carotid plaques.

Reference: http://www.merckmanuals.com