The Lips, Mouth, Tongue and Teeth: Part I

24

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24.1 Applied Anatomy and Physiology

The oral region is a complex structure which includes the mouth (oral cavity), teeth, gingivae (gums), lips, anterior two thirds of the tongue, floor of the mouth, sublingual and minor salivary glands, deep lobes of the submandibular glands and the palate. It extends from the lips anteriorly, to the circumvallate papillae in the tongue and the junction of the hard and soft palates posteriorly, with the anterior faucial arch (tonsillar pillars, or "fauces") laterally. Behind these structures lays the oropharynx (oral part of the pharynx), or the throat (containing the posterior third of the tongue and tonsils). This is discussed in its own chapter. Laterally, the oral cavity is bounded by the buccal mucosa, lining the cheeks. The palate separates the oral cavity from the nasal cavity. The oral cavity is where food is initially broken down and ingested. Deglutition (swallowing) is initiated here.

24.1.1 The Tongue and Floor of the Mouth

24.1.1.1 The Tongue

This is a mobile muscular structure that can produce a variety of shapes and positions. It is partly in the oral cavity proper and partly in the oropharynx. It is made up mostly of multiple groups of muscular bundles that pass in different directions. These are divided into intrinsic muscles (with their attachments entirely within the tongue) and extrinsic muscles (with their origins arising beyond to the tongue).

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These muscles work together as a functional unit, supported by the underlying mylohoid muscle, enabling complex, precise and reasonably powerful movements of the tongue. Alongside the tongue, passing forwards in the floor of the mouth are the hypoglossal nerve, lingual nerve, and submandibular (Wharton's) duct.

Embryologically, the tongue develops from the first, second, third, and fourth pharyngeal arches. At the end of the fourth week of development, a small midline triangular swelling called the 'tuberculum impar' develops in the floor of primitive pharynx. Just behind this is a small pit which marks the site of descent of the thyroid diverticulum (future thyroid). This is known as the 'foramen cecum'. Soon after the development of the tuberculum impar, two lateral lingual swellings develop, one on each side. These are related to the first pharyngeal arch and are sometimes referred to as 'distal tongue buds'. Behind the tubercular impar, a second large midline swelling (the hypobranchial eminence, or copula of His) develops in the floor of primitive pharynx. This is closely related to the developing second, third, and fourth pharyngeal arches. The hypobranchial eminence soon subdivides into large cranial part and smaller caudal part. The two lateral lingual swellings overgrow the tuberculum impar and merge together to form the anterior two-thirds of the tongue. The line of fusion may persist as the median sulcus on the dorsal surface of the tongue. The tuberculum impar itself does not form any recognisable part of the adult tongue.

The mucous membrane of the tongue is derived from the endoderm of the primitive foregut. The overlying mucosa in the anterior two-thirds is derived from the first pharyngeal arch. It is therefore innervated by the nerve of first arch, the mandibular branch of trigeminal nerve. The posterior one-third of the tongue (including circumvallate papillae) develops from the cranial part of hypobranchial eminence. The mucosa here is derived from the third pharyngeal arch. Hence it is supplied by glossopharyngeal nerve (the nerve of third arch). The line of fusion between the anterior two-thirds and posterior one-third of the tongue is seen as a V-shaped groove, the 'sulcus terminalis'. The lowermost part of the tongue and the epiglottis develop from the caudal part of the hypobranchial eminence. Here, the mucosa develops from fourth pharyngeal arch and is therefore supplied by the superior laryngeal nerve—the nerve of the fourth arch.

During all this development, the mesoderm of the third arch grows over the mesoderm of the second arch and fuses with the mesoderm of the first arch. Consequently, the second arch gets buried underneath the third and excluded from further development. Taste sensation from anterior two-thirds of the tongue is carried by the chorda tympani branch of the facial nerve (CN VII) within the lingual nerve. The cell bodies for these neurones lie in the geniculate ganglion. Their peripheral processes "hitch a ride" with the lingual and chorda tympani nerves. Taste from the posterior third it is carried by the glossopharyngeal nerve and from lowermost part of the tongue by the internal laryngeal nerve. It is said that beer is tasted in the lowermost part of the tongue, which is why the internal laryngeal nerve is sometimes called the 'beer drinker's nerve'.

The muscles of tongue develop from myoblasts that migrate from the occipital myotomes. The hypoglossal nerve (the nerve to the occipital myotomes) accompanies these myoblasts during their migration and thus innervates the muscles of tongue. This development explains the long course of the nerve as it passes

superficial to the external and internal carotid arteries, and its innervation of the tongue. With further growth in size of the tongue, a linguogingival sulcus develops on either side, separating it from the floor of the mouth. Thus, the segmental nature and somewhat confusing anatomy of the tongue can be understood. Aglossia and microglossia are very rare anomalies, often associated with malformations in the extremities, cleft palate and dental agenesia. There develops a rudimentary, small tongue. As a consequence the alveolar arches do not develop transversely and the mandible does not fully develop, resulting a severe facial deformity. The cause is believed to be trauma during the first few weeks of gestation. A completely cleft or bifid tongue is another rare condition due to a lack of mesenchymal proliferation and merging of the lingual swellings. It is often found as one feature of the oral-facial-digital syndrome.

Clinically, the tongue effectively has two surfaces—an upper/posterior surface, called the dorsum of the tongue and an inferolateral, or ventral surface. This latter surface, together with the floor of the mouth is a common site for the development of oral cancer. In most patients the terminal sulcus and foramen cecum can often be seen, demarcating the anterior two-thirds of the tongue from the posterior third. Usually the tongue occupies most of the oral cavity when the mouth is closed. Abnormal enlargement has many cause, some localised (AV malformations), others systemic (acromegaly, amyloidosis). Both the dorsal surface and lateral edge of the tongue are covered by multiple small projections, the papillae or "taste buds" (circumvallate, filiform, fungiform and foliate). Posterolaterally, there are also clumps of lymphatic tissue, resulting in a cobblestoned appearance. This is part of Waldeyer's ring (see the chapter on the throat) and is often a common cause for concern in both patients and inexperienced clinicians. The undersurface of the tongue and the floor of the mouth are covered with a thinner and more mobile mucous membrane. A midline fold, the frenulum, occasionally extends to the tip of the tongue resulting in "tongue-tie". Under this mucosa many large worm-like lingual veins may be visible. These have a significant bloodflow, hence the use of rapid acting sublingual nitrates in this site. In the elderly these veins often become varicose and may sometimes be confused with pathology (Figs. 24.1 and 24.2).

Lingual Artery Aneurysms

Aneurysms of the lingual artery (and other external carotid artery branches) are uncommon. However there have been occasional reports of severe haemorrhage from lingual artery pseudoaneurysms during or after tonsillectomy. Case reports of presumed congenital lingual artery aneurysms also exist, based on absence of previous trauma.

All the muscles of the tongue, with the exception of palatoglossus, receive their motor innervation from the hypoglossal nerve. General sensation to the anterior two-thirds of the tongue is supplied by the lingual nerve (a branch of the mandibular division of the trigeminal nerve). Taste is supplied primarily through the chorda tympani, a branch of the facial nerve (which joins the lingual nerve in the infratemporal fossa and runs within its sheath). Both general sensation and taste to the posterior third of the tongue is mostly supplied by the glossopharyngeal nerve (CN IX), with a tiny contribution from the internal laryngeal nerve, a branch of the vagus nerve.

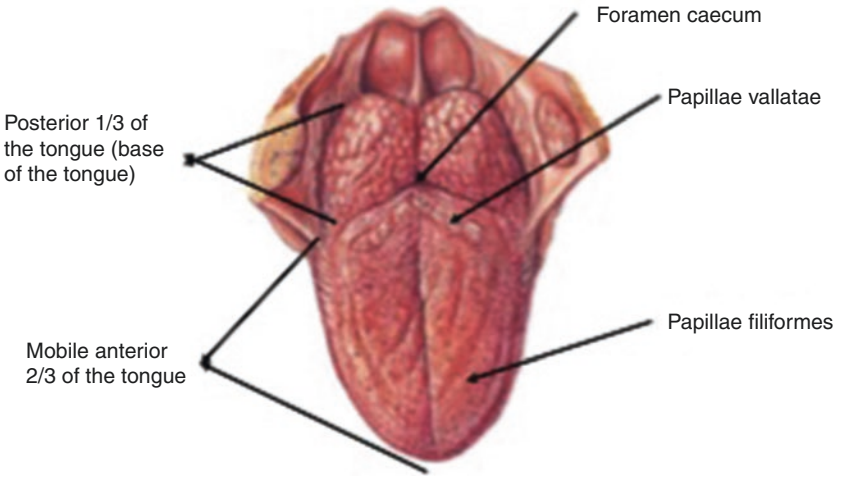
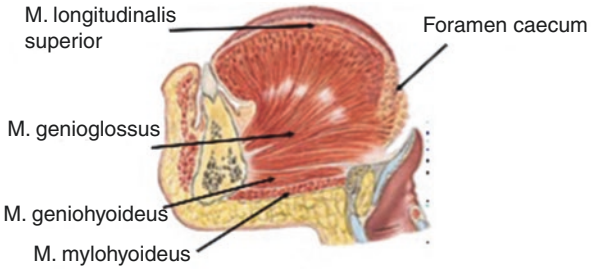


Fig. 24.1 Anatomy of the tongue

Fig. 24.2 Extrinsic and intrinsic muscles of the tongue



24.1.1.2 The Floor of the Mouth

During development the mouth is derived from two sources (i) the stomodeum (ectodermal) and (ii) the foregut (endodermal). These are initially separated from each other by the buccopharyngeal membrane. As this disappears (during the third week of intrauterine life) the foregut communicates with the exterior. Thus the oral cavity is embryologically considered as two parts

1. The primitive oral cavity, derived from ectodermal stomodeum
2. The definitive oral cavity derived from the endodermal foregut.

Following rupture of buccopharyngeal membrane the junction between ectodermal and endodermal can no longer be seen. It is estimated to occupy an imaginary plane passing obliquely downwards from the body of sphenoid, through the soft palate to the inner surface of the mandible below the incisor teeth. Most of the adult oral cavity is therefore derived from ectodermal stomodeum, except the floor of the mouth, which is endodermal in origin. Consequently the epithelial lining of the cheeks, lips, gums, and hard palate are ectodermal in origin, whereas the epithelial

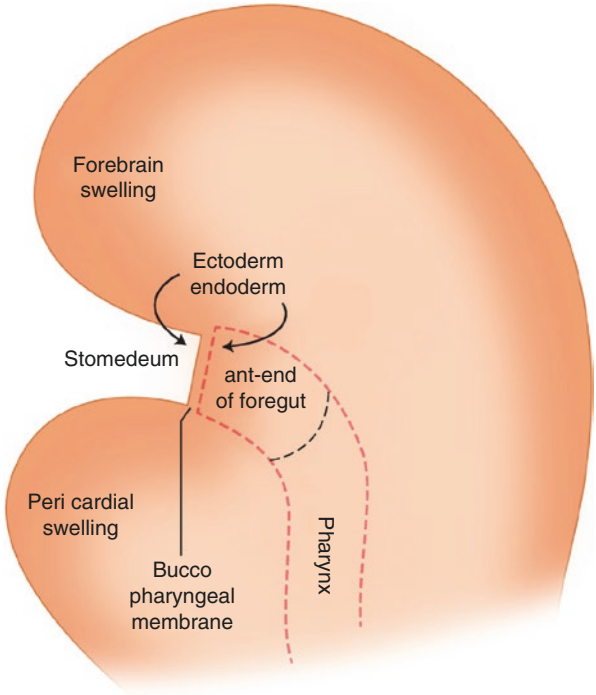


Fig. 24.3 Embryology of the oral cavity

lining of tongue, floor of the mouth, most of the soft palate and palatoglossal and palatopharyngeal folds are endodermal. Teeth are ectodermal in origin (Fig. 24.3).

In the adult, the floor of the mouth (FOM) is a U-shaped structure covered by loose, distensible mucosa. The mylohyoid muscle (sometimes referred to as the diaphragm of the mouth) provides a platform for the tongue and other structures in the floor of the mouth. Additional support comes from the geniohyoid and the anterior bellies of the digastric muscles. The space between these muscles and overlying mucosa comprises the sublingual space. This communicates with the submandibular space in the neck, around the back edge of the mylohyoid (the glosso-mylohyoid gap). The tissues in these spaces are very friable. Hence infection can spread quickly throughout both the right and left sublingual spaces and into their respective submandibular spaces (and the submental spaces in the neck), as occurs in Ludwig's angina. The sublingual space contains the submandibular duct, deep lobe of the submandibular gland, sublingual gland, hypoglossal and lingual nerves. Lymphatic drainage from the tongue and floor of the mouth generally passes to the submandibular and submental lymph nodes and from there to the deep cervical lymph nodes in the neck (Fig. 24.4).

These sites are important when oral cancer is suspected. The submandibular gland passes around the posterior edge of the mylohyoid muscle to enter the floor the mouth. From here the submandibular (Wharton's) duct passes forwards, to open into the oral cavity just behind the lower incisor teeth. Anatomically, the oral cavity

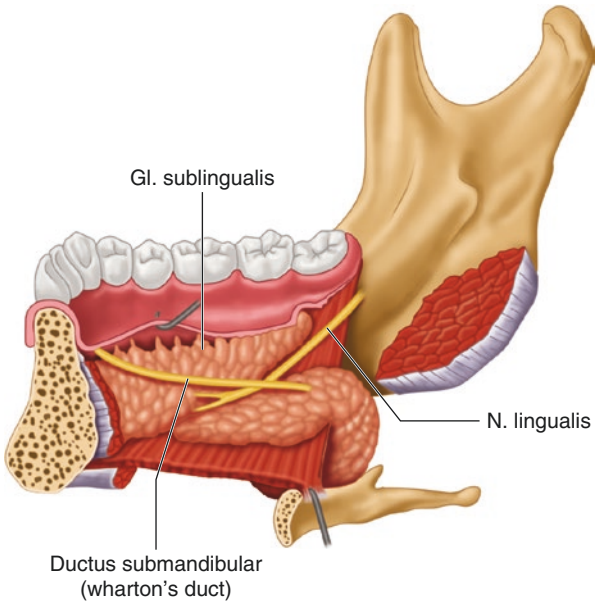


Fig. 24.4 Topographic situation of sublingual gland, lingual nerve and Wharton's duct

is usually divided into two parts (i) the oral vestibule and the (ii) oral cavity proper. The vestibule is the slit-like space between the upper and lower teeth, their gums and alveolar bone (on the inside), and the overlying lips and cheeks. The circumoral muscles include the orbicularis oris (the sphincter of the oral fissure), the buccinator, risorius, and the depressors and elevators of the lips (dilators of the fissure).

24.1.2 The Lips and Cheeks

The lips are mobile, musculofibrous structures which surround the opening of the mouth, extending from the nasolabial region and base of the nose to the chin. They are more than just the red 'kissable' part we see. They contain the orbicularis oris and labial muscles, vessels and nerves. Externally the lips are covered by tightly bound but flexible skin, internally they and lined by mobile mucous membrane. The transitional zone between these two skin types is a common site for cold sores. Lymphatics from the upper lip and lateral parts of the lower lip passes mostly to the submandibular lymph nodes. Lymphatics from the central part of the lower lip passes to the submental lymph nodes. These sites need careful examination when assessing suspected lip cancers. Sensation of the lower lip is provided by the mental nerve, a terminal branch of the inferior alveolar nerve. This exits the mental foramen of the lower jaw, near the roots of the premolar teeth. Numbness of the lower lip and chin suggests injury to this nerve, commonly as a result of trauma, infection or tumour.

The cheeks have essentially the same structural arrangement as the lips. These may become swollen as a result of pathology involving the teeth, upper jaw, lower jaw and parotid gland. The cheeks also contain the buccal fat-pads, which can

sometimes bulge into the oral cavity, appearing as a lump. The principal muscle of the cheeks is the buccinator. The muscles of both the lips and cheeks are supplied by the mandibular and buccal branches of the facial nerve. The parotid gland drains into the mouth adjacent to the second maxillary molar tooth. Here, the mucosa is pierced by Stensen's duct. A small papilla is usually present. This acts as a one way valve. Sometimes air can be forcibly driven back up the duct and into the gland (sometimes seen in wind-instrument musicians).

24.1.3 The Gingivae (Gums)

These are composed of fibrous tissue covered by mucous membrane. The "attached" gingiva is firm and adherent to the underlying 'alveolar' bone which supports the teeth. It is normally pink, stippled and keratinised. Pigmentation is common in dark skinned individuals. The 'free' or unattached gingiva is normally shiny, red and non-keratinising. This is essentially an extension of the lining oral mucosa.

24.1.4 The Teeth

Healthy teeth are firmly supported in their sockets and are important in biting, chewing and articulation. These may be deciduous (primary or 'baby'), or permanent (secondary or 'adult'). Children have 20 deciduous teeth, adults normally have 32 permanent teeth. Teeth normally erupt at predictable times during growth and usually symmetrically (±3–6 months). Significantly delayed eruption, or failure of one side to erupt requires further investigation by the patient's dentist.

The development of the teeth is closely associated with the development of the underlying alveolar processes of the jaws. Embryologically, teeth develop in each alveolar process. This involves a complex interaction between the neural crest mesenchyme in the process and the overlying ectoderm. Five stages of tooth development have been described (dental lamina, bud, cap, bell and apposition). In essence, the ectodermal epithelium overlying the alveolar process becomes thickened and projects into the underlying mesoderm to form the dental lamina. This proliferation occurs at ten sites, to produce ten local swellings called 'enamel organs' (tooth buds) within each U-shaped alveolar process, (five on each side). These will go on to form initially the 20 deciduous teeth and later the permanent teeth. At the same time, the neural crest mesenchyme invaginates into each tooth bud and becomes the dental papilla. At this stage each tooth bud is comprised of three layers (i) an outer enamel epithelium, (ii) an inner enamel epithelium and (iii) a central core of loosely arranged cells called the enamel reticulum. As the bud differentiates, the developing tooth becomes bell-shaped. The inner enamel epithelial cells lining the dental papilla develop into ameloblasts. These will subsequently form the tooth's enamel. At the same time the adjacent mesodermal cells of dental papilla develop into odontoblasts. These will form dentine and the pulp of the tooth. With further development the mesenchyme surrounding the tooth condenses to form a dental sac. This

will eventually become the cementum and periodontal ligament. During enamel formation the ameloblasts move towards the outer enamel epithelium and eventually disappear, leaving only a thin membrane—the dental cuticle. After the eruption of tooth, this membrane is gradually sloughed off. The odontoblasts produce predentin deep to the enamel, which later calcifies and forms dentine. As this thickens the odontoblasts regress, but their cytoplasmic processes (odontoblastic or Tomes processes) remain embedded in the dentine.

The root of the tooth begins to develop after the formation of enamel and dentine is well advanced. The outer and inner enamel epithelia join at the neck of the tooth to form a fold—Hertwig's epithelial root sheath. This sheath grows in the mesenchyme and initiates the formation of the root. Odontoblasts adjacent to the root sheath produce dentine. As more dentine is produced, the pulp cavity narrows and forms the pulp canal, through which nerve and vessels pass. The dental sac differentiate into cementoblasts that produce cementum. These also give rise to the periodontal ligament that holds the root of the tooth in the alveolar socket (a 'gomphosis').

In the adult, each tooth has a crown and root. The crown provides the biting/chewing surface. The number of roots varies depending on the tooth, but they are all fixed into their sockets by the periodontal ligament. The term periodontium often refers to the periodontal ligament plus the gingiva. Much of the tooth is composed of dentine, a bone-like material, which is covered by enamel over the crown and cementum over the root. Each tooth has a central pulp cavity which contains connective tissue and an extensive neurovascular supply (plexus of Raschkow) that can perceive painful sensations and precipitate inflammation. The pulp communicates with the supporting bone at the tip (apex) of each root through the apical foramen. Hence dental decay (caries), can pass through the crown, into the pulp and result in significant infection, which if untreated can spread into the jaw.

24.1.5 The Retromolar Trigone (RMT)

This is an area of mucosa just behind the upper and lower wisdom teeth, which covers the anterior surface of the ramus of the mandible. This important landmark is the junction between the oral cavity and the oropharynx. Infections here can quickly spread into the buccal, masticator and parapharyngeal spaces. It is also a common site for cancer to develop.

24.1.6 The Palate

The palate is composed of two parts, the hard palate anteriorly and the soft palate posteriorly. This forms both the roof of the mouth and the floor of the nasal cavity, separating the two. The nasal surface is covered with respiratory mucosa, the oral surface is covered with oral mucosa, which is densely packed with minor salivary glands. Occasionally minor salivary glands can be found in the respiratory mucosa

of the nose. The hard palate lies within the horseshoe-shaped maxillary alveolar process. Swellings in this region are most commonly dental in origin, although salivary gland tumours can also occur. Embryologically, the palate consists of two parts

(i) The primary palate (or premaxilla). This Intermaxillary segment develops from the frontonasal process. It will ultimately forms the philtrum of the upper lip, four incisor teeth and the primary palate.
(ii) The secondary palate. This develops from the maxillary processes.

Development involves two stages.

(i) Development of primary palate occurs following fusion of the two medial nasal processes of the frontonasal process. This forms a wedge-shaped mass of mesenchyme opposite the upper jaw, which houses the four incisor teeth.
(ii) Development of secondary palate produces the main bulk of the adult palate. This is formed following the fusion of two shelf-like outgrowths (the 'palatine shelves') which arise from the inner aspects of the maxillary processes. These appear in the sixth week of development. Initially they grow downward and medially and are sited on either side of the tongue. As the oral cavity enlarges they assume a more horizontal position and fuse with each other to form the secondary palate. This will go on to become most of hard palate and all of the soft palate.

The primary and secondary palates fuse at the incisive foramen to form the 'definitive' or permanent palate. A Y-shaped suture is seen, with each limb of Y passing between the lateral incisor and canine teeth. In adults, the junction between the primitive and secondary palates is seen at the incisive fossa. At the same time the nasal septum grows down from the frontonasal process and joins upper aspect of the hard palate (Figs. 24.5, 24.6, 24.7, and 24.8).

24.1.7 The Sublingual and Minor Salivary Glands

The sublingual glands are the smallest of the major salivary glands. Each is about 2.5 cm, sausage-shaped and lays in the floor of the mouth between the inner surface of the mandible and the tongue. The glands join in the midline to form of a horseshoe-shaped structure. Multiple small ducts open into the floor of the mouth. In addition to the three major salivary glands, numerous small accessory glands can be found scattered throughout the lining mucosa of the palate, lips, cheeks and tongue. These glands can occasionally become cystic and very rarely undergo malignant change. All lumps and firm swellings within the oral cavity, especially those in the floor the mouth, cheeks, upper lip and palate, should be taken seriously. Salivary choristoma is ectopic, histologically normal salivary tissue in abnormal sites. Such heterotopic salivary tissue has been reported in a large number of unusual locations including the paranasal sinuses, pharynx, larynx and trachea, middle ear cleft, external

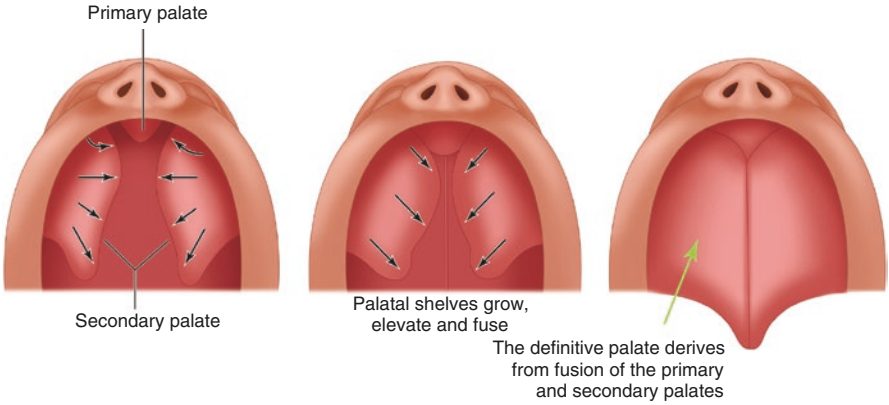


Fig. 24.5 The primary palate fuses with the secondary palate by week 9 to separate the oral and nasal cavities. (**a**) Primary palate and secondary palate, (**b**) Palatal shelves grow, elevate and fuse, (**c**) The definitive palate derives from fusion of the primary and secondary palates

auditory canal, pituitary and the cerebellopontine angle. All of these sites have the potential to develop salivary cysts and tumours.

24.1.7.1 Saliva

Saliva is mostly water. Between 1 and 1.5 L are produced daily, mostly during eating. About 45% comes from the parotid glands, 45% from the submandibular glands and 5% from the sublingual glands. The remainder comes from the minor salivary glands. Secretion is stimulated by the parasympathetic system. Saliva has many functions and is critical for maintaining the health of the oral tissues. It aids in mastication and swallowing and initiates digestion. It protects the mucosa from toxins and has wound healing factors (which is why many animals lick their wounds). It also contains mucosal protective glycoproteins, secretory immunoglobulin (Ig) A and many enzymes including lysozyme, peroxidase, amylase and lactoferrin. Contrary to popular belief saliva is actually sterile. It only becomes contaminated once it enters the oral cavity. Production of saliva and its composition can both be affected by many diseases and other external factors. These include drugs, radiotherapy, Sjögren syndrome, celiac disease, diabetes mellitus, chronic renal insufficiency, anorexia, bulimia and anxiety.

24.1.8 Nerves

The management of dental pain is mostly related to the trigeminal nerve. This is the major sensory nerve of the face (the largest of the cranial nerves), which contains both motor and sensory fibres. It exits the brainstem between the pons and the middle cerebellar peduncles and enters the trigeminal ganglion (sitting in Meckel's cave near the apex of the petrous part of the temporal bone). It then splits into three major divisions—ophthalmic, maxillary and mandibular. The ophthalmic branch (Va) runs

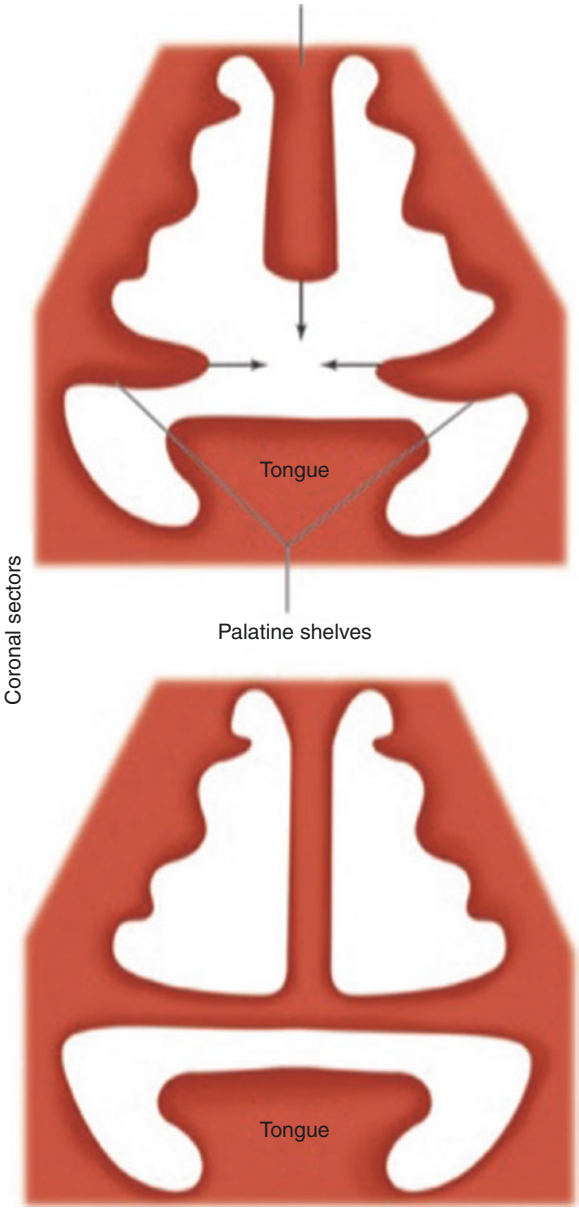


Fig. 24.6 As the tongue descends, the palatal shelves grow and elevate till they eventually fuse in their midline and with the developing nasal septal tissue

along the lateral wall of the cavernous sinus and passes through the superior orbital fissure to enter the orbit. It provides sensation to the lacrimal apparatus, cornea, iris, forehead, ethmoid and frontal sinuses and the nose. The maxillary branch (Vb) passes through the lateral wall of the cavernous sinus, exiting the cranium through the foramen rotundum. It then passes between the pterygoid plates and the palatine bone. At the pterygopalatine fossa it gives off branches to the posterior–superior

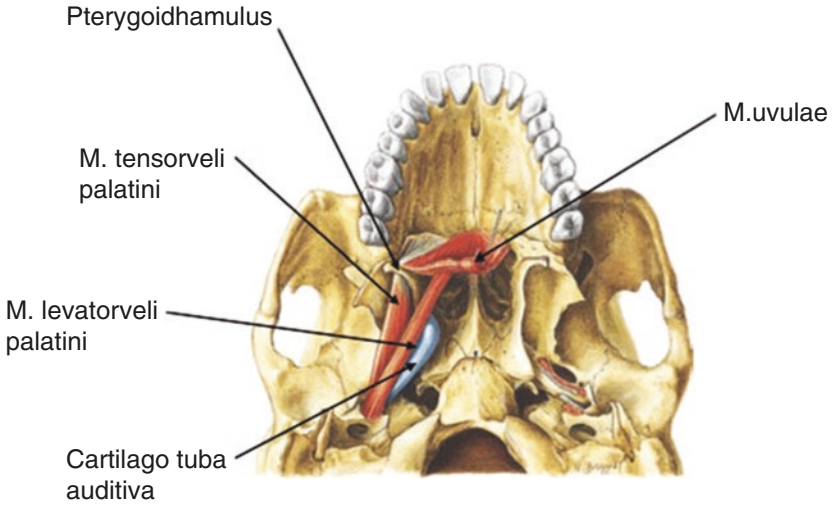


Fig. 24.7 Development of the Palate

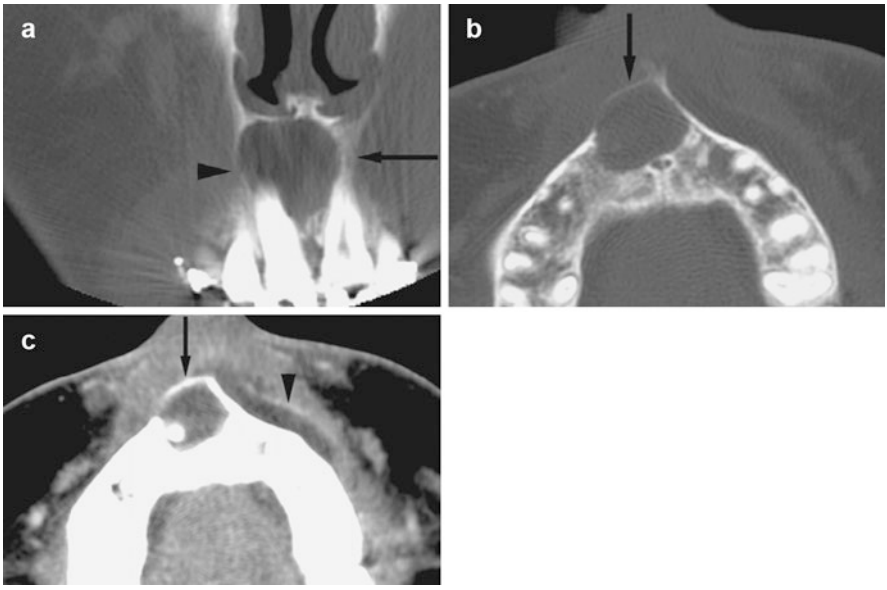


Fig. 24.8 Periapical cyst, maxilla, with abscess development; 14-year-old male with swelling of mucobuccal fold, pain, foul odor, and nonvital right central incisor. (**a**) Coronal CT image shows expansive process with well-defined border (*arrow*), thin and perforated on right side (*arrowhead*). (**b**) Axial CT image shows process (*arrow*) with no sclerotic border in alveolar bone on right side. (**c**) Axial CT image, soft-tissue window shows cyst in alveolar process (*arrow*) with inflammatory exudate (*hypodense area*) along buccal surface and elevation of periosteum (*arrowhead*)

alveolar nerve, the sphenopalatine ganglion and the zygomatic region. The nerve then continues through the inferior orbital canal and exits onto the anterior cheek, providing sensation to the maxillary sinuses, upper jaw, sides of the nose and the cheek. A branch entering the cranium (the middle meningeal nerve) travels with the middle meningeal artery to provide sensation to the dura. Some of the maxillary division's more major branches include

(i) The zygomatic nerve
(ii) The pterygopalatine nerve
(iii) The greater palatine nerve (which supplies sensory innervation to the hard palatal as far anterior as the first premolar).
(iv) The lesser palatine nerve.
(v) The posterior superior alveolar nerve (PSA)
(vi) The anterior superior alveolar nerve (branches off from the infraorbital nerve to supply the incisors, canine and their associated periodontal tissues, buccal bone and mucous membranes).
(vii) The middle superior alveolar nerves (provides sensation to the maxillary premolars and their associated periodontal tissues, buccal bone and mucous membranes).

The mandibular branch (Vc) is a motor-sensory nerve that supplies the masticatory muscles, lower jaw/teeth, parotid and sublingual glands and the anterior two thirds of the tongue. It also supplies the ear canal. This nerve exits the skull through the foramen ovale and enters the infratemporal fossa (ITF). It descends between the ramus and the medial pterygoid muscle and enters the mandible through the mandibular foramen. Its main branches include

(i) The inferior alveolar nerve (IAN), which passes forward within the mandible itself. At the mental foramen it branches into its terminal incisive and the mental nerves, which innervate the lower incisor teeth, gums, chin and lower lip.
(ii) The buccal nerve. Compression here has been reported in hyperactive temporalis muscles and may result in neuralgia-like paroxysmal pain
(iii) The deep temporal nerve (DTN) passes above the lateral pterygoid to enter the deep surface of the temporalis.
(iv) The auriculotemporal nerve. This usually has 2 roots. It encircles the middle meningeal artery and runs backwards, crossing the neck of the mandible. It then runs behind the TMJ to emerge deep in the upper part of the parotid gland.
(v) The lingual nerve (LN). This important nerve provides sensation to the anterior two-thirds of the tongue, mucous membrane of the mouth floor and lingual gums. It branches off the IAN, about 1 cm below the skull base. The nerve runs between the tensor veli palatine and the lateral pterygoid where it is joined by the chorda tympani branch from the facial nerve (carrying taste fibres and parasympathetic fibres to the submandibular and sublingual salivary glands). The LN supplies general sensation to the mucosa, the floor of the mouth, the lingual gingiva and the mucosa of the anterior two-thirds of the

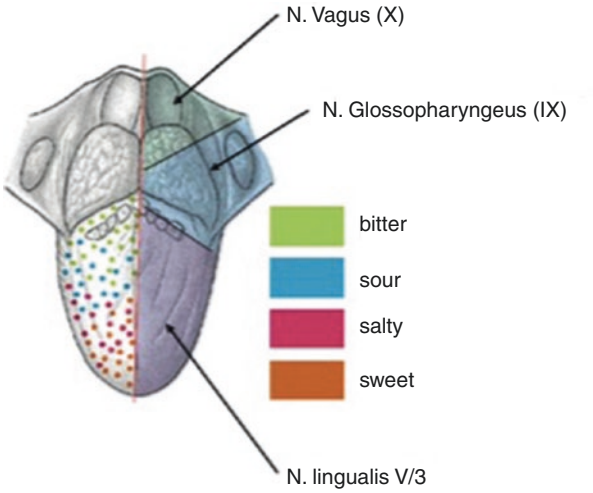


Fig. 24.9 Innervation of the tongue

tongue. It also carries fibres for taste sensation to the anterior part of the tongue via the chorda tympani.

(vi) The mylohyoid nerve branches off the IAN just before it enters the mandibular canal. This is a mixed nerve providing sensory innervation to the mandibular molars and motor innervation to the anterior belly of the digastrics and mylohyoid muscle. It is often implicated as the cause of unsuccessful anaesthesia of the molar teeth following an IAN block

(vii) The mandibular division also supplies motor fibres to the tensor veli palatini, and tensor tympani muscle (Fig. 24.9).

Entrapment or compressive neuropathy can affect several of these nerves, at points where pressure or mechanical irritation from nearby structures arises. This commonly occurs at sites where the nerve passes through a fibro-osseous canal. Impingement from bone, muscle or a fibrous band can then occur. A common site for trigeminal nerve compression is the infratemporal fossa (ITF). This complex anatomical region contains several muscles, the pterygoid venous plexus, the maxillary artery and branches of the mandibular nerve. The maxillary artery is often in close contact with the inferior alveolar nerve and lingual nerve. It has therefore been suggested that some cases of temporomandibular joint pain and idiopathic facial pains may be a form of entrapment neuropathy (Figs. 24.10 and 24.11).

24.1.8.1 Hypoglossal Nerve Lesion (Motor Weakness)

Isolated hypoglossal nerve palsy can have many causes. These include

- Metastatic disease at the base of skull
- Subluxation of odontoid process and fractures through the occipital condyle

- Sarcoidosis, autoimmune disease and vasculitis
- Arnold-Chiari malformation
- Dural arteriovenous fistula of the transverse sinus
- Periostitis of hypoglossal canal
- Post retropharyngeal infection
- Acute poliomyelitis
- Syringobulbia
- Thrombosis of median branches of vertebral artery
- Multiple sclerosis
- Carotid artery dissection/aneurysm
- Diabetes mellitus
- Lacunar infarct over the hypoglossal nerve nucleus
- Glomus tumour
- Meningioma
- Persistent hypoglossal artery

Following trauma, weakness may suggest a very high cervical spine injury. CNXII palsy is also a rare postoperative complication after airway management, and has been reported after use of the laryngeal mask airway. Symptoms include ipsilateral tongue deviation, dysarthria, and dysphagia. In addition to isolated injuries, hyperextension and lateral flexion of the neck can compress an endotracheal tube against the hypoglossal-recurrent laryngeal nerve, resulting in Tapia's syndrome. By the end of the first postoperative day, patients typically present with ipsilateral tongue deviation and may exhibit speech and swallowing difficulties.

Therefore in any patient presenting with tongue weakness, consider hypoglossal nerve lesions. On examination the protruded tongue deviates towards the affected side. It may also have accompanying fasciculation or wasting. Patients with a weak tongue may also experience slurring of speech and swallowing difficulties. Since the underlying pathology is so diverse an MRI is usually required. Treatment is dependent on the cause.

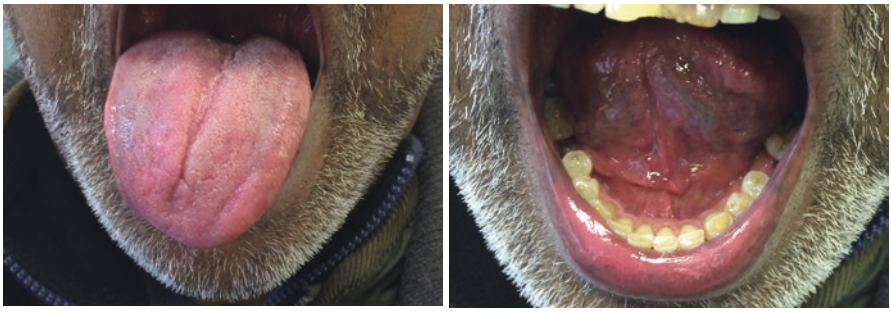


Fig. 24.10 Hypoglossal nerve palsy

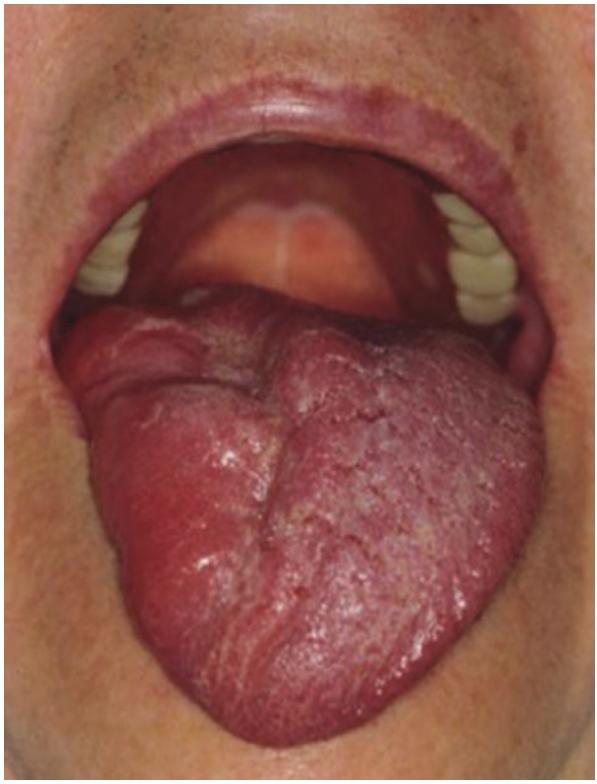


Fig. 24.11 Paralysis of the hypoglossal nerve with resulting atrophy of the right part of the tongue and diverging of the tongue to the affected side

24.1.9 Anatomical Variants

The oral region is a highly variable site. Like fingerprints, no two are ever exactly alike. Changes in the texture, mobility and pigmentation of the mucosa and changes in the bony contour are common and these can sometimes result in diagnostic confusion to those not familiar with the normal anatomy of this region. The patient is often a good guide to the inside of their mouths—we all know what is 'normal' for us. Some of the more common anatomical variants are linea alba, leukoedema, changes in pigmentation and lingual tonsils.

24.1.9.1 Lip Pits

Commissural lip pits are dimple-like invaginations of the corner of the lips. These represent failure of fusion of the embryonic maxillary and mandibular processes. They may be unilateral or bilateral and tend to occur on the vermilion portion of the lip. Pits are generally small. Paramedian lip pits are also congenital depressions that occur in the mandibular lip, most often on either side of the midline. They develop when the embryonic mandibular arch fails to regress in utero. Paramedian lip pits are often inherited as an autosomal dominant trait in combination with cleft lip or cleft palate and hypodontia (van der Woude syndrome).

24.1.9.2 Racial Pigmentation

Like the skin, the mucosa contains melanin. The amount present often reflects the amount of pigmentation in the skin. In the mouth and gums, multiple small pigmented patches are often noted. This is referred to as benign melanosis. It is usually generalised and symmetrical. Excessive pigmentation can be an indication of Addison's disease, smokers melanosis or it may be drug-related. No treatment is required, although single patches of melanosis may raise concerns regarding malignant melanoma (Fig. 24.12).

24.1.9.3 Foliate Papillae, Lymphoid Aggregates and the Lingual Tonsil

These are found on the back of the tongue. Lymphoid aggregates may also be seen on the soft palate and pharyngeal arches. These often appear as small clumps of tissue. Lingual tonsils and lymphoid aggregates may become hyperplastic, red or painful secondary to local inflammation. Occasionally small inclusion cysts may develop. These changes are often confused with oral cancer. Biopsy may be required, but usually the benign nature of these is self-evident.

24.1.9.4 Fordyce Spots

These are very common, often seen in the buccal mucosa, lining both cheeks. They are tiny collections of ectopic sebaceous glands.

24.1.9.5 Varicosities

These are localised abnormal dilations in the vein, commonly seen in the elderly, in the lips, labial mucosa and under the tongue. Occasionally they can become thrombosed. They can sometimes be confused with mucocoeles and minor salivary gland tumours, especially if they are deeper and less purple in colour. No treatment is necessary.

24.1.9.6 Fissured Tongue

The tongue can have a number of natural fissures in its dorsal surface. These can vary in number, depth and orientation and can sometimes be associated with Geographic tongue. They can sometimes become sore if they trap bacteria and become inflamed. Otherwise no treatment is required (Fig. 24.13).

24.1.9.7 Scalloped (Crenated) Tongue

This is a common finding in which indentations are seen on the lateral border of the tongue. These are caused by abnormal pressure (e.g., suction) within the mouth of persons who clench their teeth (bruxism). It occurs where the tongue is held in close contact with the teeth resulting in a distinctive pattern and a 'nice fit' between the tongue and adjacent tooth. Scalloped tongue is seen in normal patients, but can be associated with temporomandibular joint disorders, acromegaly, amyloidosis and Down syndrome. It is harmless and asymptomatic.

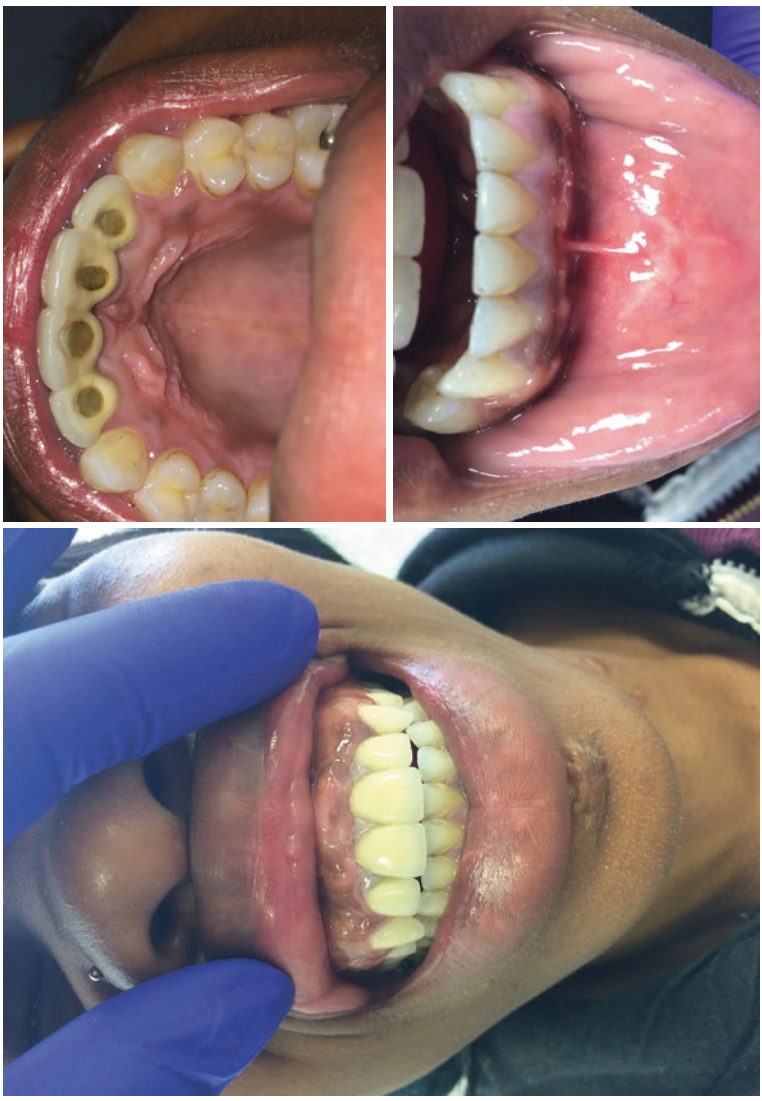


Fig. 24.12 Normal racial pigmentation

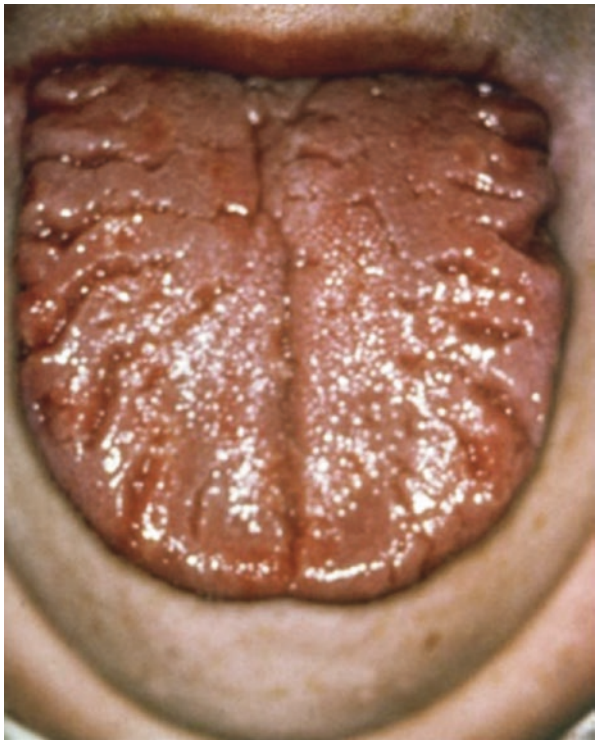


Fig. 24.13
Fissured tongue

24.1.9.8 Exostoses

These are benign localised overgrowths of the cortical bone. They are particularly common in the maxilla (especially the hard palate—Torus Palatinus) and the lingual cortex of the mandible in the premolar region, usually bilaterally. These are felt as very hard, smooth, and discrete bony lumps. In some patients they can grow to a significant size. However they are benign and do not necessarily require treatment. Sometimes they can interfere with dentures or toothbrushing, in which case they can be removed. In many cases there seems to be a natural plane of cleavage between the exostosis and the underlying bone, making them relatively easy to chisel off. Their cause is unknown (Figs. 24.14 and 24.15).

24.1.9.9 Prominent Genial Tubercle

This is seen in the edentulous lower jaw, following complete resorption of the anterior alveolar bone. As a result, the tubercle which is normally hidden from view becomes more obvious, appearing as a hard swelling in the midline of the floor of the mouth. This can often be confused with a tumour. However on palpation it can be felt to be bony hard. Unlike exostoses these should not be removed—they provide attachment to some of the muscles of the tongue and floor of mouth.

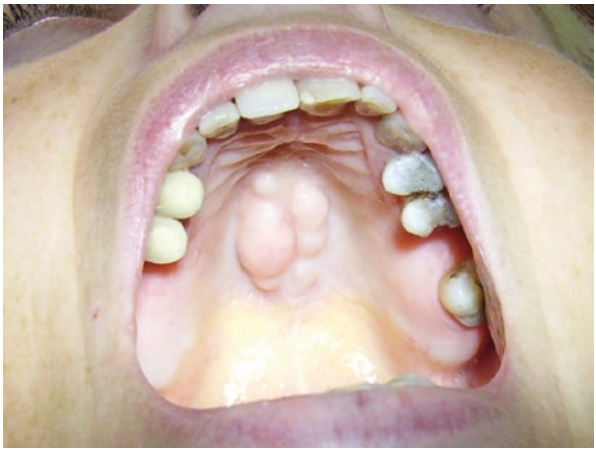


Fig. 24.14 Torus palatinus

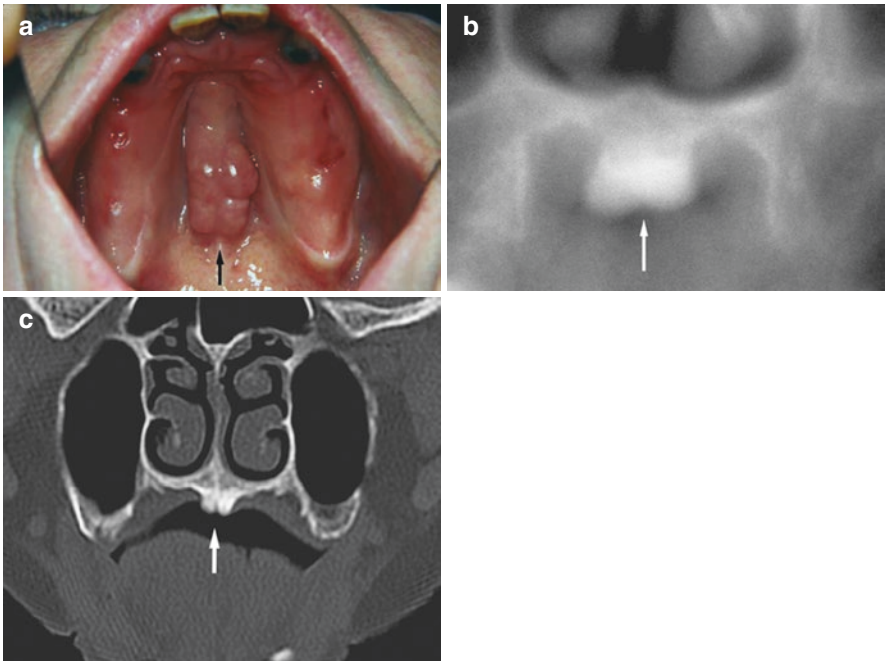


Fig. 24.15 Torus palatinus; painless hard swelling. (**a**) Clinical photograph shows large torus with normal mucosa in midline of palate (*arrow*). (**b**) Coronal tomography of same patient shows exostosis with thick layer of compact bone (*arrow*). (**c**) Coronal CT of smaller torus in another patient (*arrow*)

24.1.10 Abnormalities of the Teeth

Many varied developmental and acquired abnormalities, affecting the size, shape and number of the teeth exist. Most are rare. They include

(i) Supernumerary teeth/Hyperdontia (extra teeth). Supernumerary teeth are relatively common. When they occur in the midline between two central incisors, they are referred to as mesiodens. Extra teeth can sometimes be a diagnostic feature of a syndrome, the more common ones are (i) Cleidocranial dysplasia (CCD)—an autosomal dominant condition. Its main features include hyperdontia, small or missing clavicles, delayed closure of the cranial fontanelles and short stature and (ii) Gardner syndrome in which supernumerary teeth are seen in patients with intestinal polyposis and skeletal osteomas (Figs. 24.16 and 24.17).

(ii) Hypodontia (less than 6 congenitally missing teeth), Oligodontia (6 or more congenitally missing teeth) and Anodontia (no teeth). Congenitally missing teeth (hypodontia and oligodontia) are occasionally seen. In some patients there is a specific genetic basis. Perhaps one of the better known syndromes is hypohidrotic ectodermal dysplasia, in which ectodermal derivatives (hair, sweat glands, nails and teeth) are all affected. Scalp hair, eye lashes and eyebrows are sparse, nails are dystrophic and there is significant oligodontia. Diminished sweat glands leads to an inability to regulate body temperature (Fig. 24.18).

(iii) Taurodontism (bull teeth). This is a abnormality in the shape of the teeth. It is commonly seen in molars. It may exist in isolation or as part of as part of several syndromes.

(iv) Fusion is the joining of two adjacent tooth buds to form a single large tooth. Gemination (twinning) is an attempt by a single tooth germ to form two teeth.

(v) Dilaceration is an abnormal deviation in the root of a tooth, presumably caused by displacement of the tooth during root development. This can be seen following trauma to the unerupted teeth.

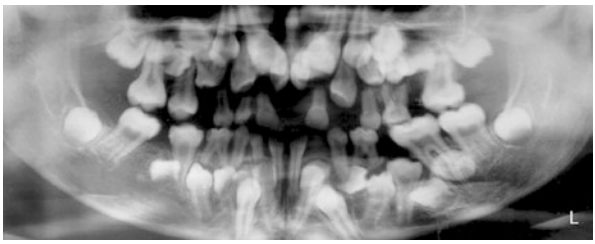


Fig. 24.16 Cleidocranial dysplasia is associated with multiple supernumerary teeth (panoramic radiograph). Affected patients often have hypoplastic or absent clavicles and have the flexibility to bring their shoulders close together in the midline (e.g., photograph)

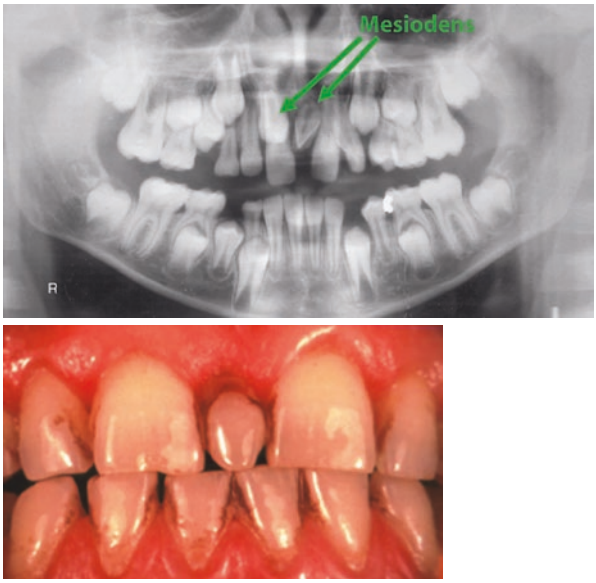


Fig. 24.17 Unerupted mesiodens *(arrow* on panoramic radiograph) is causing displacement of the adjacent regular central incisors. Uncommonly (photograph) there is room for the mesiodens to erupt and "function"

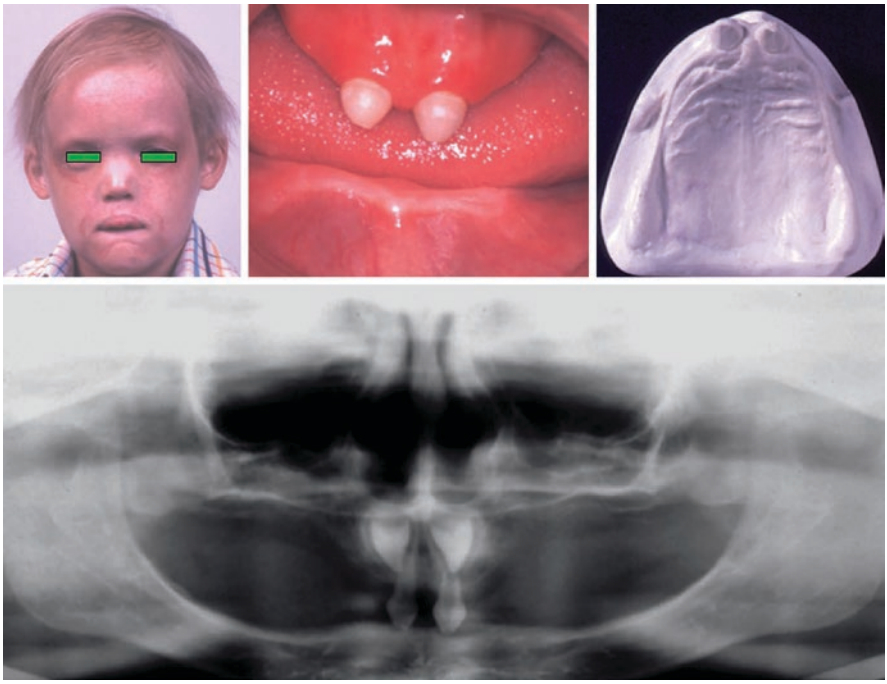


Fig. 24.18 Case of sex-linked hypohidrotic ectodermal dysplasia with severe hypodontia. The only teeth present arc the primary and adult central incisors, and these arc conical in shape. The child also demonstrates dry skin and sparse hair, including absence of eyebrows and eyelashes

(vi) Concrescence is the fusion of the cementum of adjacent teeth. Hypercementosis is the overgrowth of cementum on the root of a tooth. If the jaws are extensively involved, Paget's disease should be considered.

(vii) "Screwdriver" incisors and "Mulberry molars" are dental defects seen in congenital syphilis, caused by direct invasion of tooth germs by Treponema organisms.

(viii) Attrition is loss of the tooth surface due to normal wear. Some wear is normal (physiologic) but excessive amounts can occur in bruxism. Erosion is the chemical dissolution of a tooth structure, often as a result of regurgitation of gastric acid or excessive intake of acidic drinks. Abrasion is the wearing down of a tooth by mechanical forces. Excessive tooth brushing or nail biting are common causes (Fig. 24.19).

(ix) Internal and external resorption are where the surfaces of the roots are slowly resorbed locally. Osteoclasts in the dental pulp or in the bone resorb the calcified tissues. In many cases the precise cause is not known, although a history of trauma is often associated. One or many teeth may be involved. Skeletal bone can sometimes undergo a similar phenomenon, so-called vanishing bone disease or Gorham's syndrome. Radiographically, internal resorption may look like dental caries. Orthodontic treatment, cysts and tumours may be associated.

(x) Dentinogenesis Imperfecta (opalescent dentin) is an autosomal dominant condition affecting both deciduous and permanent teeth. These appear grey or yellow/brown with a broad crown or "tulip" shape. The enamel is friable and often chips, resulting in exposure of dentin. The teeth soon wear down. Several classifications exist. Amelogenesis Imperfecta is a group of inherited developmental defects involving enamel.

(xi) Staining of teeth. This is commonly seen in heavy smokers and people who eat or drink particular foods (coffee, tea). Fluorosis and tetracycline taken during tooth formation is also a well known cause. This is deposited in both teeth and bone.

24.2 Abnormalities of the Tongue

24.2.1 Depapillation/Atrophic Glossitis

The dorsal surface of the tongue is normally covered with numerous papillae. Depapillation is common and often associated with nutritional deficiencies, notably iron, folate or vitamin B12. Other causes include alcoholism, trauma, or friction from continued rubbing against a sharp tooth (where keratosis may also be seen). Usually there is a loss of the normal roughness on the dorsal surface. If there is associated inflammation, the term 'atrophic glossitis' is commonly used (Figs. 24.20 and 24.21).

In some patients, irregular-shaped patches of red depapillation appear to migrate over the surface of the tongue, over a period of time. This is referred to as a

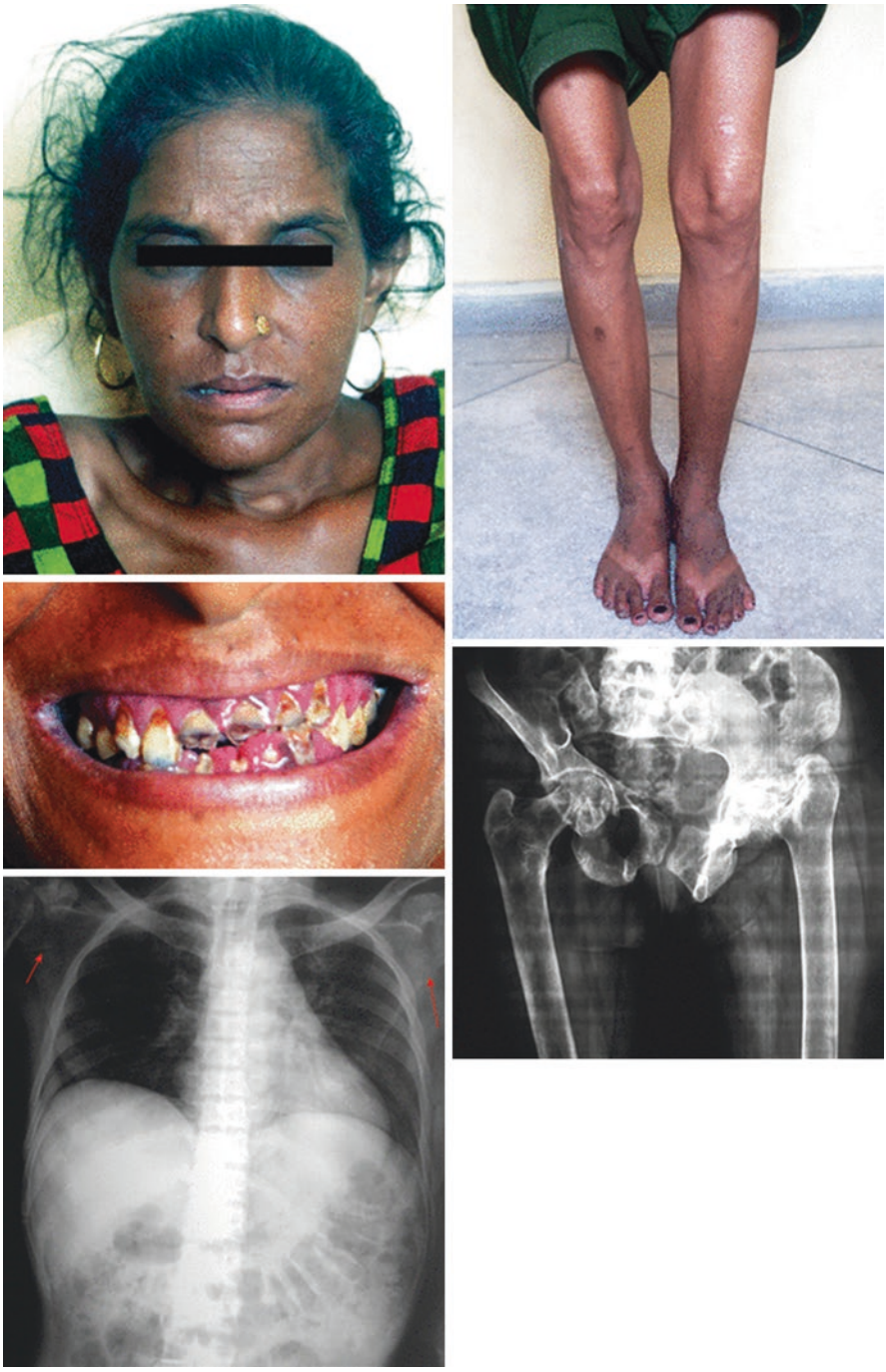


Fig. 24.19 Type I Dentinogenesis imperfecta is associated with Osteogenesis imperfecta which can affect any bone in the body resulting in bones that fracture easily, spinal curvature, loose joints and poor muscle tone

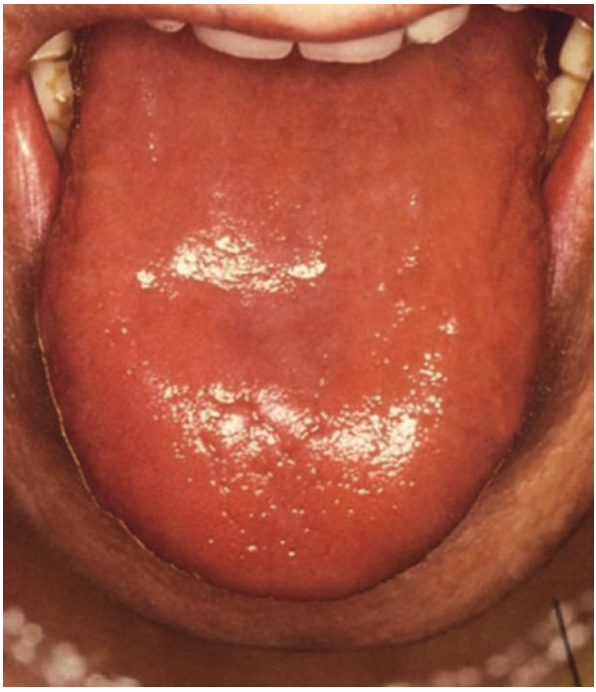


Fig. 24.20 Hunter's glossitis

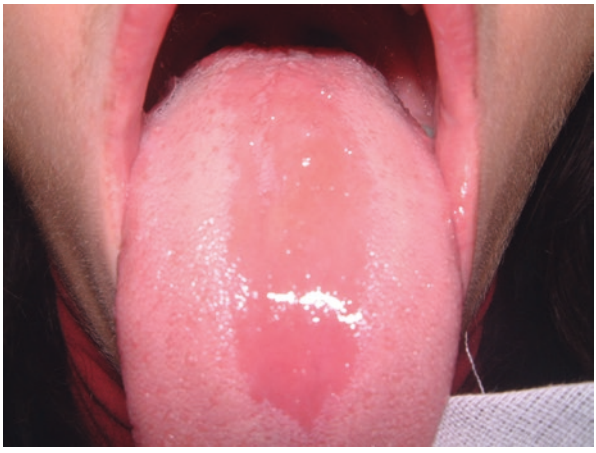


Fig. 24.21 Localised atrophy of papillae can be a normal finding, or associated with some deficiency states

geographic tongue or erythema migrans. The red patches are said to resemble a map, hence the name. The cause of geographic tongue is unknown although blood tests, to include iron, folate, Vitamin B12, magnesium and zinc should be performed to exclude any deficiencies. This is generally a clinical diagnosis and tends to be asymptomatic, with no specific treatment required. If painful, it may be treated with a topical anaesthetic gel or spray. Stomatitis erythema migrans (or areata migrans)

is a benign and unusual variant of geographic tongue in which similar looking lesions occur on other mucosal surfaces in the mouth. As with geographic tongue, these are usually painless and spontaneously resolve, only to reappear in a nearby location.

24.2.2 Hairy Tongue (Lingua Villosa)

Hairy tongue occurs as a result of elongation of the filiform papillae on the dorsum of the tongue. This is often a response to infections, fever, xerostomia and a variety of extrinsic substances, such as antibiotics and tobacco. It is rarely symptomatic although its appearance may bother patients. Diagnosis is usually clinical. Papillae can vary in length and colour. In some patients this can become quite impressive—hence the name. The matted papillae traps food debris and pigments from coffee, tea and other exogenous sources, together with bacteria and can then become stained, appearing red, brown black or other colours. Although candida can be found in some cases, it is usually a consequence, not the cause of hairy tongue. Patients may also complain of a bad taste in their mouths, or halitosis. Heavy scrubbing with a toothbrush should be avoided, as this will stimulate further keratinisation. Treatment involves reassurance, dietary advice, mouthiness and gentle tongue scraping. Sucking on a peach stone or gentle brushing may help to keep this under control.

24.2.3 Fissured Tongue (Scrotal Tongue, Lingua Plicata)

Fissured tongue is a benign condition which can be inherited or acquired. It is seen as varying degrees of grooves or fissures on the dorsum of the tongue. Some patients may complain of a burning sensation. The cause is uncertain, although nutritional and vitamin deficiencies have been suggested. If there are any indications of a systemic disease appropriate referral or laboratory tests should be carried out. Management is symptomatic, including oral hygiene. Fissured tongue can be seen in Melkersson-Rosenthal syndrome (facial nerve paralysis and granulomatous cheilitis). It is also seen in patients with Down's syndrome and can be a feature of Cowden's syndrome (multiple hamartomas and an increased risk of developing breast, thyroid, uterine and other cancers).

24.2.4 Median Rhomboid Glossitis

Median rhomboid glossitis was previously reported to be a congenital abnormality of the tongue, related to the persistence of the embryonic tuberculum impar. However, this has been questioned, since it is not seen in the newborn or infants. Lesions are sometimes associated with candida and disappear following antifungal treatment, suggesting a fungal aetiology. But uncertainty as to its cause remains.

The lesion appears as a midline atrophic, fissured area on the posterior dorsum of the tongue, just anterior to the circumvallate papillae. Diagnosis is usually made on clinical appearances. However if there is any question regarding the possibility of neoplasia, dysplasia or a granulomatous disease, a biopsy should be taken (beware ectopic thyroid tissue). Treatment usually involves a short trial of antifungal medication (Fig. 24.22).

24.2.5 White Sponge Nevus

White sponge nevus is a benign genetic condition with a hereditary element. However, it can also occur de novo without any family history. It is not associated with any other disease or condition and has no pre-malignant tendency. Clinical features range from mild, irregular epithelial keratosis to symptomatic, large mucosal projections or hairs. This is usually seen bilaterally on the buccal mucosae and lateral tongue. These features can be confused with leukoplakia. Biopsies may be performed to rule out dysplasia or malignancy. Treatment is often unnecessary.

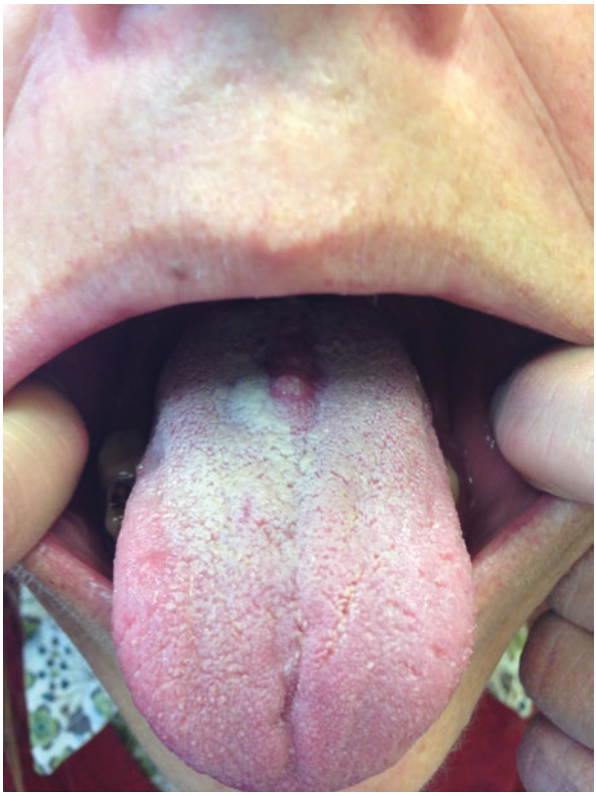


Fig. 24.22 Median rhomboid glossitis

24.2.6 Haemangiomas and Lymphangiomas

Haemangiomas often they appear as flat, sessile, or lobulated reddish-purple growths. They are usually soft to palpation and do not blanch to pressure. Lymphangiomas usually appear as sessile growths or swellings, which have the colour of the adjacent mucosa. Diagnosis is usually based on the history and appearance. In some cases, biopsy may be necessary. Imaging (CT/MRI) may be required, particularly if there are concerns regarding bony involvement and the possibility of future dental extractions. Management depends on the site, signs and symptoms. If lesions appear before puberty, they can usually be monitored—these are often self-limiting and can show regress in adolescence. Treatment is primarily surgical (laser, sclerosing agents and rarely embolisation/resection) (Fig. 24.23).

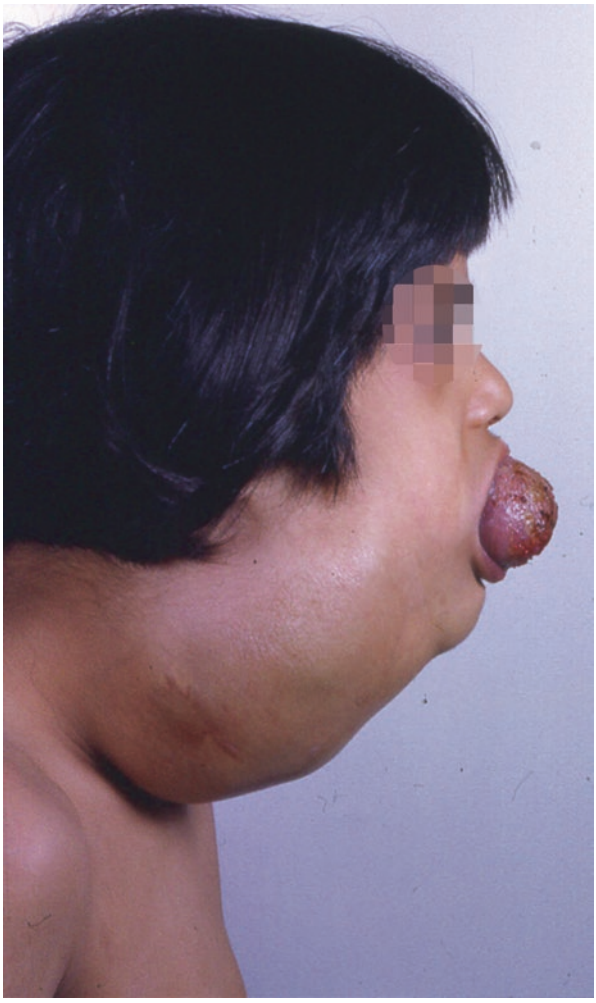


Fig. 24.23 Large mixed lymphangioma, haemangioma of tongue and oral cavity

24.2.7 Tongue Tie

This is a congenital abnormality. It is a small fold of mucosa tethers the tongue to the floor of the mouth. It may be noticed on the initial paediatric examination following childbirth. It is usually of no significance and many do not require treatment. It if interferes with feeding then a small incision can be made to separate the tongue from the floor of the mouth.

24.3 Halitosis (Fetor Oris, Bad Breath)

The most common cause of bad breath, or halitosis, is an excessive accumulation of oral bacteria, particularly gram negative anaerobes. Poor oral hygiene is the commonest reason. About 20% of the general population are reported to suffer from this to some degree. However, not all patients who think they have halitosis will have a genuine problem. Many have no halitosis when professionally examined. Of those patients who do have true halitosis, about 10% is caused by conditions involving the nasal cavity, sinuses, throat, lungs, oesophagus, stomach or elsewhere. Halitosis can therefore be a sign of infection (dental abscess, pericoronitis, sinusitis, tonsillitis, lung abscess), a pharyngeal pouch, or a large necrotic tumour anywhere in the aerodigestive tract. Usually these patients present with additional symptoms, including fever, lymphadenopathy and pain in the affected region. Rarely, the cause for halitosis may be an underlying systemic disease, such as bulimia, diabetic ketoacidosis, bowel obstruction or liver failure. Some of these disorders produce a characteristic odour.

(i) A faeculent smell can be suggestive of bowel obstruction, as faecal matter is regurgitated.
(ii) A sweet fruity or acetone smell signifies ketones, which suggest diabetic ketoacidosis.
(iii) A distinctive sweet faecal smell known as Fetor Hepaticus is a sign of liver failure and early hepatic encephalopathy.

Thus, halitosis may be a symptom of a serious illness. In all cases that cannot be explained by poor oral hygiene further investigation is necessary.

Halitosis related to poor oral hygiene is a clinical diagnosis and will often be accompanied by obvious plaque around the teeth, inflamed gums and a history of infrequent toothbrushing and flossing. Food packing (food retained between teeth) undergoes bacterial putrefaction and releases malodorous gases. Physiological halitosis may fluctuate throughout the day, following consumption of certain foods (garlic, onions, fish, and cheese), smoking and alcohol. In the context of a dental abscess, the patient will typically have a painful tooth, with associated swelling, discharge and pain in the surrounding gingivae. Halitosis associated with pericoronitis occurs following collection of plaque and bacteria under the operculum, resulting in pain and an offensive taste and smell. This will be swollen, red and inflamed,

sometimes with suppuration. An OPG will often reveal pathology associated with the offending tooth.

Sinusitis, pharyngeal pouch and other head and neck causes of halitosis are described elsewhere in tis book. Lung pathology can also present with offensive breath. This can vary simply from lower respiratory tract infections and pneumonia, to a lung abscess and empyema. Patients will present with respiratory signs, tachypnoea, reduced oxygen saturation and increased oxygen requirements. A Chest X-ray should be taken. "Bad breath" from repeated vomiting is also seen in patients with bulimia. Signs include be erosion of the palatal surfaces of the teeth, from acid attack.

Treatment of halitosis is dependent on its aetiology. In the absence of pathology the patient should improve their oral hygiene and brush twice a day, including interdental cleaning. Gently brushing or scraping the tongue may also improve the situation, as the tongue can harbour a heavy bacterial load. Antibacterial mouthwash will help to freshen the breath. Systemic disease such as diabetic ketoacidosis, bulimia, bowel obstruction or hepatic encephalopathy will require specific medical referral.

24.3.1 Oral Galvanism

This appears to occur when two or more dissimilar metals (usually in dental fillings) are bathed in saliva, or the two electrolytes of a battery come into contact with saliva (not to be recommended!). This produces an electric current, often associated with discomfort. It may also be accompanied by a metallic taste in the mouth. If significant, symptoms may be treated by replacing metallic amalgam fillings (or stop licking batteries).

24.3.2 Hypersalivation

Excessive production of saliva (ptyalism, or sialorrhea) can be due to increased production of saliva in the mouth or decreased clearance. True over production of saliva is very rare (causes include intraoral infections, foreign bodies such as new dentures and mercury poisoning). More commonly, patients are unable to swallow normal volumes of saliva effectively. Causes include Parkinson's disease, epilepsy and other swallowing disorders. It can also occur during pregnancy. Ptyalism can result in drooling, soreness and infections of the perioral skin. There are many causes, but most of these are uncommon or rare.

24.3.3 Excessive Production

(i) New dentures
(ii) Mouth ulcers

(iii) Oral infections
(iv) Pellagra (niacin or Vitamin B3 deficiency)
(v) Gastroesophageal reflux disease (water brash)
(vi) Gastroparesis
(vii) Pregnancy
(viii) Pancreatitis
(ix) Medications (clozapine, pilocarpine, ketamine)
(x) Toxins (mercury, copper, organophosphates, arsenic)

24.3.4 Decreased Clearance (Impaired Swallowing)

(i) Infections (tonsillitis, epiglottitis and mumps).
(ii) Fracture or dislocation of the mandible
(iii) Radiation therapy
(iv) Neurologic disorders (myasthenia gravis, Parkinson's disease, rabies, bulbar paralysis, hypoglossal nerve palsy).

If lip function and swallowing are normal, the complaint of hypersalivation is usually psychological. Treatment includes hyoscine patches or glycopyrronium. In severe cases botox injections into the salivary glands, chorda tympani transection, ductal rerouting, ligation of the parotid duct, or gland excision have been described with varying success (and complications).

24.4 Important Considerations When Taking a History

A clear and concise history is important when attempting to make a diagnosis, and the mouth, lips and teeth are no different in this respect. Occasionally the initial complaint may immediately suggest the diagnosis and following a detailed history this may be confirmed without the need for extensive investigation. But careful interpretation of symptoms is important. Some presentations, notably atypical orofacial pain, can easily be misdiagnosed and require careful consideration. If previous treatment has failed, the diagnosis should be reconsidered. Age, gender, ethnic group and occupation may also provide useful clues. Patients presenting with pain and altered sensation, should be asked about its site, onset, character, radiation, initiating and associated factors, aggravating and relieving factors, duration and severity. A relevant dental history is essential to diagnose dental pain or exclude the teeth as the cause of the patient's symptoms. Oral and dental pain can sometimes be vague and may conceal a more sinister aetiology. Any history of dental trauma or dental treatment may indicated a likely cause. Hot and cold sensitivity, or pain on biting also suggests a dental cause. Recent illness, infection, stress can sometimes precipitate symptoms. Neurological symptoms such as dysarthria, limb weakness, hemiplegia, tetany will indicate the need for careful examination and further investigations. If pain has arisen following dental treatment or extractions, infection and

fracture should be considered. Fever, swelling, pus, lymphadenopathy and restricted mouth opening should be looked for. Numbness, tingling, burning, shooting or electric shock pain suggest nerve injury, most commonly associated with removal of lower wisdom teeth. It can also occur following placement of an IAN block. With a dry socket the patient is likely to have severe pain but only mild swelling and no suppuration. Risk factors for this include smoking, poor oral hygiene, oral contraceptive pill, surgical extraction, past history of dry socket.

Changes in the oral mucosa need careful assessment as this can be a sign of many serious conditions. Swelling and lumps are common and may develop rapidly (infection or aggressive tumour) or slowly. Pain in a swelling related to meal times suggests salivary disease. Ulceration and blistering are common but can be disabling if very painful. Enquire about oral intake. Their number, regularity, duration, known precipitants and systemic symptoms including weight loss, malaise and joint, ocular, genitourinary and skin involvement will often help determine their cause. "Red flag" symptoms include rapid progression, loss of appetite, weight loss, restricted mouth opening and difficulty in swallowing. Bleeding from the mouth has many causes and can arise from sites beyond the oral cavity. The frequency of bleeding, timing known precipitants (following tooth brushing) can help make a diagnosis, but this symptoms frequently requires extensive investigation, including a CXR and endoscopy. Occasionally this can be a presenting symptom of a clotting disorder, haematological malignancy, or anticoagulant/antiplatelet overdose. Similarly, halitosis has many causes beyond the oral cavity so enquire about symptoms related to the chest/throat/sinus and stomach.

Systemic disease can occasionally present for the first time in the oral cavity. Common examples include anaemia, central cyanosis, Crohn's disease, autoimmune diseases and amyloid, but there are many more. The family history may provide useful clues (especially a history of cancer). Occupation, smoking, alcohol and elicit drug use should also be asked about. When considering a sinister lesion particular enquiry should be made regarding betal quid, alcohol and smoking, including smokeless tobacco.

24.4.1 Peri-Oral Tingling of Hypocalcaemia

This is a rare presentation, but hypocalcemia is a common derangement in both medical and surgical patients requiring intensive care. One of the earliest signs of hypocalcaemia is peri-oral tingling. Untreated this can progress to tetany (not tetanus) with life-threatening complications such as laryngospasm, seizures and potentially serious cardiac dysrhythmias. Causes of hypocalcaemia include hypoparathyroidism, following thyroidectomy, chronic renal failure, medication side effects and vitamin D deficiency. Transfusion of citrated blood and the effect of circulating catecholamines are also likely to contribute. Septic patients are reported to be at particular risk of hypocalcemia which has been linked to their bacteremic state and inflammatory mediators on parathyroid hormone production.

On examination there may be a positive Chvostek's sign in which percussion over the course of the facial nerve elicits facial twitches. This occurs because the nerve has become hyper excitable. This should not be confused with Tinel's sign which is used to monitor nerve recovery. Diagnosis of hypocalcaemia is made following blood tests, showing a low ionised calcium. Other biochemical abnormalities are also common including derangements in sodium, potassium, magnesium, phosphate, lactate and albumin. If identified, calcium can be replaced either orally or intravenously, with Vitamin D supplementation. Acute and life-threatening calcium deficit requires treatment with intravenous calcium. Chronic hypocalcaemia is commonly due to inadequate levels of parathyroid hormone or vitamin D, or due to resistance to these hormones. Treatment here focuses on oral calcium and vitamin D supplements, as well as magnesium if deficiency is present. All patients should be referred to an appropriate physician for investigation and not just given calcium supplements—there is no evidence on the impact of calcium supplementation on outcome in critically ill patients.

24.5 Examining the Oral Cavity and Its Associated Structures

It is an important skill for practitioners to recognise the structures in a normal healthy mouth and the variations that can occur. Abnormal appearances can then be recognised, and diagnosed successfully. The best way to examine the mouth is on a subject seated in a dental chair with overhead chair lighting and the use of a tongue spatula and a dental mirror where necessary. The entire mouth should be inspected regardless of the patient's complaint and reasons for attending visit—this is an good opportunity to look for early cancers, which would otherwise not be apparent until advanced. Detailed examination of the oral cavity must also include the relevant associated structures of the head and neck region, notably the cervical lymph nodes. Whilst in many patients a full head and neck examination will not be required (for example in a patient with accidental trauma to the lip), it is important not to assume a diagnosis too early. Tumours and infections in the mouth may spread quickly to the neck and overlooking broken teeth can result in complaints (and sometimes litigation). At a minimum, examination should include the oral cavity and its contents, the extent of mouth opening, palpation of the temporomandibular joints, assessment of facial symmetry and palpation of the lymph nodes in the neck. A methodical and through approach is needed as there is much to note. With time and experience many oral lesions may be diagnosed by pattern recognition and "spot" diagnoses become easier. Any lesion that is encountered must be assessed carefully, noting its key features. Two dental mirrors, a bright light, some dental probes and a tongue depressor may be all that is required.

Start extraorally, looking for asymmetry and signs of obvious injury or disease. Palpate the temporomandibular joints and assess mouth opening. All muscles of mastication and temporomandibular joint should be palpated for tenderness. Ask the patient to open and close their mouth several times to evaluate any limited opening or deviation. Quickly palpate the head and neck to identify any tenderness,

masses and lymphadenopathy. Palpate the lymph nodes in the scalp, parotid region and neck. Oral examination starts with the lips, then intraorally, looking inside the lips, slowly moving clockwise around the oral cavity. If patients wears dentures these should be removed to allow thorough examination. Dentures should be checked for fit, general appearance and hygiene—they are often a cause of several oral problems. Gently stretch the mucosa to look at its surface. Two dental mirrors, one in each hand, make excellent retractors that patients tolerate well. They can also be used to reflect light and make visualising the deeper recesses easier. Saliva may present visualisation of the soft tissues and teeth and so suction should be available, or use gauze to dry the surfaces for examination. Continue to assess the buccal mucosa, then proceed to the floor of mouth and then the palate. Any intraoral lesion should be noted with regard to its to size, extent, thickness, colour, texture, consistency, any coatings, tenderness and friability (Fig. 24.24).

24.5.1 The Lips and Cheeks

Look at the inner lining of the lips, checking mucosal integrity and looking for abnormal discolouration. Look for signs of actinic (sun) damage, especially on the lower lip. This is often seen on the sun-exposed aspects, just above the vermillion. This surface of the lips is normally dry and slightly keratotic to varying but minor degrees. Note any non-healing lesions, these may be dysplastic or neoplastic. Erythema, cracking and crusting at the corners of the mouth (angular cheilitis) may be associated with a number medical problems. Evert the lips and look the lining labial mucosa. This surface is often slightly irregular due to the presence of the minor salivary glands. These can be felt like tiny son cobblestones, but otherwise

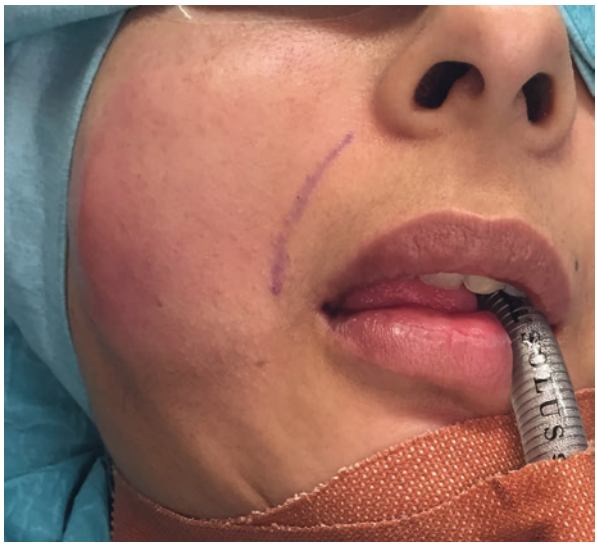


Fig. 24.24 Swellings require palpation to determine extent and consistency

the mucosa should feel smooth, easily stretched, soft and moist. The lower lip is a common site for mucocele, abnormal and physiological pigmentation, polyps, ulcers and haemangioma. Lumps in the lower lip are common but usually benign. Lumps in the upper lip are less common and should be viewed with concern—tumours of the minor salivary gland are more common in the upper lip than lower. A crude but useful guide is the 90% rule—90% lumps in the lower lip are benign, whilst 90% of lumps in the upper lip are likely to be tumours. The percentage may not be completely accurate, but its helps convey the importance of anatomical site.

Assess the surface of the buccal mucosa in the same way, looking for any lumps, ulcers etc. or change in colour. The normal colour is often described as "salmon-pink", but variations in pigmentation, vascularity and keratinisation are common. Pigmentation can be patchy and depends on ethnicity. It can sometime be linear and follow patches of chronic trauma. Sometime the mucosa appears grey/milky-white when stretched but returns to normal appearance—this is referred to as leukoedema and should not be confused with thrush (candida). The linea alba is a horizontal whitish ridge in the mucosa that is seen running along a level parallel with the occlusal plane. This is common and arises as a result of chronic irritation caused by movement of the teeth on the mucosa (cheek biting, cheek sucking). It is benign. Fordyce spots may be prominent and numerous. They are tiny collections of ectopic sebaceous glands. The orifice of the parotid gland duct can be seen as a small raised flap of mucosa opposite the second maxillary molar tooth. Gently milk the parotid gland and assess the duct orifices patency and salivary flow, noting the quality of saliva (e.g., pus, frothy, blood stained, clear). Severe dryness of the mouth suggests xerostomia. This has many cases and is discussed later in this chapter. Finally palpate the mucosa for induration and scarring—not all pathology may be visible, yet it may be easily palpable. Minor salivary glands and Fordyce spots may impart a granular texture to the buccal mucosa. This is crucial part of the oral examination.

24.5.2 The Tongue and Floor of the Mouth

The tongue is much larger than can be appreciated by intraoral examination. This is because it extends into the oropharynx. Even with the tongue fully protruded, it is not possible to see its full extent although careful use of a dental mirror can reveal some additional features. Assessment of the tongue can be tricky in some patients. To ensure complete examination the tongue should be protruded and moved from side to side. It should then be raised to the roof of the mouth to allow visualisation of the floor of the mouth. If necessary, gently grasp the tongue with gauze and move it side to side. The dorsum (upper surface) is covered by numerous small hairlike papillae, interspersed among which are dozens of mushroom-shaped fungiform papillae. These contains one or more taste buds. Atrophy and glossitis results in loss of these papillae and a shiny red appearance of the dorsal surface. This may be the result of a number of causes, notably nutritional deficiencies, some types of candida infection and other mucocutaneous diseases. Alternatively, coating or a 'hairy" tongue may be seen. This is due to hyperplasia of the filiform papillae with

subsequent accumulation of keratin, food debris and extrinsic stains. Patients may complain of bad breath. The tongue may also show an irregular keratotic pattern, referred to as a 'geographic tongue' (erythema migrans). Posteriorly, at the junction of the anterior two-thirds and posterior third of the tongue are the circumvallate papillae and clumps of lymphatic tissue. The circumvallate papillae normally are 8–12 in number and are arranged in a V-shaped pattern, just anterior to the foramen cecum (the developmental remnant of the thyroglossal duct). Like the fungiform papillae, the circumvallate papillae also contain numerous taste buds. Fissuring of the dorsal surface tongue is common and is generally of no clinical consequence. Palpate the surface of the tongue, slowly working your way to the back of the mouth, being careful not to induce gagging. The lateral borders of the tongue should be carefully examined by moving the tongue to the side. Note any difficulty or pain on movement and look specifically for ulcers. The side of the tongues no papillae and the mucosa is smoother. Posteriorly are lymphoid aggregates and lingual tonsil. The under (ventral) surface of the tongue is then inspected by asking the patient to raise their tongue to the roof of the mouth. The veins and lingual frenum are prominent here. In some patients the frenum extends to the tip of the tongue, restricting movement—"tongue tie". Tongue ties are sometimes removed in childhood if they cause problems, but contrary to popular belief they usually do not result in speech impediments (Fig. 24.25).

The lateral surface of the tongue merges into the floor of the mouth. This is also lined with thin smooth lining oral mucosa similar to that found on the under surface of the tongue.

On the inner surface of the mandible (lingual cortex) tori may be found. These are symmetrical bony lumps and are not significant. The orifices of the submandibular glands (Wharton ducts) are seen as 2 midline lumps on either side of the lingual frenum. Again, massage the associated gland and look for clear saliva, pus and stones. This saliva here is usually more viscous than from the parotid gland (due to

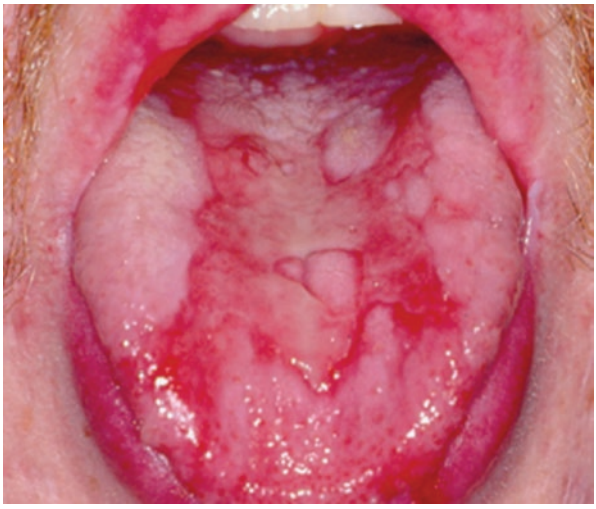


Fig. 24.25 Tongue lesions in chronic graft versus-host disease in a 25-year-old patient treated with bone marrow transplantation 7 months earlier

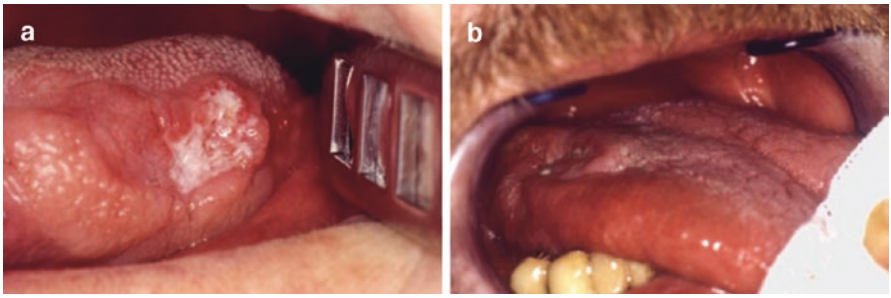


Fig. 24.26 Tongue cancer. (**a**) exophytic with little deep infiltration; (**b**) infiltrative with large submucosal extension

the higher mucous content). Special importance should be given to the junction of the ventrolateral surface of the tongue and the floor of the mouth—these are common sites for squamous cell carcinoma. Posteriorly, this site is sometimes referred to as the "coffin corner" (Fig. 24.26).

In most cases the airway will be clearly patent. However it is good practice to develop a routine of noting the degree of mouth opening and any degree of restriction (trismus). Assess tongue movement and look for swelling of the floor of mouth swelling. Ask the patient if they have had any change in speech or pain on swallowing. Inspect the posterior airway space. All this information is useful to the anaesthetist if a general anaesthetic is required.

24.5.3 The Palate and Oropharynx

The palate is divided into the hard palate anteriorly and the soft palate posteriorly. Direct inspection of the hard palate is best accomplished with an intraoral mirror. The hard palate is covered by relatively thick keratinised mucosa. It is pale red in colour and looks similar to the attached gingiva. It is usually less red than other oral mucosal sites because of its thicker keratin layer. Anteriorly is covered by multiple fibrous ridges, the palatal rugae. The incisive papilla, overlying the lower end of the nasopalatine duct, is just behind to the upper central incisors. Exostosis may occasionally be seen in the midline of the hard palate—a palatal torus. These are usually incidental and of no clinical importance. The hard palate contains numerous minor salivary glands. Their openings can sometimes be seen, with a tiny drop of saliva, or they can become inflamed. Minor salivary gland tumours can also arise here. The soft palate is non-keratinised and lighter in colour. It should move symmetrically on swelling and speaking. Note any swellings and ulcers (Fig. 24.27).

The soft palate is discussed in the chapter on the throat. In contrast to the hard palate, this is non-keratinised and more 'fleshy-looking' being salmon-pink in colour. It can be seen easily by depressing the tongue and asking the patient to say "Ahhh." It should appear symmetric. Any deviation or displacement should be noted as this may indicate a tumour or neurologic problem. Note also the appearances of

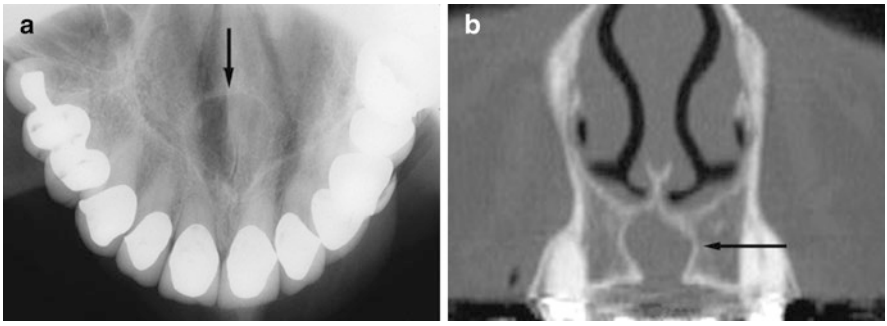


Fig. 24.27 Incisive canal cyst maxilla; 60-year-old male with incidental finding at routine dental radiography. (**a**) Intraoral occlusal view shows round radiolucency in midline with sclerotic border (*arrow*) and normal apical periodontal ligament. (**b**) Coronal CT image shows radiolucency with sclerotic border (*arrow*)

the throat and tonsils. The tonsillar pillars can be seen by moving the tongue to each side and asking the patient to say "ahh." Tonsillar crypts are very vascular and appear more reddish than the surrounding tissues. Accumulations of food and debris in this area may result in local irritation and halitosis. Accessory lymphoid tissue is common.

24.5.4 The Teeth and Gums

These will usually need to be assessed by the patient's dentist. From a general medical perspective a more limited examination is required, looking for obvious signs of mobility, decay, trauma and present or missing teeth. Starting in the upper right quadrant, work clockwise. The gingivae should be examined with the mouth partially closed and the lips retracted. The attached gingiva (adjacent to the crowns) is paler because it is more keratinised (cf hard palate). It is therefore usually firm, slightly stippled, and closely attached to the underlying bone. Further away from the gingival margin, the non-keratinised alveolar mucosa passes into the vestibule. This is redder and more pliable. The attached gingivae is frequently pigmented. There are many causes for changes in the appearance of the gingivae, most commonly gingivitis, but also as a result of systemic diseases. The gingiva is also a common site of some mucocutaneous diseases (such as lichen planus, mucous membrane pemphigoid, pemphigus vulgaris). Healthy gums thus look pale pink and are tightly adherent. Note any swelling, bleeding or pus from around the necks of the teeth or their sockets. Swollen, friable, ulcerated, bleeding gums usually indicate periodontal or dental disease. The gingival crevice should then be probed. Mucosal nodules, especially those on the gingivae should also be gently probed to identify any sinus or fistulae. Check each tooth for tenderness by lightly tapping each one in turn. Tenderness to percussion usually indicates infection at the apex of the tooth. Teeth may be mobile, but this may be longstanding. Teeth may also

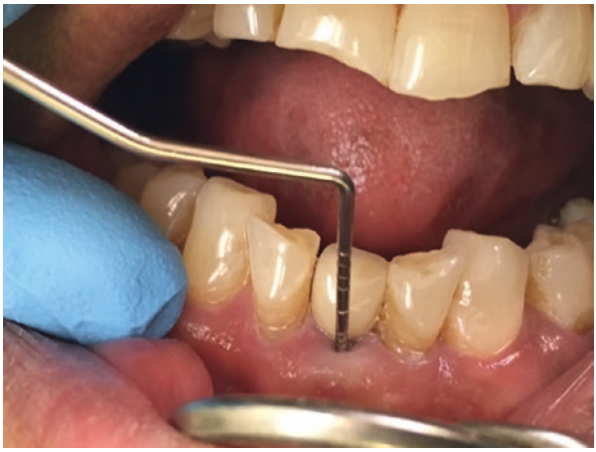


Fig. 24.28 Periodontal probing for pocketing and signs of inflammation or infection

appear worn down, most likely due to grinding. However this may also be a sign of acid reflux. Missing teeth following trauma should be documented and need to be accounted for. Gross decay of the teeth usually appears as discoloured cavities, however decay involving the interproximal surfaces of the teeth may not be obvious without X-rays. Decay around the necks of the teeth may be a sign of dry mouth or acid relux. Root surface caries are also commonly seen in elderly patients with gingival recession (Fig. 24.28).

If one or more teeth are thought to be the cause of symptoms their vitality should be checked. To determine whether the pulp (nerves and blood vessels) are alive, several tests may be performed:

(i) Cold Testing: Cold compressed air or ethyl chloride is applied to the tooth. The patient is then asked to indicate if they become aware of the stimulus. Normal teeth respond quickly but discomfort settles within a few seconds. A tooth with a necrotic pulp will not react as the nerve endings are non-vital. Inflammation in the pulp (pulpitis) will often show result in sensitivity that lasts several minutes before becoming to a throbbing pain.
(ii) Electric Pulp Testing: This device applies an increasing electric current to the tooth until it starts to tingle. A quantitative value can therefore be noted. Non-vital teeth will not respond whereas vital teeth will produce a response with low levels of current. A progressive change in reading over time may indicate a loss of vitality.
(iii) Laser Doppler flowmetry (LDF). This is a relatively new pulp testing device. A laser beam is directed at the coronal aspect of the pulp. Light is scattered by the pulp blood cells that produces a Doppler frequency shift. In theory pulp vascularity can be detected.
(iv) A tooth sleuth (hard plastic instrument) can be placed on the cusp of a sore tooth and the patient instructed to bite on it. Pain on release from biting can indicate a hairline crack or fracture within the tooth.

24.6 Investigating Symptoms and Signs

24.6.1 Laboratory Tests

These may be required in a number of oral condition.

(i) Recurrent aphthous stomatitis
(ii) Persistently sore or dry mouth
(iii) Any oral lesions with an atypical history or that is resistant to treatment
(iv) Sore mouth or dry mouth with no mucosal changes
(v) Candidosis

Swabs are not routinely taken for oral lesions but may be indicated in cases of suspected streptococcal pharyngitis, unusual infections, or if there is pus discharging form somewhere. Cultures may later be helpful if there is a poor response to antibiotics. Actinomycosis requires long term antibiotics and therefore sensitivities must be confirmed. If the patient has already been commenced on empirical antibiotics at the time the swab is taken, these should be documented on the pathology request. Viral polymerase chain reaction cultures may be taken in some cases of painful ulcers. However false negative results are common. Caution is therefore required in interpreting results. Candida is also a normal commensal in around 90% of the normal population and therefore a positive growth for *Candida albicans* is indicative of carriage rather than infection. Fungal culture is therefore rarely requested unless an atypical infection is suspected. Cytology may also provide helpful information in the diagnosis of Candida and HSV infections.

Anaemia is often associated with a number of oral symptoms. An elevated white cell count is usually indicative of infections and inflammation and if very raised may suggest infection is spreading throughout the fascial spaces. A rise in the neutrophil count is usually seen in bacterial infection, acute illness, myeloid leukaemia and steroid therapy. Lymphocytosis is seen in viral infection, sepsis, bone marrow failure and some chemotherapeutic agents. A rise in eosinophils is seen in parasitic and fungal infections and lymphoma. Acute or chronic inflammation with show a rise in C-reactive protein. A rise in CRP above 100 is typical for systemically unwell patients.

An abnormal platelet count has a number of causes. A significant reduction may cause spontaneous oral bleeding, or excessive bleeding following a tooth extraction and is not uncommon. If the platelets are less than 40, haematology advice should be sought before an elective procedure is undertaken, or if the patient presents as an emergency. If a patient presents with excessive or spontaneous bleeding from the gingivae blood should be taken urgently. If the FBC reveals a pancytopenia with blasts, leukaemia is the likely diagnosis, and urgent haematology referral is then required. An abnormal coagulation screen may also signify an underlying bleeding disorder. The prothrombin time (PT) is a measure of the extrinsic clotting pathway. This should be measured unless the patient is on warfarin, in which case the INR (international normalised ratio) is more useful. If the PT is raised possible causes include anticoagulant use, liver failure, DIC, sepsis, deficiency of clotting factors or

heparin use. Activated partial thromboplastin time (APTT) is a measure of the intrinsic clotting pathway and may be useful, unless the patient is receiving heparin. If the APPT is raised causes include heparin use, haemophilia, von Willibrand disease, DIC, sepsis, deficiency of clotting factors, liver disease and anticoagulant use. If the INR is less than 2.0 patients are unlikely to have significant bleeding following surgery (including extraction). An INR of greater than 3.0 will usually require haemostatic measures to stop post-operative bleeding.

Urea and creatinine assess renal function. With severe oral infections or other disorders resulting in dysphagia the patient may become dehydrated. This will be reflected by an increase in the urea and creatinine. Urea may also increase in acute illness. Creatinine may increase following any disease, injury or procedure which results in muscle injury. Zinc and magnesium levels are also useful in the assessment of oral ulceration and burning mouth, although they are rarely required in the acute setting. In some patients correction of deficiencies have resolved some oral lesions and pain. Liver function tests are required in all patients with a history of alcoholic liver disease or viral hepatitis. They should also be taken in any bleeding patient where there is no obvious cause. In alcoholics there will be a rise in the Gamma GT, ALT, AST, MCV and a reduction in platelets. Liver dysfunction will also increase the prothrombin time.

Haematinic screening is particularly useful in patients with recurrent aphthous ulceration, burning mouth syndrome or atrophic glossitis. A low haemolglobin may indicate anaemia. This may be microcytic, with a low MCV or macrocytic with a high MCV. Causes of microcytic anaemia include low iron and ferritin, thalassemia, haemoglobinopathies and sideroplastic anaemia. Causes of a macrocytic anaemia include folate/Vitamin B12 deficiency, alcoholism, liver disease, thyroid disease, myelodysplasia and drug (particularly those that lower folate such as methotrexate). Ferritin, Vitamin B12 and folic acid levels should therefore be measured. However Ferritin is an acute phase protein which can be falsely elevated in acute inflammation. A CRP should therefore be taken as well to exclude false positive results. Serology may be indicates in some infectious diseases (notably syphlysis, gonorrhea, tuberclosis), viral antibodies for suspected HIV and EBV, or with suspected autoimmune disease (SLE, rheumatoid artheritis, ceolic disease, sjogren syndrome). Some intra oral lumps can be investigated using a fine needle aspiration under ultrasound guidance. However tissue biopsy is generally the gold standard for oral lesions (incisional biopsy). Tests include histology and direct fluorescence antibody test.

24.6.2 Plain Films

Common films used in dentistry include the Orthopantomogram (OPG/OPT), posterior anterior films, bitewings X-rays, long cone periapical films and occlusal films. In an emergency department setting these may not all be available. The orthopantomogram is an excellent screening film as it includes all the teeth and much of the jaws in one film. This is discussed further in the chapter on the lower jaw. OPGs are often requested in most emergency departments, when looking for dental fractures,

avulsions and subluxations. However the images do not provide fine detail and are unevenly magnified. Alternatively, long cone periapical views provide more detail of specific teeth, especially anterior ones. Ideally, the gold standard for detecting any fracture is two films at right angles, but this is not possible with teeth. An OPG will usually detect large caries, periapical disease, jaw cysts, abscesses and fractures. Interpretation usually requires experience, as overlapping normal anatomical opacities and lucencies (such as the cervical spine, hyoid bone, maxillary sinus, mental foramen and submandibular depression) can all be mistaken for cysts or dental abscesses. Gas shadows between the tongue and soft palate can also be confused with fractures of the posterior mandible. In children an OPG can help to determine the state of any unerupted permanent dentition. If a mandible fracture is suspected a PA plain film is also required (Figs. 24.29 and 24.30).

Long cone periapical films are X-rays which are limited to 2 or 3 teeth. These are commonly taken in dental practice, although many hospitals now also have this expertise. They are useful in the assessment of root morphology and in detecting bone loss in the alveolar process. Occlusal radiographs may also be used when trying to determine the location of any unerupted or impacted teeth or submandibular duct stones. Bitewing radiographs are rarely used outside the dental setting (Fig. 24.31).

24.6.3 CT and MRI

These are useful in determining the presence and spread of infection and malignancy. They are commonly used in other areas of the head and neck, or if oral disease is suspected to have extended into the neck. If the patient shows signs of a deep fascial space infection, or is at risk, CT may be useful to determine spread throughout the neck and into the mediastinum. Cone beam CT scans, with the lower

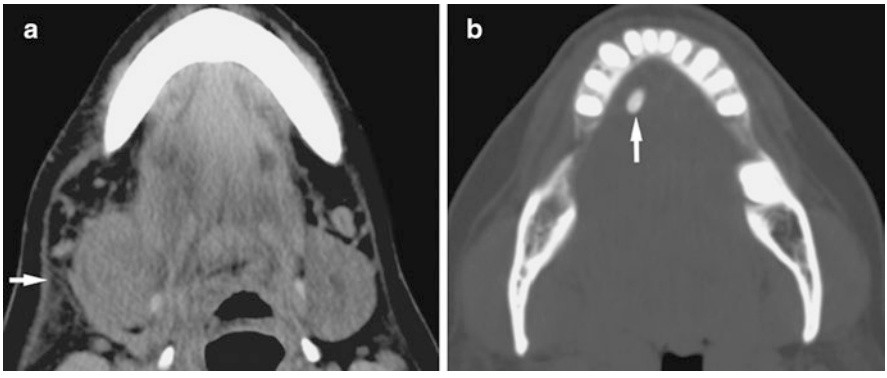


Fig. 24.29 Submandibular sialoadenitis due to duct stone; 59-year-old female with right submandibular swelling. (**a**) Axial post-contrast CT image shows enlarged submandibular gland with stranding and reticulation of periglandular fat (arrow). (**b**) Axial CT image shows stone in anterior part of Wharton's duct (*arrow*)

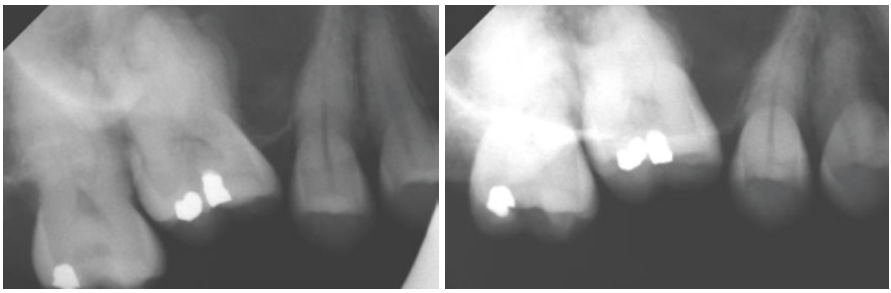


Fig. 24.30 Dental X-rays need to be taken carefully. Images must be sharp and of appropriate penetration in order to be diagnostically useful

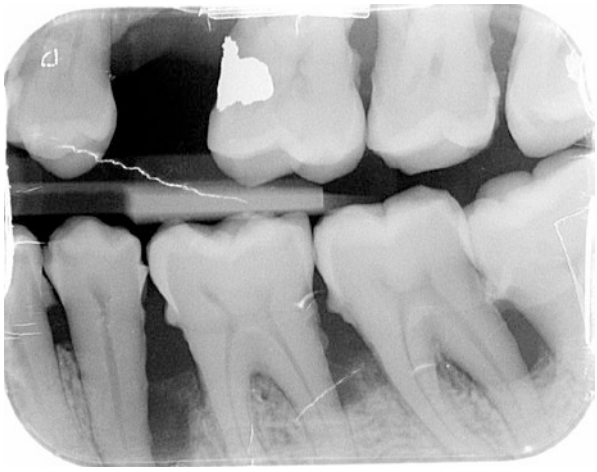


Fig. 24.31 Bite wing radiograph (with artefacts)

radiation dose are increasingly being used to assess oral conditions, notably the proximity of the inferior alveolar nerve to suspected pathology or buried teeth. The benefits of CT over plain film are self evident, but requires a co-operative patient. MRI is better suited for soft tissue pathology and is commonly used as first line investigation in oral malignancy. Many hospitals now have protocols in the assessment of cancer. Ideally imaging should be obtained before biopsy—if biopsy is undertaken first bleeding and reactionary inflammation at the biopsy site and regional lymph nodes may be misinterpreted as signs of malignancy. However this sequence may not always be possible.

24.6.4 Ultrasound

This is useful for determining if a swelling is a drainable collection, or simply swollen tissue. It is also useful to distinguish an enlarged lymph nose from a salivary gland (submandibular). Intraoral ultrasound is now also available and can be used to guide FNA.