A Brief Review of Intra oral defects treatment

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Abstract

Background: Intraoral soft tissue defects can be induced by various etiologies. Cleft palate and cleft alveolus are congenital defects that accompany bone defects. Oroantral fistula is often observed after tooth extraction in cases of severe sinus pneumatization. Tumor or trauma also shows various degrees of soft tissue defects. For the treatment of cleft palate sealing the communication between the oral cavity and the nasal cavity is essential for successful treatment. Salivary gland tumors are a rare group of complex, heterogonous histologies that are located in the parotid glands, submandibular glands, sublingual glands and minor salivary glands of the upper aerodigestive tract. Congenital epulis or congenital gingival granular cell tumor refers to an exophytic congenital tumor that arise from the alveolar ridge and has histologic features identical to those of granular cell tumors. Jaw tumors and cysts are relatively rare growths or lesions that develop in the jawbone or the soft tissues in the mouth and face. Jaw tumors and cysts sometimes referred to as odontogenic or nonodontogenic, depending on their origin can vary greatly in size and severity. For all these causes of intraoral lesions, over decades plastic surgeons have introduced many types of flaps for reconstruction of sealing such intraoral defects. In this brief review, we will highlight some of the most commonly met intraoral defects and some of the most commonly used flaps in their reconstruction.

Keywords: Intra oral defects

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Introduction

During the embryonic period, the oral cavity undergoes a series of complex developmental events. The formation of the oral cavity begins around the fourth week of gestation and involves the fusion of the frontonasal process, maxillary processes, and mandibular processes. This fusion gives rise to the primary palate, secondary palate, and associated structures **(1)** .

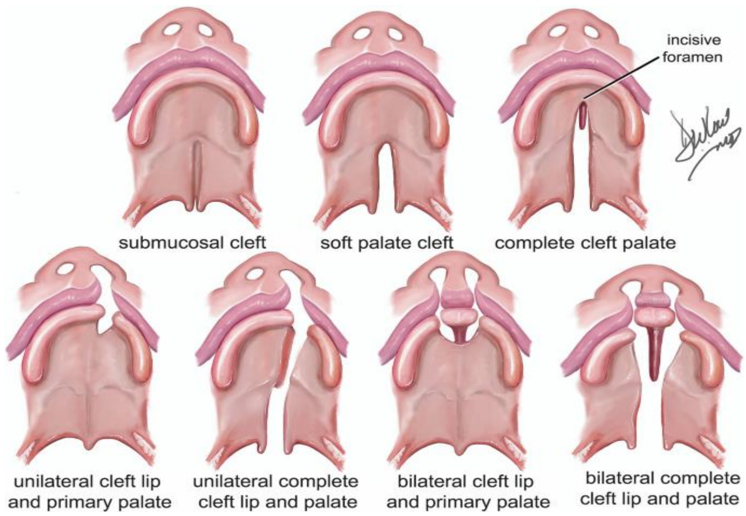
The Primary Palate:

The primary palate forms the anterior part of the oral cavity and includes the upper lip and the premaxilla. It develops from the fusion of the medial nasal processes and the maxillary processes. Any disruptions in this fusion process can lead to cleft lip and palate, which are among the most common congenital anomalies **(2).**

The Secondary Palate:

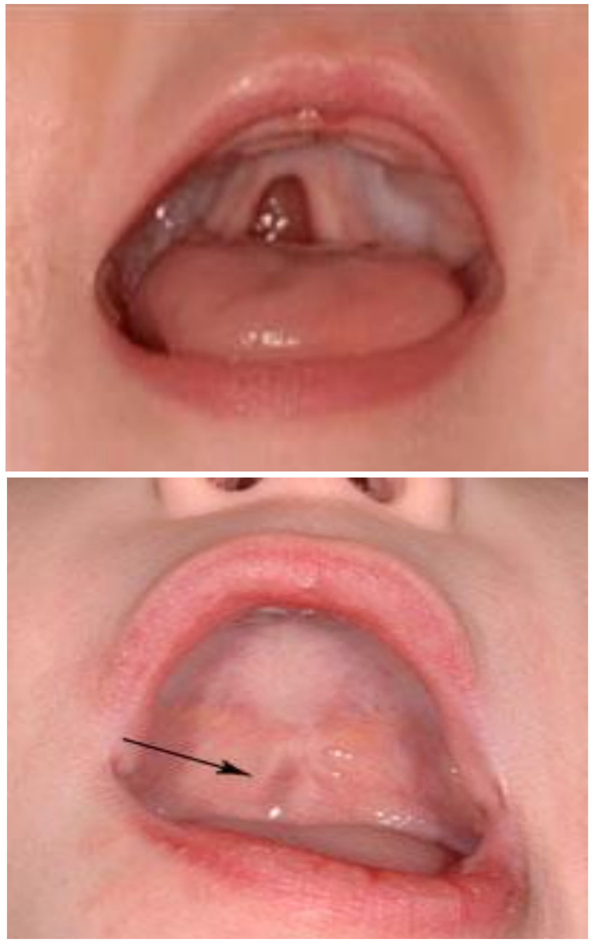
The secondary palate, also known as the definitive palate, develops from the fusion of the two palatal shelves. These shelves grow horizontally and eventually fuse with each other and with the vomer, separating the oral and nasal cavities. Failure of this fusion can result in cleft palate, another common congenital anomaly **(2) .**

Congenital defects



Fig(2): showing classification of cleft palate. **(3).**

I. Subnucosal cleft.
II. Veau Class I: Incomplete cleft, soft palate only (no unilateral/bilateral designation)
III. Veau Class II: Hard and soft palate, secondary palate only (no unilateral/bilateral designation)
IV. Unilateral cleft lip and primary palate
V. Veau Class III: Complete unilateral cleft including lip (primary and secondary palates)
VI. Bilateral cleft lip and 1ry palate
VII. Veau Class IV: Complete bilateral cleft



Fig(3): showing various types of cleft palate a) complete b) incomplete c) submucous cleft. **(3).**

Oroantral fistula

Another congenital defect is oroantral fistula (OAF) it can be defined as an epithelialized pathological unnatural communication between the oral cavity and the maxillary sinus. (**3).**

They can arise as late sequelae from perforation and last at least 48–72 h. An oroantral fistula (OAF) may develop as a complication of maxillary molar or premolar extraction due to the proximity of the bicuspid apices and molars to the antrum. **(4).**

Furthermore, oroantral fistula might originate following the removal of maxillary cysts or tumors, facial trauma, dentoalveolar or implant surgery, and infection or may even be iatrogenic in nature. Oroantral fistulas are common between the ages of 30 and 60. **(4).**

Fig(4): showing oroantral fistula. **(5.)**..

Osteonecrosis of the jaw bone

Osteonecrosis can result from radiation therapy during the treatment of malignancy or medications**,** such as bisphosphonate and denosumab. The main mechanism of osteonecrosis is vascular impairment and resultant hypoxia. Additional microbial invasion and dental procedures are subsequent events that lead to the progression of osteonecrosis. **(6).**

Nonsurgical therapy for osteonecrosis consists of regular dressing and prescription of supplemental antibiotics. Because of the avascular nature of the disease, hyperbaric oxygen therapy has also been used in some studies. **(6).**

Surgical intervention involves the complete removal of necrotic bone and subsequent reconstruction with rich vascularized tissue. Microvascular reconstruction has been used for the reconstruction of osteonecrosis because of poorly vascularized tissue beds in recipient sites. **(6).**

Unsuccessful epithelial healing on the bone is frequently observed in cases with incomplete resection of the necrotic bone. These cases could be treated by additional resection of sequestrum. **(7).**

Fig (5): showing osteonecrosis of the Jaw. **(7).**

Tumors

Squamous cell carcinoma

Squamous cell carcinoma (SCC) of the oral tongue and floor of the mouth (OTFOM) may be approached in several ways depending on its extension, involvement of other structures (such as mandible, cheek or base of tongue), and nodal staging. **(8).**

The ideal treatment should provide adequate margin resections to allow for ideal reconstruction aiming to restore the oral lining and preserve residual tongue mobility and volume **(8).**

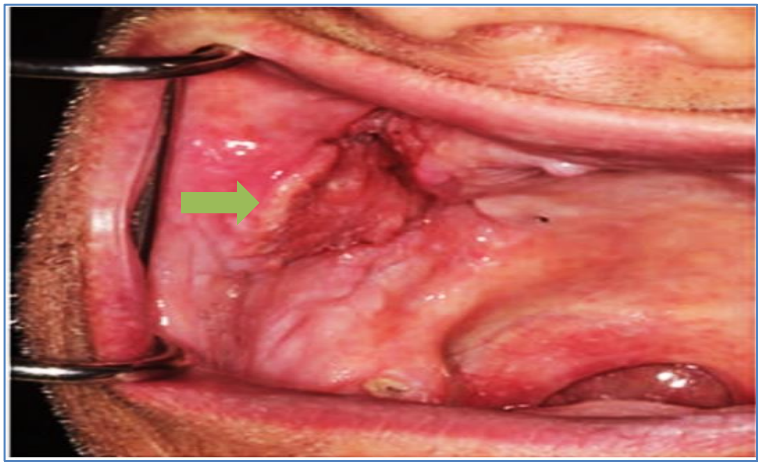
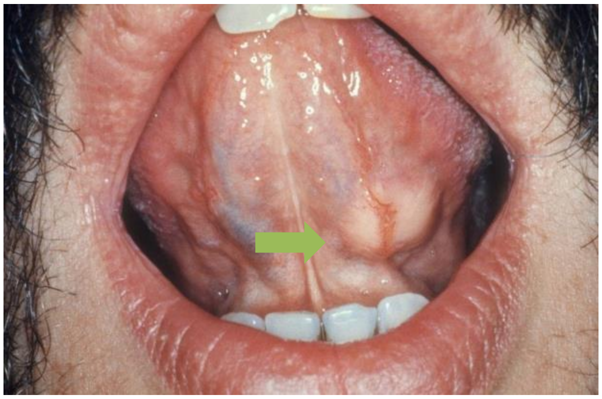


Fig (6) : intra oral defect due to SCC (green arrow) **(8).**

Salivary gland tumors

Salivary gland tumors are a rare group of complex, heterogenous histologies that are located in the parotid glands, submandibular glands, sublingual glands, and minor salivary glands of the upper aerodigestive tract**. (8).**

The most common histologies include mucoepidermoid carcinoma (MEC), acinic cell carcinoma (ACC), adenoid cystic carcinoma (AdCC), carcinoma ex-pleomorphic adenoma (CExPA), and adenocarcinoma. **(6).**



Fig(7): showing sublingual salivary gland tumor (green arrow). **(6).**

Epulis

Epulis is a general term used to describe a number of reactive gingival lesions with vascular, fibroblastic, and granulation tissue proliferation. **(9).**

Congenital epulis or congenital gingival granular cell tumor refers to an exophytic congenital tumor that arise from the alveolar ridge and has histologic features identical to those of granular cell tumors. **(9).**

Fig(8):showing congenital epulis in upper alverolar ridge. **(9).**

Jaw tumors

Jaw tumors and cysts are relatively rare growths or lesions that develop in the jawbone or the soft tissues in the mouth and face. Jaw tumors and cysts sometimes referred to as odontogenic or nonodontogenic, depending on their origin can vary greatly in size and severity. **(10).**

These growths are usually noncancerous (benign), but they can be aggressive and expand, displace or destroy the surrounding bone, tissue and teeth. **(10).**

Odontogenic tumors are quite uncommon lesions. It can be either derived from odontogenic ectoderm or ectomesenchyme. Odontogenic tumors are also classified into benign and malignant tumors. **(10).**

Benign Odontogenic tumors include: ameloblastoma, calcifying epithelial odontogenic tumor , squamous odontogenic tumor, while malignant odontogenic tumors include: ameloblastic carcinoma, clear cell odontogenic carcinoma and primary intraosseous carcinoma, Non otherwise specified (NOS) sclerosing odontogenic carcinoma, Ghost cell odontogenic carcinoma, Odontogenic carcinosarcoma, and Odontogenic sarcomas. **(10).**

while non-odontogenic tumors of the jaw are rare pathologies that can often be overlooked, resulting in delayed diagnosis and treatment. They cover a vast range of pathologies such as: Osteosarcoma , central giant cell granuloma , fibrous dysplasia , osteoma and osteoblastoma. **(11).**

Fig(9):showing calcifying cystic odontogenic tumor. **(11).**

Fig(10): showing osteosarcoma at rt upper buccal sulcus. **(11).**

Reconstruction of intraoral soft tissue defects from traumatic injuries, treatment of pathologic lesions, or congenital wide palatal clefts are interesting. Replacement of resected or defective oral mucosa with appropriate tissue has concerned surgeons for long. Special kind of fasciocutaneous, myocutaneous, and muscle flaps for coverage of intraoral soft tissue defects have been designed. Replacement of mucosa with the same kind of tissue is physiologically optimal. **(8).**

Choosing a pedicled flap to reconstruct an intraoral defect depends on the size and the anatomic position of the tissue defect. The goals are to restore form and function and minimize donor site morbidity. Regional pedicled flaps available for intraoral reconstruction are the buccal fat pad flap, buccinator myo-musocal flap, facial artery myomucosal flap, platysma, and temporalis muscle flap. **(12)**

BUCCAL ARTERY MYOMUCOSAL FLAP (BAMM) **(13)**

Bozola et al.,1989 first described an axial musculomucosal flap based posteriorly on the buccal artery. It is an axial flap based and centered on the buccal artery, an internal maxillary artery branch, extending from the maxillary tuberosity to the oral commissure.

The buccal mucosa and the buccinator muscle are incised to the level of the bucco- pharyngeal fascia, and the flap elevated in an anterior to posterior direction in the loose areolar plane between the buccinator muscle and the bucco-pharyngeal fascia. The buccopharyngeal fascia is preserved because it prevents herniation of the buccal fat pad and avoids injury to branches of the facial nerve. Small branches of the facial artery are ligated, as are anterior venous tributaries from the pterygoid plexus.

The dissection proceeds posteriorly until just anterior to the pterygomandibular raphe, where the main neurovascular bundle enters the flap. The donor site is closed primarily.

It can be used to close small mucosal defects of the posterior hard palate, soft palate, and maxillary alveolus. BAMM flap can be used successfully to reconstruct ipsilateral oral and oropharyngeal cavity soft tissue defects. No second surgery for pedicle section is required and defects up to 8 cm in lenght can be properly reconstructed.

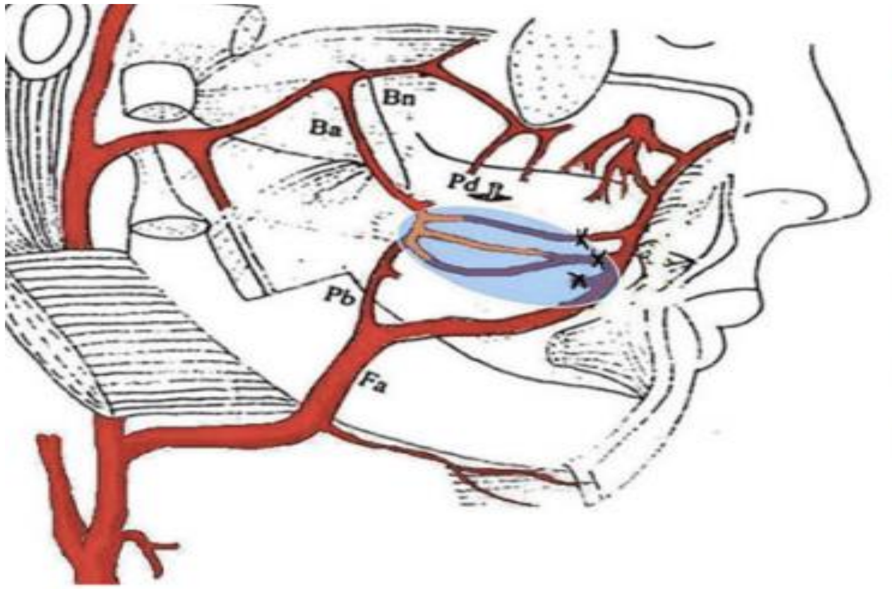


Fig (11): BAMM vascular pattern and shape. **(13).**
Ba: Buccal artery **Bn**: Buccal nerve **Pd**: Parotid duct
Fa: Facial artery **Pb**: Posterior branch of facial artery

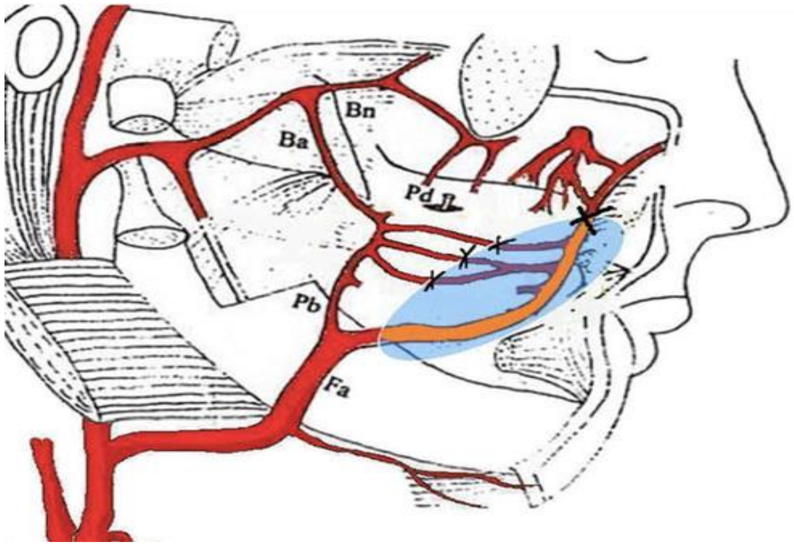
FACIAL ARTERY MYOMUCOSAL FLAP (FAMM) **(13,14,15)**

In 1992**, Pribaz** described an axial musculomucosal flap based on the facial artery, combining the principles of the nasolabial and buccal mucosal flaps. He designated the flap, the facial artery myomucosal (FAMM) flap.

The FAMM flap is an axial flap based and centered on the facial artery with an oblique orientation, extending from the retromolar trigone to the ipsilateral gingival labial sulcus at the level of the alar margin.

It can be used to close small mucosal defects of the posterior hard palate, soft palate, tonsillar fossa, alveolus, floor of the mouth, and lower lip. In dentate patients, this flap requires a bite bloc during healing and a second operation time for pedicle section and vestibuloplasty.

The FAMM flap has an excellent venous outflow based on a rich venous submucosal plexus that allows the mobility of the flap without venous congestion.



Fig(12): FAMM flap vascular pattern and shape **(13).**
Ba: Buccal artery **Bn**: Buccal nerve **Pd**: Parotid duct
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BUCCAL (BICHAT) FAT PAD FLAP **(16)**

The buccal fat pad (BFP) is a specialized type of tissue that is distinct from subcutaneous fat. In infants it prevents in-drawing of the cheek during sucking; in adults the BFP enhances intermuscular motion. The BFP lies in the masticatory space between the buccinator muscle medially and the masseter muscle laterally. It separates the muscles of mastication from one another, from the zygomatic arch, and from the ramus of the mandible and is wrapped within a thin fascial envelope. It is thought to serve as a rich venous net, with valve-like structures, possibly involved in exo/endocranial blood flow through the pterygoid plexus. The landmarks to identify the BFP are the superior buccal sulcus in the molar region and the anterior border of the mandibular ramus. The advantages of this flap are as follows: quick, simple, and easy dissection; can be used under local anesthesia; there are no visible scars; low rate of morbidity; very low failure rate; and it can be associated with other pedicled flaps.

During harvesting, Stensen's duct should be identified first with a lacrimal probe before incision to avoid damaging it during the procedure. A 2–3-cm mucosal incision is made at least 2 cm below the Stensen's duct. Two or three tagging sutures are placed at the margin of the mucoperiosteal flap to gain appropriate surgical fields. The buccinator and zygomaticus major muscles are cut, and blunt dissection is carefully performed to create sufficient openings for herniating the fat pad without injuring the capsule overlying the fat pad. After the superficial fascia of the face is cut, the fat pad herniates spontaneously. The capsules overlying the fat pad should not be torn so as to maintain its volume, and the arterioles and venules overlying the fat pad should be preserved to maintain the rich blood supply. Tissue forceps are used for the traction of the fat pad with minimal force to avoid tearing the capsule. Pedicled buccal fat pad is sutured and positioned using absorbable suture materials with minimal tension. Making the incision at the bone is a good technique for maintaining the position of the fat pad.

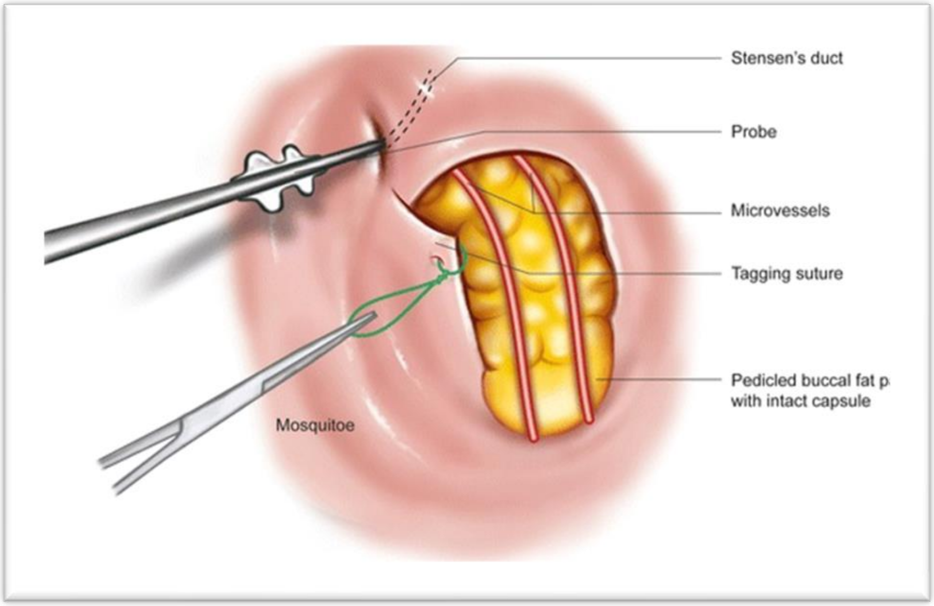


Fig (13): Surgical procedure for the buccal fat pad flap. A blunt dissection is carefully performed without injuring the capsule overlying the fat pad. After the superficial fascia of the face was cut, the fat pad herniated spontaneously. **(16).**

PLATYSMA FLAP **(17)**

Detailed anatomic studies of the platysma flap described the vascular anatomy with the submental artery as the predominant blood supply to the platysma muscle as well as to the skin paddle. It was found that sacrificing the facial vessels is critical as far as flap survival is concerned, however, this matter is debatable.

As the flap can be either used superiorly or posteriorly-based, it can provide an adequate amount of pliable soft tissue that is sufficient for reconstruction of small to medium-sized skin or mucosal defects of the facial skin, oral cavity, and pharynx. The platysma myocutaneous flap can be easily harvested and has low donor site morbidity, but has not gained widespread acceptance due to some concerns about the flap's vascularity.

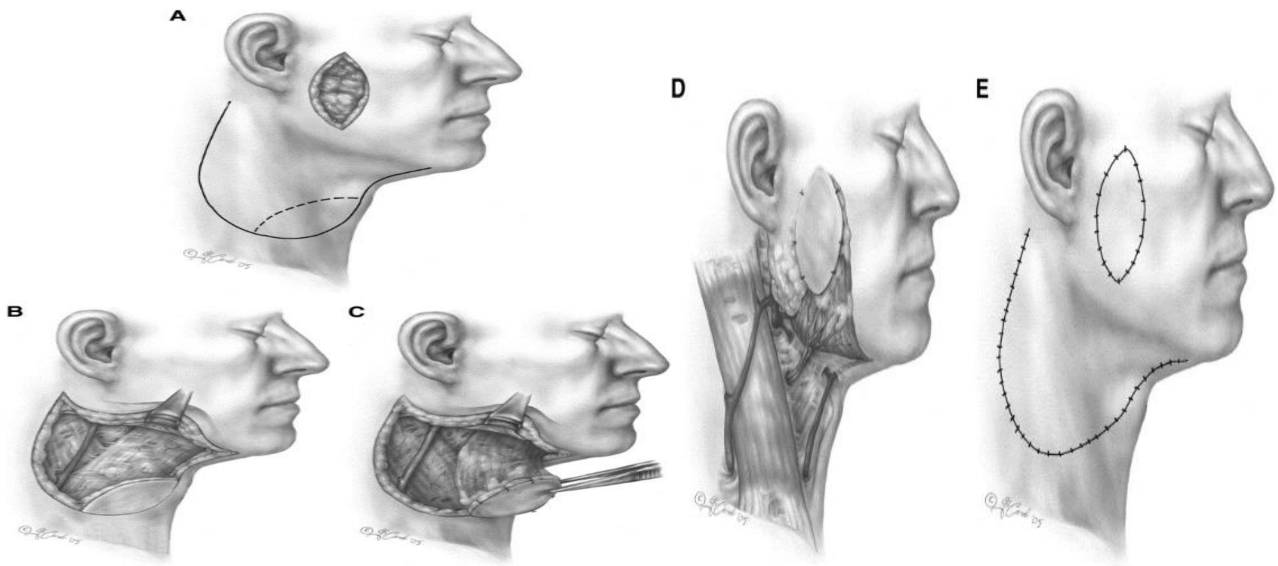


Fig (14): Steps of platysma flap harvesting **(17).**

(A) Surgical defect with incision and skin flap corresponding to defect size outlined. (B) Skin flap elevated without the platysma. (C) Skin paddle sutured to the underlying muscle and raised. (D) Flap sutured in place. (E) Postoperative result.

SUBMENTAL FLAP **(12)**

The submental flap is designed across the midline over the submental area: the upper limit is just under the mandibular arch in the midline; the lateral limits are marked below the mandibular angles, bilaterally. The inferior limits depend on the redundant tissue available to allow primary closure. The area of a submental flap is between 5 cm × 5 cm minimum, even in younger patients, and 7 cm wide × 12 cm long maximum.

The arc of rotation of the submental flap pedicle has its pivot point at the level of the angle of the mandible.

During harvesting, the inferior border of the skin paddle is incised, with the platysma muscle included. Once the submandibular gland is reached, the dissection must be close to the gland, ligating each branch coming from the submental artery. It is necessary to proceed up to the upper-third of the gland. It is then possible to see the facial vessels clearly.

Care must be paid to identify the marginal branch of the facial nerve in the sub-platysma fatty layer before proceeding to dissect. Staying close to the posterior (deep) aspect of the platysma prevents any injury to the nerve. The upper incision is made through the platysma muscle.

The flap is raised from the contralateral side to the ipsilateral pedicled side. The contralateral anterior belly of the digastric muscle is left deep, but the platysma muscle is taken with the flap. At this point, the contralateral submental artery and submental vein are ligated.

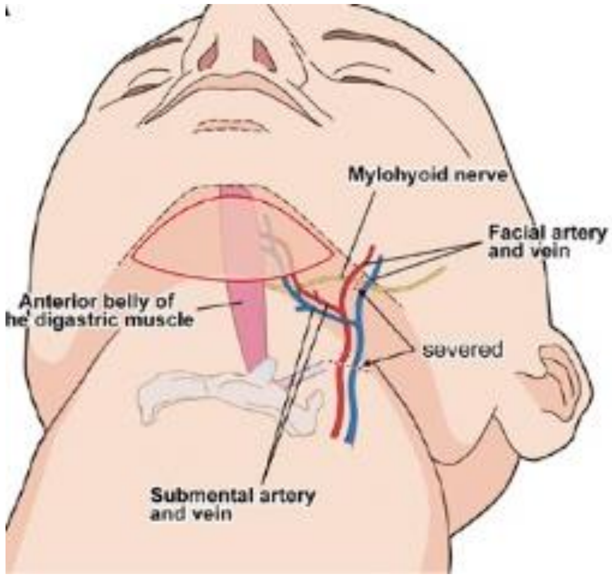


Fig (15): a diagram of submental flap. **(12).**

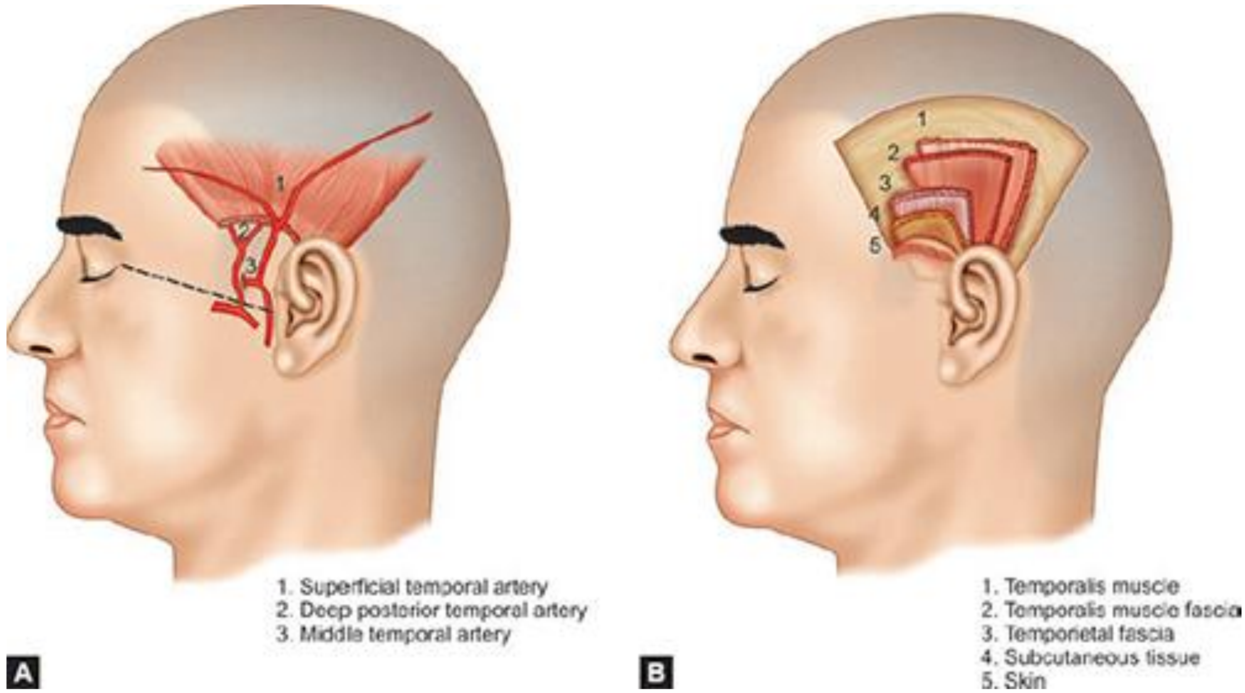
TEMPORALIS MUSCLE FLAP **(18,19)**

The temporalis muscle is a fan-shaped muscle on the lateral skull, filling the entire temporal fossa. It passes under the zygomatic arch to insert onto the coronoid process and the anterior aspect of the ramus of the mandible.

The temporalis muscle flap (TMF) has a type III pattern of circulation. The dominant pedicles are the anterior deep and posterior deep temporal arteries and venae commitants. Both arteries arise from the internal maxillary artery, which is a branch of the external carotid artery. The artery enters the muscle at its undersurface, deep to the zygomatic arch. It should be kept in mind that the internal maxillary artery courses deep and close to the coronoid process of the mandible.

An additional minor pedicle comes from the superficial temporal vessel, through the middle temporal artery and venae comitantes. This additional pedicle has to be cut.

While free tissue transfer is the mainstay of complex intraoral reconstructions, knowledge of pedicled regional flaps is very useful for smaller defects or salvage of partially failed reconstructions. The BAMM flap, the BFP flap, FAMM flap, platysma, PM flap, TMF, and TFs offer a plethora of options for local/regional flap reconstruction for intraoral pathology.



Fig(16): a diagram of Temporalis muscle flap **(18,19).**

A) Showing the flaps blood sipply

B) Showing the layers from skin to temporalis muscle.

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