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Oral Anatomy Pertinent to the Dentist

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Abstract

The oral cavity is a common site for mastication, deglutition, and respiration, phonation with different health specialties working within or in close proximity to it. The purpose of this article is to present clinically applied oral anatomy with the identification of the spectrum of normal and abnormal findings. In addition. The relation between the oral cavity and neurological/ophthalmic complications is also discussed.

Keywords: Oral mucosa; Pigmentary lesions; Diastema; Ankyloglossia; Maxillary sinus; Infratemporal fossa; Pterygoid venous plexus; Pterygopalatine fossa; Maxillary nerve; Maxillary artery; Lingual nerve; Mylohyoid nerve

Introduction

The oral cavity forms the first portion of the digestive tract and hence the entry to the whole body. It extends anteriorly from the oral fissure (which is formed by the upper and lower lips meeting at the commissures) to the fauces marking the boundary with the pharynx posteriorly. The oral cavity has important communications with the nasal cavity, maxillary sinus, orbits and even the cranium. This connection can be either direct via bony canals or indirect through vascular anastomosis. Various functions associated with the oral cavity include mastication, deglutition, phonation, respiration and facial expression. These functions are affected in case of structural or pathological abnormalities. Therefore, the oral cavity is a common anatomical location where various procedures are performed by dentists, otolaryngologist and oral and maxillofacial surgeons. Other specialties like anesthesiologists, pneumologist and gastroenterologists need the oral cavity as a port during their different procedures. In addition, the oral cavity is often viewed as a reflection of general health as many manifestations of systemic diseases manifest in the oral region before systemic ones. Knowledge of the normal anatomical structures in the oral cavity is therefore required by many health care professionals. The oral cavity is divided into the vestibule and the oral cavity proper. The aim of this article is present clinically relevant anatomy of the oral cavity along with common conditions involved.

Oral Mucosa

Mucous membranes consist of epithelium covering connective tissue which in turn is made of lamina properia and reticular layer. The epithelium is keratinized stratified squamous epithelium which loses keratinization at the areas of lining mucosa. Attached or masticatory mucosas is keratinized and have lamina properia that have numerous papillary projections. The submucosa is the loose layer of CT under the mentioned layers which contains: blood vessels, muscles, fat, glandular tissue, and loose CT. The submucosa contains deep vascular plexus that send branches to the papillary layer forming the secondary or superficial plexus close to the basement membrane of the epithelial layer [1]. The general arrangement of blood supply is such that blood vessels travel along the buccal and lingual vestibules towards the gingiva and run from posterior to anterior direction [2]. Due to the posterior anterior orientation of blood vessels, it probably is better to avoid distal releasing incisions during flap surgery to avoid healing complications. The described vascular distribution would leave the mid crest of the alveolus relatively avascular. Therefore, crestal incisions may heal slowly and may be involved in wound dehiscence.

The normal mucosal color varies between pale pink to dark red depending on the type and thickness of the overlying epithelium and the number and size of the vascular content. In general, discoloration is related to vascularity, hemoglobin concentration and the presence of endogenous pigments like hemosiderin and melanin or foreign bodies [3]. Anemic patients are generally pale due to reduced concentration of hemoglobin in blood. Other features include dyspnea and fatigue. Anemia also causes epithelial atrophy due to changes in the basic metabolic demands by keratinocytes which leads to glossitis, angular cheilitis, oral ulceration and mucosal pallor [4]. Diagnosis of anemia is made by measuring hemoglobin concentration and serum ferritin. Anemia may be associated with congestive heart failure, dizziness, apathy and cognitive impairment altogether increasing the risk of falls, morbidity and mortality.

Hemosiderin is a product of hemoglobin breakdown and causes bluish-gray pigmentation. In hemochromatosis which is autosomal recessive disease of increased iron absorption, oral pigmentations are commonly found at the hard palate and gingiva. Hemochromatosis may be associated with liver cirrhosis, bronze diabetes and heart failure [5]. Melanocytes are present in the basal layer of oral epithelium and many pigmentary conditions are attributed to either their increase function or number (Table 1). Melanocytes produce and store melanin in intracellular vesicles called melanosomes. Melanin is produced to protect cellular DNA from ionization radiation. Other sources of color change of oral mucosa include ingestion of heavy metals, drug side effects and foreign bodies [6,7].

Vestibule

The vestibule is the U-shaped space between teeth and cheeks. It is where the mucous membrane reflects from cheeks or lips to the alveolar mucosa and gingiva. Important contents of the vestibule include freni, Stenson’s duct orifice, lina alba and Fordyce granules.

Freni are folds of mucous membrane seen across the vestibule attaching the lips and cheeks to the alveolar mucosa, gingiva and periosteum [8,9]. Variable contents may be observed histologically including loos connective tissue, elastic fibers and in about third of the times, portion of adjacent skeletal muscle fibers [10]. Fibers of orbicularis oris are present in median freni while buccal freni may contain fibers of levator anguli oris and depressor anguli oris in the maxilla and mandible, respectively. Freni have prominent position which allows easy inspection and examination. Abnormally positioned freni are those that extend their attachment to the level of the interdental papilla or even to the other side of the alveolar ridge [8]. A simple clinical test includes pulling the lip and observing the blanch or gingival retraction that occur. Abnormal size or attachment level have clinical significance in gingival recession and mucogingival defect [11]. In addition, tight freni pull the gingival sulcus during mastication causing trauma and enhancing plaque accumulation. Muscle attachment may also contribute to flap retraction after suturing causing wound dehiscence. Prominent upper midline frenum prevents midline space closure causing persistent diastema and interrupting the eruption of the upper lateral incisors [12]. Frenal abnormality may also be one of the manifestations of syndromes, for example: Ehler’s Danlos and Ellis-van Creveld syndromes [13].

The parotid papilla or the orifice of parotid duct, after it pierces the buccinator, is located next to upper 6 or 7 and located about 3.56 mm above the buccal cusps of upper molars [14]. Stenson’s duct stone may associate with pain and swelling in the parotid gland. Clinical examination may reveal pus discharged from the duct or frankly show the associated stone at the orifice. Aberrant location of Stenson’s duct may confuse the clinician between buccal space infection and parotitis [unpublished case report].

Lina alba is a whitish raised line on the inner aspect of cheeks and at the level of the occlusal plane. Its extension is from the commissures to the molar area. The whitish discoloration along this line is attributed to keratosis which may be a response to parafunction. Lina alba may be present in as much as 5-7% of the population and other than correlating to parafunction or TMD, it may be considered as a normal finding [15,16].

Fordyce granules are sebaceous glands with no hair follicles and commonly found in the lips, cheeks and genitalia [17,18]. They appear as separated or coalesced yellowish granules and become obvious at puberty possibly in response to androgenic hormones. These granules are considered normal, but unilateral distribution has been attributed to skin hypopigmentation [18]. Cosmetic concerns indicate their removal [19].

Oral Cavity Proper

The area in the oral cavity from the vestibules to the oropharynx constitutes the oral cavity proper and contains the teeth, hard and soft palate, tongue and the floor of the mouth.

The hard palate is formed by fusion of the horizontal plates of the maxilla with the premaxilla in a Y-shaped suture at the midline. It is covered by tight keratinized epithelium which is thinnest at the midline and becomes thicker at the posterolateral aspect. The incisive papilla is a soft tissue bulge covering the incisive foramen retro-incisal in position and contains nasopalatine nerves and blood vessels. Another surface anatomical landmark includes the greater and lesser palatine foramina. Normal palate is required to make tongue contact during oral phase of swallowing and pronunciation of palate-lingual sounds [20]. Abnormal palatal height is generally considered when it exceeds 2 cm at the intermolar area. Average linear width is about 3.4 cm. High narrow palate is associated with increased nasal resistance due to reduced nasal volume. This leads to increased air turbulence and nasal obstruction, contributing to obstructive sleep apnea [21]. Constricted palate also predisposes to skeletal and occlusal disharmonies including crowding of anterior teeth, cross bite and open bite deformities. It appears that tongue forces are required for transverse palatal and maxillary growth. Torus palatinus is a form of bony exostosis at the center of hard palate and is composed of dense cortical with or without spongy bone [22]. Incidence varies from 0.5%-74% based on racial differences. Strong association between masticatory stress and tori formation. It is hypothesized that masticatory stress generates functional hyperplastic response. Genetic factors may contribute 30% in the formation of tori. Tori may be associated with TMD, bruxism, sleep disturbance, irritation and ulceration. Denture fabrication may be difficult in the presence of tori and mandates its removal. Tori may be similar to osteomas which may be multiple in Gardener’s syndrome. Other clinical applications of anatomy of the hard palate includes choosing the thickest areas for connective tissue harvesting. Areas lateral to the median raphae at next to the first premolar appears to have the thickest tissue [23].

The tongue is a muscular organ covered by specialized mucosa containing filiform and fungiform papilla. Other specialized taste papillae are present in the laterally oriented foliate and the V-shaped circumvallate papilla. Bald tongue or atrophic glossitis denotes the absence of tongue papilla and may be related either to factors accelerating their damage and removal like chemical, physical and chemical factors or to those causing reduced rate of their production as in metabolic, hematologic, immunologic and nutritional disorders [24]. The pathophysiology of atrophic glossitis is related to abnormal cellular oxygenation in relation to nutritional deficiency or indirectly by the presence of a disease that causes nutritional deficiency either by interference with absorption or metabolism. Lingual freni attach between the tongue and the mandibular alveolar ridge. It contains fibrous tissue and parts of the genioglossus muscle. Abnormality in lingual freni is called ankyloglossia or tongue tie. In ankyloglossia, the tongue tip cannot be protruded beyond the lower incisors [25]. Ankyloglossia is also implicated when the free tongue (defined as the distance from the frenal attachment in the ventral surface of the tongue to the tip) is shorter than 16 mm.

Clinical findings associated with ankyloglossia include breastfeeding problems, interference with speech, gingival recession and diastema. Its hypothesized that ankyloglossia may predispose to dentofacial deformity sequala like narrow maxillary and mandibular prognathism [26]. The floor of the mouth is present underneath the tongue and contains the mylohyoid muscle, the sublingual glands, the lingual nerves, Wharton’s ducts and branches of the lingual and facial arteries. These arterial branches pass through and supply the mandible through multiple lingual foramina.

The sublingual caruncle is a mucosal elevation at the base of the lingual frenum which contains duct opening of the sublingual and submandibular glands. The sublingual gland opens via multiple ducts the largest of which is the Bartholin’s duct which joins the Wharton’s duct of the submandibular gland beneath the sublingual caruncle. The remaining openings form the ducts of Rivinus and open at the sublingual fold which extends from the sublingual caruncles posteriorly. Due to the presence of dense vascular structures and the potential to develop hematoma that may displace the tongue placing the airway in danger, vertical releasing incisions in the lingual aspect of the mandible are better avoided [27]. Sialolithiasis of submandibular or sublingual glands may be associated with swelling of the involved gland or its duct with history of prandial pain. The stone may be detected clinically by bimanual palpation. Radiographic evaluation is needed to determine the location and number of duct or gland stones [28]. Another clinically important swelling in the floor of the mouth includes ranula or mucocele of the sublingual gland. Plunging or cervical ranulas are not confined to the floor of the mouth and by dissecting through the mylohyoid muscle and reach the submental or submandibular space. Removal of the mucocele and the gland is needed to avoid recurrences.

The Maxillary Sinus

The paired maxillary sinuses are the largest paranasal air sinuses. The maxillary sinus development starts at 5th IU as an extension from the nasal capsule. Its rate of growth is slow and at birth its dimensions are about 3mm, 6mm, 8 mm. At about the age of 7 years, the maxillary sinus growth is accelerated and stops with the eruption of maxillary third molar. The maxillary sinus has a role in facial growth and modeling as it provides surfaces for bone resorption and deposition [29]. Secondary pneumatization occurs throughout life and is especially accelerated in association with tooth loss [30]. The average adult sinus dimensions are about 26mm, 28mm, 40 mm and volume of about 30 cm^3. The maxillary sinus is pyramidal in shape and occupies the posterior maxilla, with its base towards the lateral nasal cavity and its tip extending into the zygomatic body. The canine fossa and infratemporal fossa are located in the anterior and posterior aspect, respectively. The roof formed by the orbit, while the floor is just above the alveolar process of maxillary teeth. During development of the face, the sinus floor is located above the nasal floor by about 4mm, while in adults, however, the sinus floor is about 4-5 mm lower than that of the nasal floor. This is possible as a result of facial growth and sinus pneumatization. In about 50% of the population, the floor of the sinus is in the confines of the maxillary alveolar process. Regarding which tooth is closes to the sinus, different studies [29,31] reported first molar while still others [32] found that the second molar are closest to the maxillary sinus. Dental proximity to the maxillary sinus bears significant clinical correlation as will be described. Sinus septa are bony elevations of more than 3mm and are found in the sinus floor about 27-33% of the time [33,34]. Three locations have been defined and these are: premolar, molar and third molar area with the molar areas as most common location. This may correspond to three different periods of dental development of premolars, molars and third molars. In edentulism, subsequent pneumatization may increase rate of septal formation. Buccolingual orientation is the most common.

The maxillary sinus has a role in humidification and conditioning of inspired air as well as trapping dust and foreign bodies. This is made possible as the sinus is lined with a ciliated pseudostratified columnar epithelium layer that have abundant mucus secreting goblet cells. Mucus acts as a barrier that reduces water loss and traps foreign bodies. Cilia are hair-like extension from the apical part of the columnar cell, the core of which contains 9+1 pairs of microtubules allowing it to beat back and forth. Ciliary beats move the mucus blanket spirally and toward the sinus ostium and into the nasal cavity. The maxillary sinus drains into the middle meatus which is located at the posterior aspect of the hiatus semilunaris corresponding to 2/3rd the way up along the medial wall. The presence of rich vascularity allows for thermoregulation of the inspired air. Other important functions include regulation of intranasal pressure, imparting resonance to voice and shock absorption.

Arterial supply to the maxillary sinus is derived from superior alveolar (which gives anterior, middle and posterior branches), the infraorbital and the palatine arteries. These branches extensively divide and anastomose forming intraosseous and extraosseous plexus. Venous drainage is through facial, sphenopalatine veins and pterygoid venous plexus. It is important to point that through the pterygoid venous plexus there exist communication to the cavernous sinus intracranially making infection spread possible.

Nerve supply to the maxillary sinus is derived the same nerves that supply the maxillary dentoalveolar structures: superior alveolar nerve (with its anterior, middle, posterior branches), infraorbital and anterior palatine nerves. This common source of nerve supply and proximity to the sinus makes referred pain a possibility between both the sinus and maxillary teeth.

Sinus health is dependent on patent ostium and continuous drainage. If mucus is not cleared, bacterial accumulation causes sinusitis. Therefore, ostium obstruction, ciliary dysfunction and increased nasal secretions are important etiological factors in the development of sinusitis. The clinical features of sinusitis include nasal obstruction, offensive purulent discharge with pain in malar area. Examination reveals mucosal redness, turbinate edema and tenderness on palpating the anterior and posterior wall of the maxilla.

Referred sinus pain may produce diagnostic problems regarding the source of pain. Sinusitis may irritate branches of the superior alveolar nerve during their course through the wall of maxillary sinus. Conversely, dental pain may project over the sinus area. However, pain may be the result of both dental and sinus problems. Dental source has to be explored and teeth should be examined thoroughly with inspection, palpation, percussion and radiographs. A suggested differentiating test is to keep a piece of cotton saturated with 5% lidocaine in the nostril of the affected side for 20-30 seconds. Pain relieve occur in case of sinusitis [29].

Sinusitis of odontogenic origin was noted as periodontal lesions were long associated with sinus membrane thickening [31,32]. It seems that microorganisms and their toxins can permeate through tissue barriers and affect the sinus. The rate of this group of sinusitis ranges between 5-10% and in some studies up to 40% [35,36].

Oroantral communication developed after tooth extraction allows oral flora and other contaminants to lodge within the sinus causing sinusitis. Oroantral communication after extraction occurs when the roots are inside the sinus [31]. Punwutikorn reported sinus perforation rate of about 0.3 % (a study on 27,984 extractions at the posterior maxilla). OAC needs identification and primary closure if has a diameter more than 5 mm [37].

Foreign bodies like surgical burrs, root tips or even a teeth, dental implants and bone graft particles all initiate foreign body reaction and cause sinus inflammation. A special challenge is presented to the endodontist when treating teeth in close relation the maxillary sinus as even if instrumentation is kept within the confines of the root canal, extrusion of infected tooth debris or irritating filling material still possible. Sodium hypochlorite, calcium hydroxide, gutta percha and silver points have all been reported to irritate the sinus [38].

Sinus augmentation procedures are done in dental implant practice to avoid violation of the maxillary sinus. Special attention should be directed to the type and location of septa [33,34]. Implant displacement or migration has been reported. Possible explanation to this phenomenon includes negative pressure exerted by the sinus during inspiration causing suction effect on the implant. This can be helped by loss of osseointegration and traumatic occlusal forces. Another clinically useful note regarding the maxillary sinus is that orthodontic movement of roots across the sinus results in tipping movement and root resorption [39].

The Infratemporal Fossa (ITF)

Due to its location and contents, the THE infratemporal fossa (ITF) is part of the skull base and the masticatory compartment. The ITF is an irregularly shaped space located deep to the ramus, posterior to the maxilla and anterior to the styloid process [40]. Major contents include: the pterygoid muscles, the maxillary artery, the pterygoid venous plexus, the mandibular and chorda tympani nerves. Deep lobe of parotid gland and carotid sheath area are located posterior to the ITF and medially, it is bounded by the lateral pterygoid plate and the fibers of the superior pharyngeal constrictor muscle. The roof is formed by the infratemporal surface of the greater wing of the sphenoid bone which contains many of the foramina leading to the skull base. The ITF communicates superiorly with the temporal fossa by a space deep to the zygomatic arch where the fibers of temporalis muscle course to insert to the coronoid process. More importantly, it is connected to the cranium by the foramen ovale, foramen spinosum and sphenoidal emissary foramen (of Vesalius). Valveless emissary veins connect the pterygoid venous plexus to the cavernous sinus intracranially. The pterygopalatine fissure is the cleft between the maxillary tuberosity and the pterygoid plates provides route for communication with the pterygopalatine fossa anteriorly. The ITF has no floor and it is continuous with neck spaces.

The maxillary artery (MA) is one of the terminal branches of the external carotid artery, the other being the superficial temporal artery. The originates at the level of the condyle within the parotid gland. As it enters the ITF it is located between the condyle and sphenomandibular ligament and runs in close relation to the inferior border of the lateral pterygoid muscle [41]. The MA is located 15 mm from the pterygopalatine fissure. The maxillary artery is divided into three parts according to its anatomical relations. The first or mandibular part is related to the mandibular condylar neck. In this location, the MA is inferior to the auriculotemporal nerve and superior to the maxillary vein.

Branches of the first part include the deep auricular artery to the tympanic membrane, skin of the external auditory meatus and TMJ. The anterior tympanic artery leaves the ITF through petrotympanic fissure to the middle ear. The middle meningeal artery supplies the dura mater and enters the cranial fossa through the foramen spinosum. The accessory meningeal artery runs deep to the mandibular nerve in relation to the tensor veli palatini and levator veli palatini and enters the cranium through the foramen ovale. The inferior alveolar artery (IAA) follows the inferior alveolar nerve (IAN) and together, enter the mandibular foramen. Before entering the canal, it gives a branch to the mylohyoid muscle. Inside the mandibular canal, the IAA is found lateral to the IAN and provides dental branches to supply teeth. More anteriorly, it divides into the incisive artery that continues within the canal to the incisor area and to a mental branch that exits through the mental foramen with the mental nerve. The second or pterygoid part of the MA is related to the pterygoid muscle and gives five muscular branches. These are the deep temporal, masseteric, pterygoid, buccal and lingual artery. The third part of the MA enters and terminates within the pterygopalatine fossa. It gives branches that follow those of the maxillary nerve.

The pterygoid venous plexus (PVP) is a dense collection of interconnected venous channels located around the lateral pterygoid muscle and maxillary artery. Essentially, all structures within the ITF drain into the PVP. Emissary veins pass through of the foramen ovale, foramen lacerum and emissary sphenoidal foramina and connect the PVP with the cavernous sinus. A branch from the PVP also pass through the inferior orbital fissure and joins the inferior ophthalmic vein. The PVP drains into the maxillary vein which runs with the first portion of the MA deep to the neck of mandibular condyle. Within the parotid gland, the maxillary vein joints the superficial temporal vein forming the retromandibular vein.

During extraction of upper third molar, its displacement into the ITF is possible especially in cases where only thin bone is present. Improper force application without distal support may direct the tooth toward the ITF. Inadequate vision and poor surgical access also play important roles [42]. Clinically, the patient may be asymptomatic or complain of limited mouth opening due to pain or mechanical obstruction. If infection develops, swelling with collection of pus may be seen by radiographic imaging. Infection of the ITF may be difficult to diagnose and low threshold to obtain adequate radiographic evaluation should be practiced when a patient complains of trismus. Dimitrakopoulos reported subcutaneous emphysema because of Valsalva maneuver was tried [43]. This was attributed to the created communication between the maxillary sinus and the ITF by the displacement. Radiographic evaluation also allows localization of the displaced tooth. Usually the tooth is lateral to lateral pterygoid plate and inferior to lateral pterygoid muscle. Position of the tooth may change by the movement of the muscles and recent CT or MRI is recommended before surgical intervention [44,46]. Different surgical approaches have been described to remove displaced teeth into the ITF. Borgonovo used high maxillary vestibular incision along with bone window in the posterolateral wall of the maxilla [45]. Other reported surgical approaches include Gillies approach and hemicoronal incision [44]. Delaying removal of the tooth to 2 weeks after displacement may be advantageous as fibrous tissue may form. However, some would argue that fibrosis may not occur at all and more urgent decision regarding its removal should be taken to avoid complications like infection in this vital space [42,43].

Nerve block to both IAN and PSAN require deep needle insertion hence, vascular injury may occur. During the PSAN block, the maxillary artery, PSAA, or the PVP may be injured and hematoma formation may be the result. Gupta et al reported immediate hematoma formation during PSAN block despite proper injection technique [47]. Swelling may develop rapidly and could be seen in the buccal, temporal area and eyelid areas.

Hrishi explained that bleeding could reach such areas given the central position of the ITF [51]. LA anesthetic solutions diffusion in proximity to the orbit have the potential to cause ophthalmic manifestations like dipoplia, amaurosis and loss of power of accommodation. Intraarterial injection can reverse the blood circulation and allow backflow of the anesthetic agent to reach the maxillary artery if forcefully injected into the PSAA or IAA [48]. The anesthetic agent then may reach the ophthalmic artery through the meningeal and lacrimal arteries. In addition, injecting PVP can also affect the orbit by way of the emissary veins that connect it to the cavernous sinus which has close association with CN III, IV, VI, and ophthalmic artery. Another possible route is that during injection, the backflow reaches the internal carotid artery (ICA) which transmits the agent to the brain [47].

Infection may reach the ITF from adjacent structures like maxillary molars, maxillary sinus, the parotid gland and as complication of invasive procedures like exodontia and TMJ arthroscopic procedures [49]. Needle injury to the major vasculature of the ITF may produce hematoma that with seeded microbes can produce infection [50].

The Pterygopalatine Fossa

The pterygopalatine fossa (PPF) is inverted cone-shaped concavity located between the posterior surface of the maxilla and the pterygoid process of the sphenoid bone [52]. The PPF contains the maxillary nerve and the pterygopalatine ganglion in addition to important foramina that provide communication to the oral cavity, nasal cavity, ITF, orbit and the cranial cavity. The PPF is about 18 mm long and 2.3 mm wide. It communicates superiorly with the orbit via the inferior orbital fissure which contains the infraorbital and zygomatic nerves. Inferiorly, the PPF converge into a canal measuring approximately 17 mm in length [52]. This canal opens in the oral side through the greater and lesser palatine foramina carrying the greater and lesser palatine nerves, respectively. The anterior wall of the PPF is formed by the infratemporal surface of the maxilla. Laterally, the ptrygopalatine fissure, extends longitudinally between the pterygoid plate and the posterior wall of maxilla ending at the posterior aspect of the inferior orbital fissure superiorly. Through the pterygopalatine fissure, the third part of the MA enters and the posterior superior alveolar nerve leaves the PPF. At the superomedial aspect of the PPF, the sphenopalatine artery and nerve and the posterior superior nasal nerve enter the lateral aspect of the nasal cavity through the sphenopalatine foramen. The posterior border of the PPF is formed by the pterygoid process of sphenoid bone and greater wing of sphenoid bone. The foramen rotundum and pterygoid canal connect the PPF with the middle crania fossa. The former conveys the maxillary nerve and the later contains the nerve of pterygoid canal.

Branches of the third part of the maxillary artery include the posterior superior alveolar artery (PSAA) which arises inside the pterygopalatine fossa and leaves through the pterygopalatine fissure. Over the tuberosity it becomes imbedded into the maxillary bone. The PSAA supplies the posterior maxillary teeth, maxillary sinus, and the buccal gingiva. The infraorbital artery leaves the pterygopalatine fossa and enters the orbit through in the inferior orbital fissure. It runs along the floor of the orbit through infraorbital canal and gives the anterior superior alveolar artery that supply the anterior teeth and anterior wall of the maxilla. The infraorbital artery exits the canal along with infraorbital nerve via the infraorbital foramen. The third part of MA also gives a branch that passes through the pterygoid canal to supply the auditory tube, tympanic cavity and upper part of the pharynx. The descending palatine artery leaves the PPF through the palatine canal where it divides into greater and lesser palatine arteries and these leave the canal through the greater and lesser palatine foramina to supply the hard palate and soft palate, respectively. The sphenopalatine artery leaves the PPF through the sphenopalatine fossa to enter the nasal cavity and supply the posterior lateral wall of the nose. It has branches that supply the posterior aspect of the nasal septum as well.

One of the important contents of the PPF is the maxillary nerve (MN). It is the second division of the trigeminal nerve (V2) arising from the trigeminal ganglion at the middle cranial fossa and reaches the PPF via the foramen rotundum. MN provides sensory innervation to the maxillary dental and sinus structures, hard and soft palate and portion of the nasal cavity. It also contributes to the formation of the PPG. Direct branches of the MN include the meningeal, zygomatic, ganglionic, posterior superior alveolar and infraorbital nerves. The zygomatic nerve leaves the MN in the PPF to enter the lateral aspect of the orbit through the inferior orbital fissure. The zygomatic nerve then divides to the zygomaticotemporal and zygomaticofacial branches which exit the orbit through corresponding foramina to supply the overlying skin. The zygomaticotemporal nerve gives off a lacrimal branch that carries autonomic fiber to the lacrimal gland. The posterior superior alveolar nerve leaves the PPF through the pterygopalatine fissure and runs in area above the tuberosity. It passes through the maxilla posteriorly to supply the maxillary sinus and posterior teeth. The infraorbital nerve (ION) is the terminal branch of the MN and leaves the PPF through the inferior orbital fissure. It then runs through the inferior orbital groove and canal and leaves the orbit through infraorbital foramen located 4-10 mm below the infraorbital rim. In 15% of the times, there exist an accessory infraorbital foramen [48]. Within the infraorbital canal, the ION gives off the middle and anterior superior alveolar nerves that supply the maxillary premolars and incisors and the anterior maxillary sinus wall by contributing to the formation of the maxillary dental plexus. In some situation, the middle superior alveolar nerve is absent and the nerve supply to the premolars is derived from the PSAN or the dental plexus. Terminal branches of the ION includes inferior palpebral, external nasal, internal nasal and superior labial branches.

The pterygopalatine ganglion (PPG) is small heart-shaped neural mass located close to the sphenopalatine foramen [53]. It is connected to the MN from its upper pole by two ganglionic nerve fibers. The PPG supplies sensory and autonomic fibers to the nose, palate, tonsils and lacrimal glands. It contains nerve fibers from the facial, trigeminal and autonomic nervous system. The parasympathetic component of the PPG originates at the superior salivatory nucleus of the brain stem with fibers carried by the nervus intermedius (NI) branch of the facial nerve (CN VII). The NI gives greater petrosal nerve that passes through the facial canal of the temporal bone to enter the middle cranial fossa. It then courses towards the PPF by passing through the foramen lacerum to enter the pterygoid canal. The sympathetic fibers, pass through the PPG without synapse, originate from the superior cervical ganglion which sends branches surrounding the ICA. A branch from this plexus emerge as the deep petrosal nerve and enters the pterygoid canal where it fuses with the greater petrosal nerve to form the pterygoid nerve. Sensory fibers from the MN pass without synapsing through the PPG. Branches from the PPG include orbital, nasal, nasopalatine, greater palatine, lesser palatine and pharyngeal nerves. Orbital branches of the PPG carry sensory fibers to the orbital periosteum and the maxillary sinus in addition to sympathetic fibers to the orbitalis smooth muscle overlying the infraorbital groove. The nasopalatine nerve enters the nasal cavity through the nasopalatine foramen and supplies the posterior inferior portion of the nasal septum. It then courses towards the nasopalatine canal and reach the oral cavity to supply mucosa of the hard palate through the incisive foramen. It provides nerve supply to the anterior hard palate, incisor teeth and palatal gingiva. The greater and lesser palatine nerves leave through the greater and lesser palatine foramina to supply the hard and soft palate, respectively. The greater palatine foramen is located next to the third molar, between the third and second molar or distal to the third molar in about 54%, 6.19%, 38.9%, respectively [48]. Pharyngeal branches to the nasopharynx via the palatalovaginal canal.

With a single injection and small amount of local anesthetic solution, the whole maxillary nerve and the PPG can be anesthetized. Blocking the PPG has also important roles in diagnosis and management of various pain disorders like headache, neuralgias and atypical facial pain [53]. The PPF can be approached either intraorally through the greater palatine foramen or extraorally through the pterygopalatine fissure. Piagkou et al [52] calculated the mean distance from the overlying surface mucosa till the PPF and found it to be around 23 mm. Therefore, to avoid overpenetration during the maxillary nerve technique, it is recommended to bend the needle at a distance of about 23 mm from its tip. The greater palatine foramen is located next to the third molar midway between the median palatine suture and the CEJ of the wisdom tooth in 54% of the time and in 38.9% it may be located more distally. Palpation helps confirm the foramen location. However, it should be remembered that using this technique may endanger adjacent important vital structures like the orbit and the brain by several routes. Diffusion has been suggested to be through the inferior orbital fissure. In addition, intravascular injection may involve a branch from the PVP that is connected to the inferior ophthalmic vein that is also connected to the cavernous sinus [54,55]. Bleeding and hematoma formation may also affect orbit. Orbital hematoma has been reported despite proper technique. Injection at recommended depth, avoiding rapid injection and performing aspiration before injection in at least two planes is recommended to avoid such complications.

The Lingual Nerve

As the mandibular branch of the trigeminal nerve (V3) descends to the ITF through the foramen ovale, it divides to anterior and posterior trunks. The lingual nerve is a branch from the posterior trunk arising above the mandibular notch about 13.5-14.3 mm below the foramen ovale [56]. The lingual nerve then courses between the tensor veli palatini and the lateral pharyngeal muscle [56,57]. At the level of inferior border of the lateral pterygoid muscle about 15 mm inferior to the foramen ovale, the lingual nerve is joined by the chorda tympani which provides secretomotor supply to the submandibular and sublingual glands and taste sensations to the anterior 2/3rds of the tongue. The LN crosses the medial pterygoid muscle and passes below the pterygomandibular raphe reaching the mandibular third molar area. It is documented that the LN is only covered by mucosa and periosteum at the area just distal to the distal root of the mandibular third molar [56,57]. In addition, the incidence of LN location above the alveolar crest is 17.6% being covered by gingiva only, while in 62% of the times, the LN is actually making contact with the lingual crest of the mandible. The mean vertical and horizontal distance from the lingual plate is 0.58-3.45 mm and 2.2-8.3 mm, respectively [48,58]. As the LN approaches the superficial lobe of the submandibular gland, it gives two to three glandular branches and continues over the mylohyoid muscle. The lingual nerve makes medial turn and becomes crossed by the submandibular gland duct at area corresponding to the first or second mandibular molars. The LN is located inferior and lateral to the duct [59]. The LN provides general, gustatory and secretomotor innervation to the presulcus tongue, floor of the mouth, mandibular gingiva and sublingual and submandibular glands.

The LN may be at risk at various surgical and non-surgical procedures. LN dysfunction is manifested by sensory (e.g.: anesthesia, hypoesthesia, paresthesia), gustatory (dysgeusia) and secretomotor deficits (xerostomia). Other reported symptoms include affected speech and drooling [56,59]. Most common procedure that risks the LN is third molar surgeries [48]. Incidence of temporary and permanent LN injury in relation to third molar surgery is 1% and 0.3%, respectively. The mean distance between distal releasing incision and the LN is only 4.4 mm. Factors that increase the risk of LN injury during 3rd molar surgery include lingual flap retraction, vertical sectioning of the tooth, perforation of the lingual plate, insertion of lingual flap retractor, follicle attached to the lingual gingiva and procedure performed under general anesthesia [48,56]. Other than 3rd molar surgeries, inferior alveolar nerve block (IANB) using the Halsted technique also places the LN at risk of injuries. The incidence of associated temporary and permanent LN injury is 0.15% - 0.54% and 0.001-0.01%, respectively [56]. During the injection procedure the needle may actually penetrate the nerve during insertion and after hitting bone it becomes barbed and causes more injury during withdrawal as well. Such injury is capable of producing direct nerve damage. Compression as a result of extraneural or intraneural hematoma may be of greater concern. Tan et al have found that the LN is surrounded by larger number of perineural layers when compared to the IAN which makes ischemia a possible sequela of hematoma formation. In addition, the LN becomes unifascicular or oligofascicular at the level of the mandibular foramen which reduces the chances of nerve recovery if injury occurs [59].

Entrapment neuropathy is another possible mechanism by which LN injury can occur [4,7]. Various structures within the ITF may mechanically impinge on the LN. Passage through or close to hyperactive muscle places sufficient compression on the nerve to cause neuropathy [6]. Other possible sources of compression neuropathy include calcified pterygospinous ligament and enlarged lateral pterygoid plates [57,60].

The Mylohyoid Nerve

Another branch of the posterior division of V3 is the mylohyoid nerve which branches from the IAN at area 14 mm superior to the mandibular foramen [48]. In some cases, the mylohyoid nerve was given off directly from the main trunk of V3 or even from the LN [61]. The mylohyoid nerve provides sensory innervation to mandibular teeth and submental skin and motor fibers to the anterior belly of digastric and mylohyoid muscles. Along with the IAN which passes through the mandibular foramen, the mylohyoid nerve descends more posteriorly and around the sphenomandibular ligament to lodge in the mylohyoid groove at the lingual aspect of the ramus. The presence of retromandibular foramina suggests sensory supply to mandibular molars. The mylohyoid nerve continues anteriorly beneath the mylohyoid muscle till it enters the mandible through the midline retromental foramen and joins the ipsilateral or contralateral incisive nerve or directly supplies the lower incisors [62]. The mylohyoid nerve also sends sensory branches through the lateral lingual foramen to the premolar teeth.

The mylohyoid nerve provides accessory innervation to the mandibular ipsilateral or contralateral incisors in about 60% of the time [62]. With inferior alveolar nerve block alone, the patient feels numb lip but still painful manipulation at the incisors. Anesthesia to the posterior mandibular teeth may need supplemental mylohyoid nerve block. The mylohoid nerve may be spared from anesththsia during the IANB due to several reasons. The mylohyoid nerve is positioned higher than the IAN. In addition, the mylohyoid may pass in a position shielded by the sphenomandibular ligament or the pterygomandibular fascia [57,60]. Therefore, supplemental injection at the pterygomandibular fossa for posterior teeth and at the submental fossa for anterior teeth may be needed. The mylohyoid nerve communication with the lingual nerve has been reported [63]. It is possible that the mylohyoid neve has role in tongue innervation which may serve to explain incomplete anesthesia to the tongue. This also may provide a means of LN recovery after injury.

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