SHAHIN
SHAHIN AESTHETIC ACADEMY & CLINIC

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CONTENTS

AESTHETICS TERMINOLOGY

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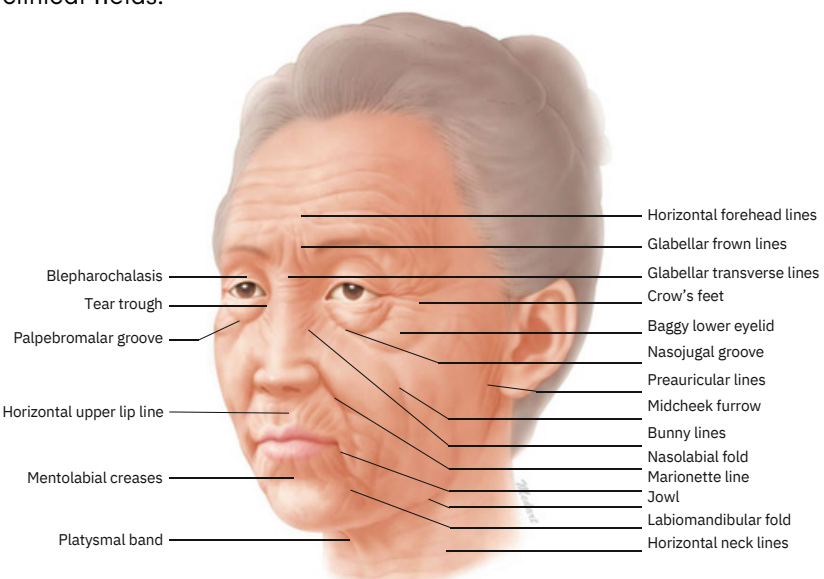
Inconsistencies exist between anatomical and aesthetic terminology. We attempt to re-define common clinical terms according to anatomical regions.

BASIC AESTHETIC TERMINOLOGY

Facial Creases

Facial creases are deep, shallow creases caused by changes in the structural integrity of the skin. It occurs due to loss of skin and muscle fiber elasticity caused by repetitive facial movements and changes in facial expressions. Creases are generally termed wrinkles and lines. Other terms such as furrow, groove, and sulcus are used in the clinical fields.



Aging facial creases and wrinkles

Skin Folds

Skin folds occur due to sagging, loss of tension, and gravity. Representative skin folds are the nasolabial fold, the labio mandibular fold, etc.

Baggy Lower Eyelids (or Cheek Bags, Malar Bags)

Baggy lower eyelids occur due to a drooping of the adipose tissue underneath the orbicularis oculi m. This should be distinguished from the festoon since the baggy lower eyelid occurs inferior to the orbital margin.

Blepharochalasis

Blepharochalasis occurs due to sagging of the eyelid skin.

Bunny Line

The bunny line is the oblique nose furrows lateral to the nose bridge that is pronounced by various facial expressions. The levator labii superioris alaeque nasi m. below the skin and the medial muscular band of the orbicularis oculi m. participate in the formation of the bunny line.

Commissural Lines

Commissural lines are short, vertical lines appearing on each sides of the mouth corner. Occasionally, deep creases may form starting from the perioral regions.

Crow's Feet (Lateral Canthal Wrinkles)

Crow's feet are thin, bilateral wrinkles at the lateral sides of the eyes formed by the orbicularis oculi m.

Festoon

Festoon is the bulged appearance of the lower eyelids caused by a sagging of the skin and of the orbicularis oculi m. and by a protrusion of the inferior orbital fat compartment underneath the orbital septum.

Horizontal Forehead Lines (Worry Lines)

Horizontal forehead lines are horizontal lines across the forehead region where the frontalis m. is located.

Glabellar Frown Lines (Glabellar Creases or Lines)

Glabellar frown lines are vertical creases along the glabellar region caused by the corrugator supercilii muscle fibers.

Glabellar Transverse Lines

Glabellar transverse lines are horizontal lines on the radix that are typically produced during facial distortion. They occur perpendicular to the fibers of the procerus m.

Gobbler Neck (Platysmal Bands)

The gobbler neck appears as bilateral vertical skin bands on the neck along the anterior cervical and submental region. This occurs due to sagging of the medial border of the platysma muscle.

Horizontal Neck Lines

Horizontal neck lines are horizontal skin folds on the anterior cervical region. They are produced by a combination of platysmal muscle fibers and sagging neck skin.

Horizontal Upper Lip Lines (Transverse Upper Lip Lines)

Horizontal upper lip lines are 1-2 horizontal lines located at the philtrum on the upper lip.

Jowl (Jowl Sagging)

Jowl is the protrusion and sagging of the subcutaneous adipose tissue along the mandibular border. The anterior border of the prejowl sulcus clearly signifies the existence of mandibular retaining ligaments.

Oral Commissure

The labial commissure is the region where the upper and lower lips join on each lateral side. The joining point is referred to as the cheilion.

Labiomandibular Fold

The labiomandibular fold spans from the corner of the mouth to the mandibular border and becomes prominent with age. The depressor anguli oris m. (DAO) defines the fold's medial and lateral borders. The attachment of the mandibular retaining ligament causes the labiomandibular fold to be located more anteriorly and medially.

Marionette Line

The marionette line is a long, vertical line that proceeds inferiorly from the corner of the mouth. It occurs commonly with age but with unknown causes. It is more pronounced in people with less fat tissues than in those with more fat tissues. This line is also called the "disappointment line."

Mentolabial Creases (or Furrows)

Mentolabial creases are horizontal creases (one or more) between the lower lip and the chin (mentum). These creases lie between the orbicularis oris m. and the mentalis m.

Midcheek Furrow (Indian Band)

The midcheek furrow is a downward and lateral band, or furrow, that extends the nasojugal groove from the lateral aspect of the nose to the region superior to the anterior cheek. This band may carry on inferior to the cheek. With age, the cheek and the midface droop inferiorly and medially, and the band forms along the inferior margin of the zygomatic bone at the same height where the zygomatic cutaneous ligament attaches to the skin in this region.

Nasojugal Groove

The nasojugal groove is formed at the border between the lower lid and the cheek and runs inferolaterally from the medial canthus. The nasojugal groove region corresponds with the lower border of the orbicularis oculi m. and becomes more pronounced with the existence of the medial muscular band of the orbicularis oculi m. With age, this groove obliquely continues downward to the midcheek furrow.

Nasolabial Fold (or Nasolabial Groove)

The nasolabial fold starts from the side of the nasal ala and extends obliquely between the upper lip and the cheek. With age, the subcutaneous adipose tissue of the anterior cheek sags, causing the fold to deepen and move downward. The adipose tissue of the anterior cheek cannot descend inferior to the nasolabial fold due to compact attachment of the fascia, the skin, the cutaneous insertions of upper lip elevator muscles, and the zygomaticus major m. into the skin in this area. In addition, the facial area tends to lie underneath the nasolabial fold with variable depths.

Palpebromalar Groove

The palpebromalar groove is the border between the lower lid and the malar region.

Preauricular Lines

Periauricular lines are several vertical skin lines located near the tragion, the ear lobule, and the anterior region of the auricles.

Ptotic Chin

The ptotic chin is a flat and contracted chin associated with a deepened submental crease.

Tear Trough

The tear trough is a line originating from the medial canthus and proceeding inferolaterally along with the infraorbital margin. With age, the inferior and medial portions of the orbit sink due to contraction of the soft tissues (skin, muscle, and fat) covering the area. The tear trough has various forms according to how the medial part of the orbicularis retaining ligament and the fibers of the medial muscular band of orbicularis oculi m. come into contact with the skin.

Temple Depression

Temporal depression is the gradual decrease in volume of the soft tissues of the temporal region expressed with age. The bone structure of the temporal crest becomes more pronounced.

Vertical Lip Line

As aging is processed, the tooth is lost and alveolar bone is absorbed. It leads perioral muscle and lip contracts, so the vertical lip line appears along the vermilion border.

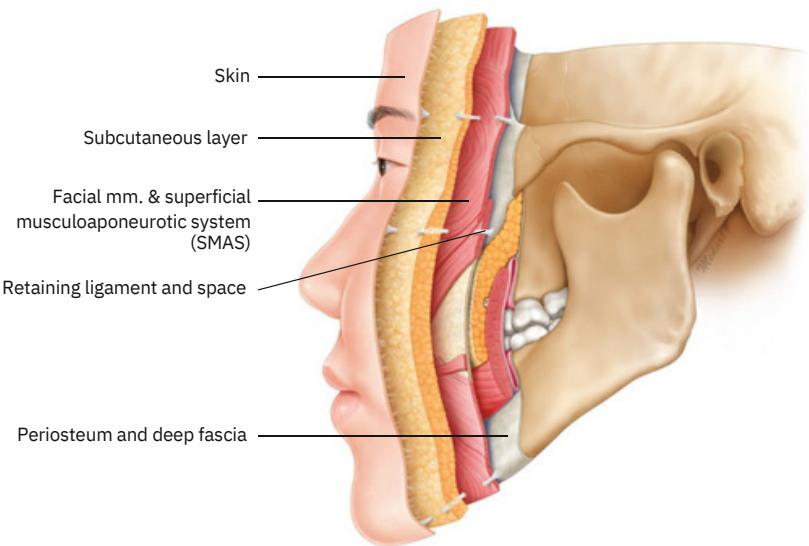
FACIAL LAYERS

LAYERS OF THE FACE

The face, with its diverse ability to portray emotions whilst communicating, is one of the most uniquely recognizable areas of the human body. An increasing interest in facial aesthetics, coupled with considerable research, has extended our understanding of the facial layers and the subtle physical variations resulting from underlying bone structure and genetic factors. With progressive aging, the face undergoes asynchronous changes which may present unique surgical challenges.

Insightful understanding of facial anatomy as pertaining to the aging process facilitates treatment planning and predictable outcomes.

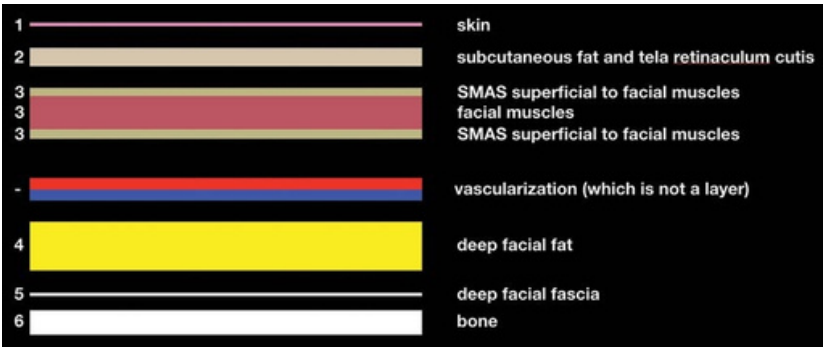
Traditionally the face has been divided into upper, middle and lower horizontal thirds with the upper face extending from the trichion to the glabella, the mid-face from glabella to the subnasale, and lower face extending to the menton.



However, by distinguishing between functional regions and considering the anatomy in terms of a layered construct bound together by retaining ligaments.

Seven major layers may be differentiated:

1. Skin
2. Superficial fat
3. Superficial muscular aponeurotic system (SMAS)
4. Muscle
5. Vasculature
6. Deep fat
7. Bone



The schematic illustration of the facial layers.

SKIN

The skin represents the superficial layer of the face and is an important indicator of age.

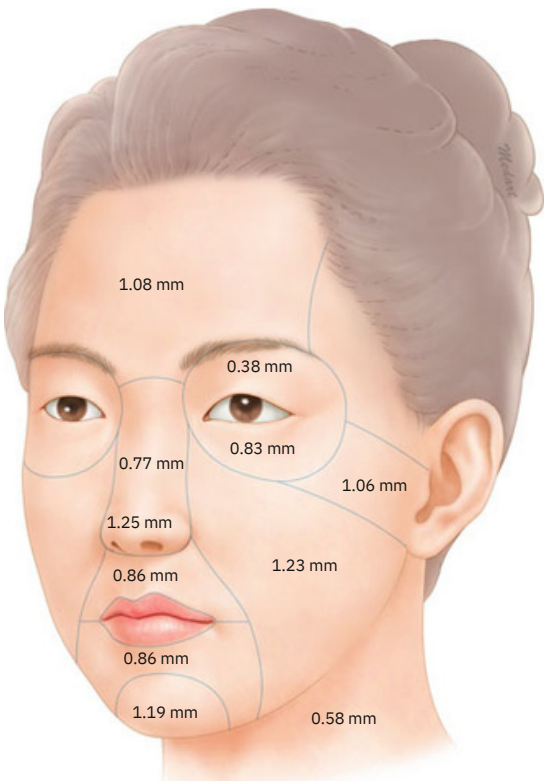
In youth, the skin is smooth, firm, unblemished, and retains a uniform texture. The skin may be histologically divided into epidermis and dermis, with the dermis consisting of collagen, elastic fibers, and ground substance comprising mucopolysaccharides, hyaluronic acid, and chondroitin sulphate.

Cutaneous aging often escalates from the fourth decade under the influence of contributory genetic, hormonal, behavioral and environmental factors. During soft tissue aging, the two distinct processes of deflation and descent manifest as excess skin. Wrinkles start to appear in the lower eyelids and lateral orbital areas, along with the development of dyschromia, textural changes, pigmentation, dryness, thinning, folds, drooping, and mimetic lines. The midface is particularly susceptible to UV-induced aging, with the subsequent development of rough, wrinkled and leathery skin carrying a higher incidence of telangiectasias, premalignant conditions and malignancies. Other causes of extrinsic aging include smoking, pollution, infrared-A radiation and also visible light.

Recent advances in the understanding of volume loss as a critical component of facial aging, and the subsequent integration of volume replacement into both surgical and non-surgical treatment algorithms, arguably represents one of the most significant advances in the field of facial rejuvenation.

THICKNESS OF THE SKIN

The general thickness of the facial skin is described in the illustration below. When treating in areas with thin layers of skin, a filler injection should be cautiously performed while trying to avoid shallow filler placement. Upper and lower eyelids, glabellar regions, and nasal regions have an exceptionally thin skin layer. On the other hand, the skin layer of the anterior cheek and the mental region are relatively thicker. During filler treatment, the skin's flexibility and internal space should also be considered along with its thickness.



Average skin thickness of the face

SUPERFICIAL FAT

In youth, the facial fat consists of a diffuse, balanced spread of superficial and deep fat which create the different arcs and convexities of the face.

The superficial and deep layers are separated by the superficial muscular aponeurotic system (SMAS). Superficial fat is understood to be separated into unique compartments, which are divided by fascial septae containing vascular structures. The major role of the fat layers is as a gliding plane for the facial mimetic muscles.

The superficial fat compartments comprise the nasolabial, medial, middle, and lateral temporalcheek, central, middle, and lateral temporal-cheek (found within the forehead) and superior, inferior, and lateral orbital fat pads. The nasolabial fat, located medial to the cheek fat pads, plays a pronounced role in sagging of the nasolabial fold. The orbicularis retaining ligament (ORL) is situated 2-3 mm below the inferior orbital rim and forms the superior border of both the nasolabial and medial cheek fat compartments. The middle cheek fat compartment, juxtaposed between the medial and lateral temporal-cheek fat compartments, contains a superior fascial border known as the superior cheek septum.

The individual fat compartments age at different tempi and vary metabolically, thus contributing to segmental loss of fullness and the stigmata of aging. The periorbital, forehead, malar, temporal, mandibular, mental, glabellar, and perioral sites are prone to volume loss, whilst the nasolabial and inferior jowl compartments may hypertrophy. The infraorbital and malar fat pads often become more prominent, with anterior protrusion of the malar fat causing it to bulge against the nasolabial crease, thus emphasizing the nasal fold. It is important to understand that individual fat pads behave differently after injection with fillers, with inferior displacement of the superficial nasolabial, middle cheek, and jowl compartments after injection. However, injection into the medial and lateral cheek and superficial temporal compartments lead to an increase in local projection without inferior displacement.

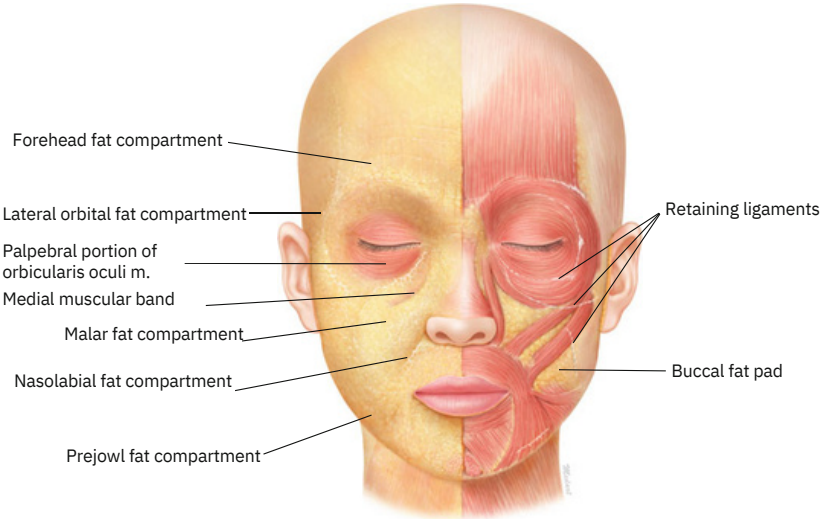
SUPERFICIAL MUSCULAR APONEUROTIC SYSTEM (SMAS)

The SMAS, which has been recognized since 1799, is a unique subcutaneous fascia which is continuous with the platysma below and galea above. It acts as an investing fascia for the facial mimetic muscles, thus playing an important role in facial expression. The SMAS is firmly adherent to the parotid-masseteric fascia in the lateral aspect, where it is known as the immobile SMAS. The facial retaining ligaments, which originate from either the periosteum (zygomatic and mandibular retaining ligaments) or underlying muscle fascia (masseteric and cervical retaining ligaments) transmit through the SMAS to the overlying skin and serve as barriers between the superficial and deep facial fat compartments. Neurovascular structures, or "facial danger zones," are located between these retaining ligaments.

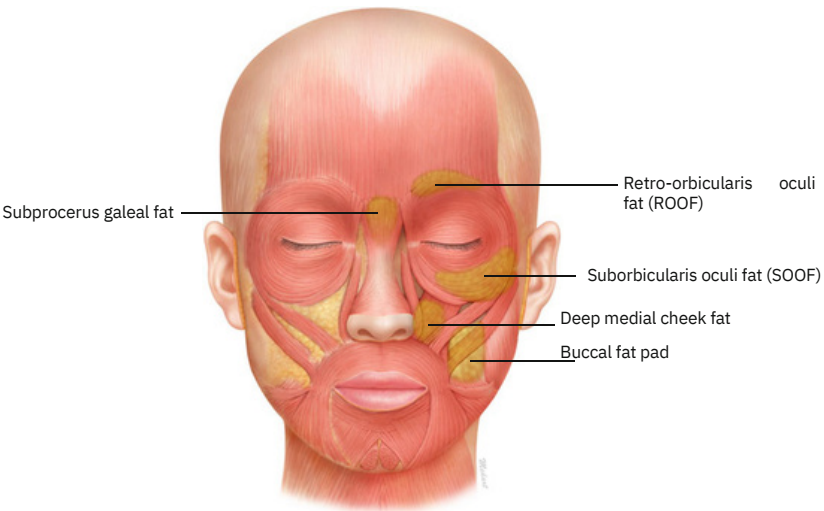
Superiorly, the SMAS passes over the zygomatic arch to meet with the superficial temporal fascia. The SMAS is considerably thicker over the parotid gland, but thins substantially as it courses medially. Superior to the zygomatic arch, the SMAS is known as the superficial temporal fascia where it splits to accommodate the temporal branch of CN VII and the intermediate temporal fat pad.

Degenerative changes in the viscoelastic properties and three-dimensional structure of the SMAS result in ptosis. Researchers have hypothesized that there is earlier and more progressive aging in the mid-face due to a decreased amount of SMAS. With increasing age, retaining ligaments are at risk of weakening, thus leading to further ptosis of the masseteric SMAS and resultant jowl formation.

Due to the proximity of the SMAS to the temporal branch of CN VII, any dissection in this location should be performed deep to the superficial temporal fascia in order to avoid accidental denervation-related injuries.



Superficial fat and superficial muscles of the face



Deep fat compartments of the face

DEEP FAT

The deep fat comprises the medial and lateral suborbicularis oculi fat (SOOF), and the deep medial cheek fat. Whilst the majority of the SOOF is found inferior to the lateral aspect of the infraorbital rim, it is also found underneath the orbicularis oculi muscle. Other deep fat compartments include the temporal fat pad and a deep addition of this pad known as Bichat's fat pad. The deep, supraperiosteal fat layer is located beneath the SMAS. Although the SMAS is sandwiched between fat layers, there are bilaminar connecting membranes or fusion zones containing neurovascular structures.

Compared with the superficial fat layer, the deep fat layer is composed of segmental, large white lobules containing a scant system of thin fibrous septae. With aging, the deep fat layers may disintegrate and descend, resulting in a more prominent appearance of the inferior border of the orbicularis oculi which may accentuate the malar crescent and the nasojugal fold. Post-menopausal changes due to decreased estrogen may cause increased fat deposition in combination with decreased superficial fat.

FACIAL MUSCLES

MUSCLE

The facial muscles can be categorized as periocular and perioral and broadly organized into four layers, where CN VII runs between the deepest and third layer. The first, superficial layer consists of the orbicularis oculi, the zygomaticus minor, and the depressor anguli oris. The second layer contains the levator labii superioris alaeque nasi, the zygomaticus major, the risorius, the depressor labii inferioris, and the platysma. The third layer includes orbicularis oris and levator labii superioris. The final, deepest layer consists of the buccinator, the levator anguli oris, and the mentalis. Whilst the major function of facial muscles relates to facial movement, they also play a significant role in maintaining soft-tissue support. The SMAS unites and advances the facial muscles, especially the zygomaticus major and orbicularis oris.

The mimetic muscles of the cheek are separated into a superficial and deep layer. The superficial layer consists of zygomaticus major and minor, levator labii superioris, risorious, depressor anguli oris, orbicularis oculi, and the orbicularis oris. The deep layer contains the levator anguli oris, buccinator, depressor labii inferioris, and the mentalis.

Muscular aging can cause prominent changes such as declining muscle mass and strength. An example of this can be seen in the midface, where the orbicularis oris thins with age while the orbicularis oculi does not. Extensive investigations of facial MRIs at different ages have shown that the midface muscles start to shorten and straighten simultaneously.

Researchers have hypothesized that this, in addition to a lifetime of facial contractions, may cause prolapse of the deep midfacial fat compartments.

MUSCLES OF FACIAL EXPRESSIONS AND THEIR ACTIONS

Facial mm. are attached to the facial skeleton, or membranous superficial fascia, beneath the skin, or subcutaneous tissue. The topography of the facial m. varies between males and females and between individuals of the same gender. It is important to defi ne muscle shapes, their associations with the skin, and their relative muscular actions in order to explain the unique expressions people can make.

The face divides into nine distinct areas:

(1) the forehead including glabella from eyelids to hair line
(2) temple or temporal region anterior to the auricles
(3) orbital region
(4) nose region
(5) zygomatic region
(6) perioral region and lips
(7) cheek
(8) jaws, and
(9) auricle.

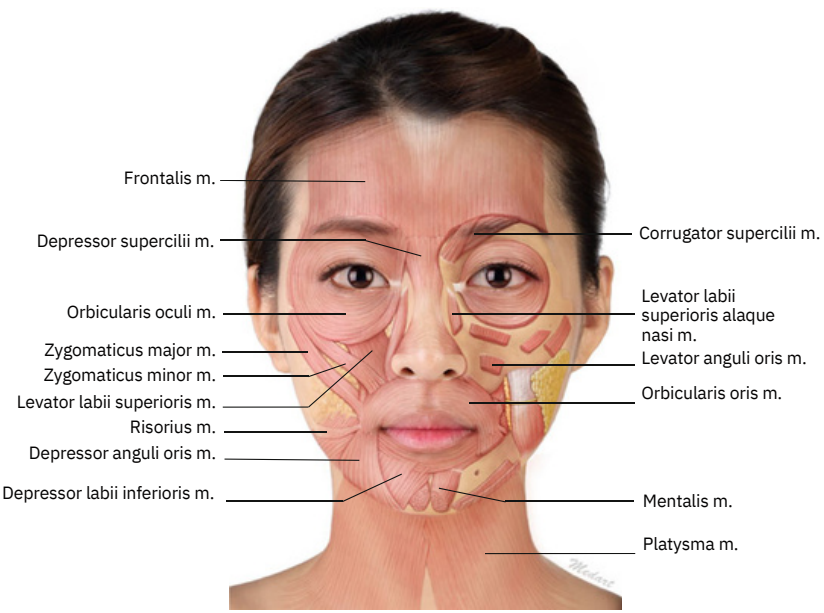
These muscles are distributed in different locations and (1) direct the openings of the orifices as dilators or sphincters and (2) form various facial expressions. These facial muscles, located within the superficial fascia, or subcutaneous tissue layers, originate from the facial bone or fascia and attach to the facial skin. They reveal various expressions such as sadness, anger, joy, fear, disgust, and surprise.

Facial mm. are widely distributed in different regions of the face. However, they are generally categorized different regions such as the forehead, the orbital, the nose, and other perioral regions. The platysma m., which is involved in the movement of the perioral region, is also considered a facial muscle.

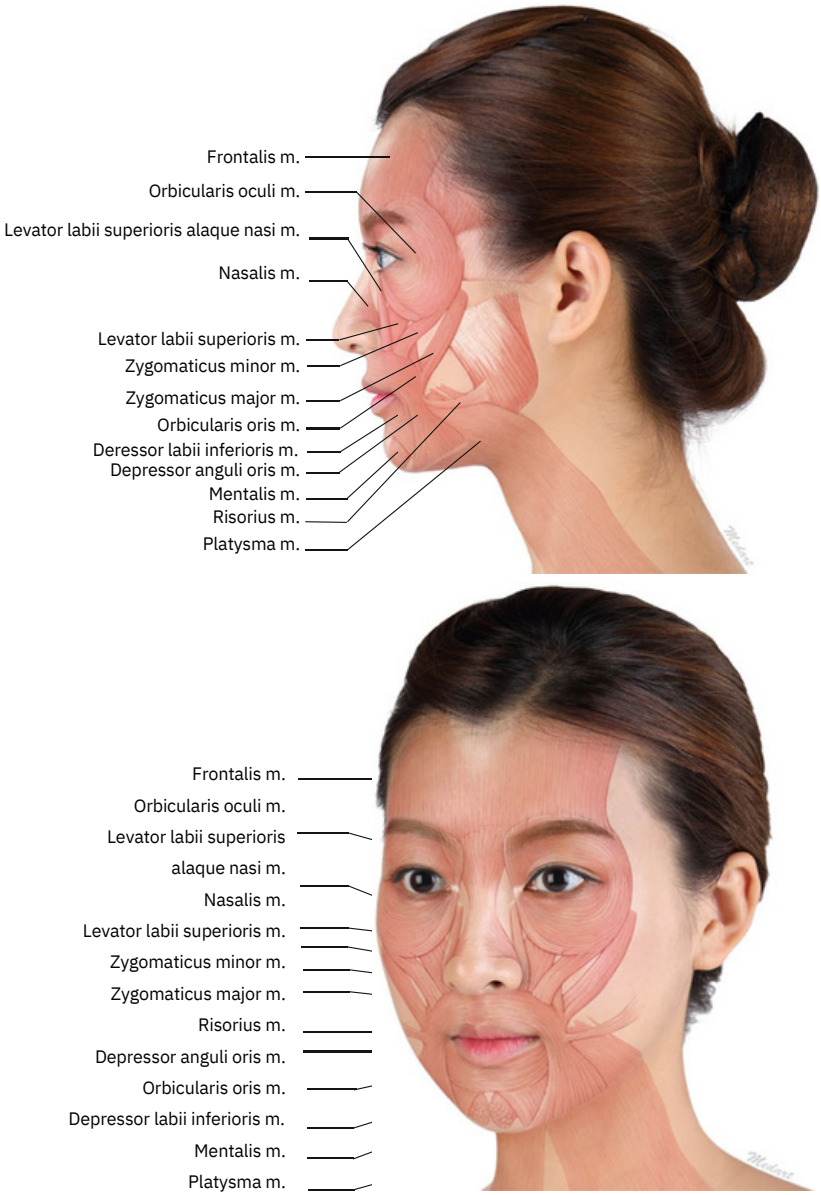
FOREHEAD REGION

The occipitofrontalis m. is a large, wide muscle underlying the forehead and the occipital area. It is divided into the frontal belly of the forehead region and the occipital belly of the occipital region. Clinically, the frontal belly of the occipitofrontalis m. is referred to as the "frontalis muscle" and arises from the galea aponeurosis and inserts into the orbicularis oculi m. and the frontal skin above the eyebrow. The width and contraction of the frontalis m. vary between individuals; during an individual's anxiety and surprise, this muscle produces transverse wrinkles on the forehead.

The frontalis m. is rectangular and possesses bilateral symmetry. Its muscle fibers are vertically oriented and join the orbicularis oculi and the corrugator supercilii m. near the superciliary arch of the frontal bone. The frontalis m. lies beneath the skin of the forehead (3–5 mm in average), though depth can differ considerably (27 mm) between individuals.



Facial muscles. (a)Frontal view, (b)lateral view, (c)oblique view



Facial muscles. (a)Frontal view, (b)lateral view, (c)oblique view

TEMPORAL REGION (OR TEMPLE)

The temporal region is confined within the boundary of the temporal fossa. Within the temporal fossa, a fan-shaped temporalis and its vessels and nerves occupy this concavity. The temporalis m. is divided into two layers: superficial and deep. A majority of the temporalis belong to the deep layer and arise from the broad temporal fossa, whereas the superficial layer of the temporalis m. arises from the internal aspect of the deep temporal fascia (temporalis muscle fascia). The deep temporal fascia (temporalis muscle fascia) is the tenacious fascia attached superiorly to the superior temporal line and inferiorly to the upper margin of the zygomatic arch. Though the superficial layer of the temporalis developed in four-legged animals, the superfi cial layer in human seems very thin and rudimentary. All the temporalis muscle fibers converge as a tendon and attach to the tip of the coronoid process and to the anteromedial side of the mandibular ramus. The temporalis holds a flat, fan shape due to its broader origin and narrower attachment.

There is a region in which the muscle fibers transition into tendons. The upper half of the temporalis superior to the zygomatic arch is composed only of the muscle belly, and the lower half (roughly twoor three-digit widths) is occupied by a converged tendon and a part of the deep layer of the temporalis that is covered by the aponeurotic structure.

The temporalis m. is divided into three parts: anterior, middle, and posterior temporalis m. While its anterior temporalis fibers proceed almost vertically, the fibers of the posterior temporalis run almost horizontally. The main functions of the temporalis differ according to muscular orientation. A whole temporalis m. raises the mandible for mouth closing, providing tension to prevent the mouth from opening against gravity. The temporalis m. is innervated by the anterior, middle, and posterior deep temporal nerves from the mandibular n. It is supplied by the anterior and posterior deep temporal arteries for the anterior 2/3 of the temporalis and by the middle temporal a. for the posterior 1/3 region as well.

ORBITAL REGION

The shape of the eyes is well framed by moving muscles that surround it, which determine basic facial expressions. Orbicularis oculi m. is a broad, fl at, elliptical muscle composed of an orbital part and a palpebral part. The palpebral part is then divided again into a superficial portion (ciliary bundle) and a deep portion (lacrimal part).

The main function of the orbicularis oculi m. is to mediate eye closure. The orbicularis oculi m. has many neighboring muscles (e.g., corrugator supercilii m., procerus m., frontalis m., zygomaticus major m., and zygomaticus minor m.), and various direct and indirect muscular connections exist between the orbicularis oculi m. and the surrounding musculature. These connections may participate in the formation of various facial expressions. In Asians, the lateral muscular band and the medial muscular band of the orbital portion of the orbicularis oculi m. are observed in 54 % and 66 % of the cases, respectively. Furthermore, it is observed that 89 % of Asians possess direct muscular connections between the zygomaticus minor m. and the orbicularis oculi m.

The corrugator supercilii m. originates from the periosteum of the frontal bone on the medial side of the superciliary arch, proceeds superiorly and laterally, and then merges with the frontalis m. It consists of two distinct bellies—the transverse and oblique belly. The origin of the transverse belly of the corrugator supercilii m. is superior and more lateral than the origin of the oblique belly, and most of them attach to the frontalis m. and to the superolateral orbital part of the orbicularis oculi m. The transverse belly is located deeper and proceeds in a more horizontal direction than the oblique belly. This muscle makes narrow, vertical wrinkles on the glabellar region and presents an aged appearance by producing these wrinkles with the frontalis m. The depressor supercilii m. is a fan-shaped or triangular-shaped muscle that originates from the frontal process of the maxilla and from the nasal portion of the frontal bone above the medial palpebral ligament. The depressor supercilii m. proceeds through the glabellar region while being mixed with the corrugator supercilii m., and it intermingles with medial fibers of the orbicularis oculi m.

NOSE REGION

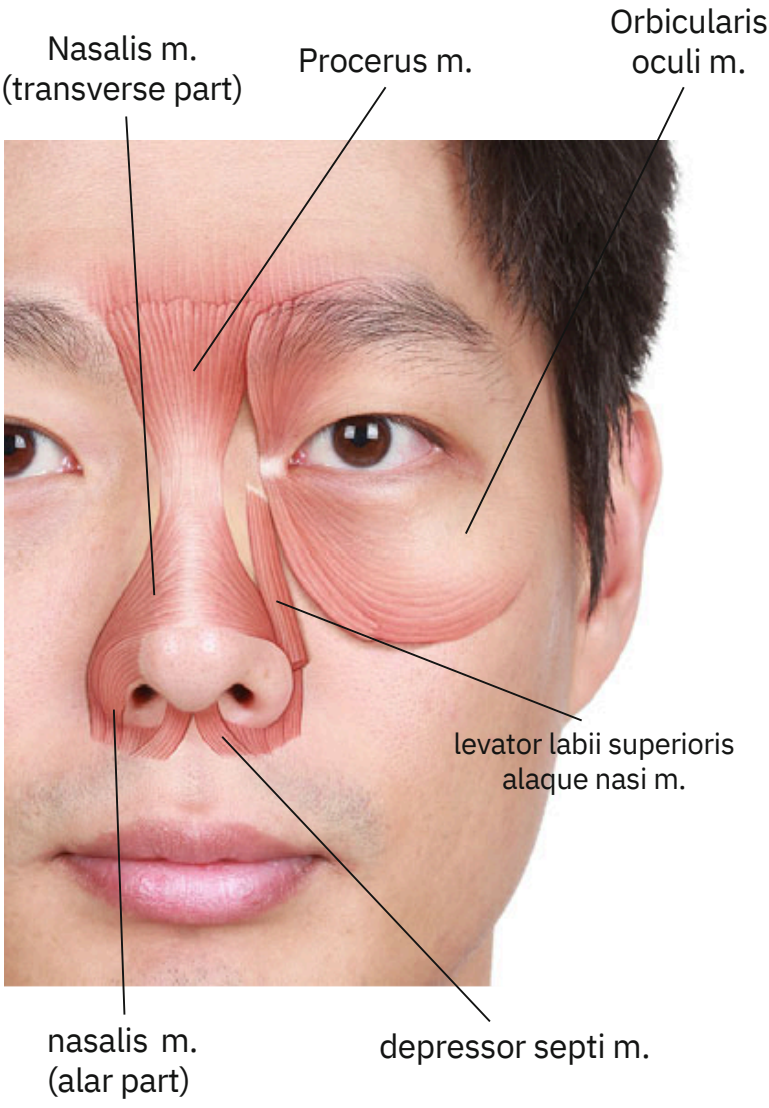
The nose is a dynamic structure that moves nasal cartilages and plays an important role in the nasal physiology. Muscles of the nose and the nose region contain of the procerus m., the nasalis m., and the depressor septi nasi m., along with several other muscles attached to the nasal ala.

The procerus m. is a small muscle that originates from the nasal bone, proceeds superiorly, and attaches to the skin of the radix. Fibers of the frontalis m. at the insertion point are cross-locked. This muscle makes a horizontal line on the radix below the glabella by pulling the medial side of the eyebrow down.

The nasalis consists of a transverse part and an alar part. The transverse part is a C-shaped, triangular muscle raised from the maxilla and the canine fossa to the nasal ala. The transverse part extends from the superfi cial layer of the levator labii superioris alaeque nasi m. The alar part is a small rectangular muscle arising from the maxilla superior to the maxillary lateral incisor and inserting into the deep skin layer of the alar facial crease of the alar cartilage. The transverse part compresses and decreases the size of the naris, while the alar part serves to enlarge the size of the naris.

The depressor septi nasi m. is located on the deep part of the lip. This muscle arises from the incisive fossa (between the central and lateral incisors) and inserts into the moving part of the nasal septum. It pulls the nose tip inferiorly to enlarge the size of the naris.

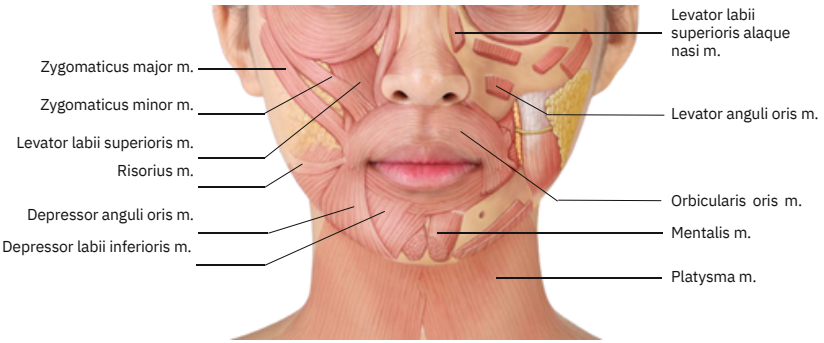
Furthermore, it was observed that all of the LLSAN m., 90 % of the LLS m., and 28 % of the additional fibers of the zygomaticus minor m. were attached to the nasal ala.



Perinasal muscles

PERIORAL MUSCLES

Intrinsic Muscles of the Lip and Cheek



Perioral muscles

Orbicularis Oris Muscle (OOr)

The orbicularis oris m. is a mouth constrictor surrounding the mouth region. Most muscle fibers are continuations from various muscles in the mouth region. Intrinsic orbicularis oris muscle fibers originate from the alveolar bone of the maxillary and mandibular incisors. This muscle works to close the mouth and pucker the lips.

Buccinator Muscle

The buccinator m. originates from the lateral side of the alveolar portion of maxillary and mandibular molars and from the anterior border of the pterygomandibular raphe. The buccinators consist of four bands: the first band (the superior band) originating from the maxilla, the second band originating from pterygomandibular raphe, the third band originating from the mandible, and the fourth band (the inferior band) originating inferiorly to the third band, extending inferiorly, and medially proceeding inferiorly to the orbicularis oris muscle fibers. The inferior band, unlike other bands, continues bilaterally to the median plane of the mandible.

Dilators of the Lips

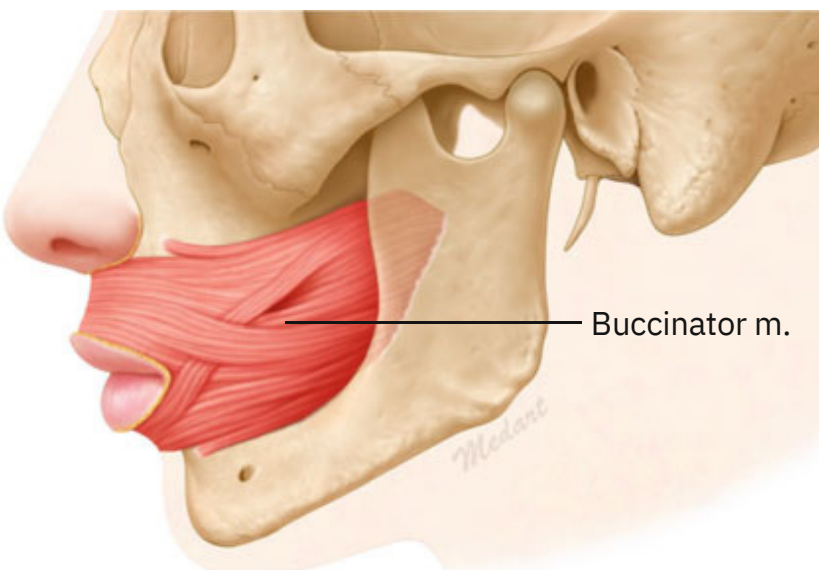
Muscles Inserted into the Modiolus

Zygomaticus Major Muscle (ZMj)

The zygomaticus major m. originates from the facial side of the zygomatic bone, proceeds inferiorly and medially, joins the orbicularis oris m., and attaches to the modiolus. Thus, the well-known function of the ZMj is elevating the mouth corner. However, the insertion pattern varies, and the fiber running deeper than the levator anguli oris m. is always observed. These fibers insert into the anterior region of the buccinators,

Levator Anguli Oris Muscle (LAO)

The levator anguli oris m. originates from the canine fossa inferior to the infraorbital foramen, joins the orbicularis oris m., and attaches to the modiolus. It serves to elevate the mouth corner



Buccinator muscle (depressor anguli oris muscle (DAO) is reflected superiorly to show the mandibular portion attachment at the buccinators)

Depressor Anguli Oris Muscle (DAO)

The depressor anguli oris m. is a triangular muscle that is on the most superficial layer of the perioral m. along with the risorius m. It arises from the oblique line of the mandible and merges with the depressor labii inferioris m. at the origin. This muscle becomes more narrow, proceeds to the mouth corner (modiolus), and merges with the risorius m

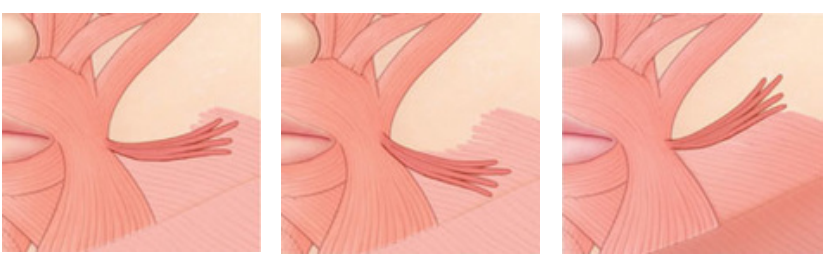
Risorius Muscle

The risorius m. is a thin and slender muscle. This muscle is predominantly located 20–50 mm lateral to the mouth corner and 0–15 mm below the intercheilion horizontal line. Most fibers originate from the superficial musculo aponeurotic system (SMAS), the parotid fascia, and the masseteric fascia. It sometimes also originates from the platysma m. Its fibers insert into the modiolus and pull the mouth corner when smiling

Muscles Inserting into the Upper and Lower Lip Between the Labial Commissure and the Midline

Levator Labii Superioris Muscle (LLS)

The levator labii superioris m. originates from 8 to 10 mm inferior to the infraorbital margin of the maxilla and inserts into the lateral side of the upper lip. The levator labii superioris m. is rectangular shaped rather than triangular shaped, and its medial fibers are attached to the deep side of the alar facial crease. Also, 90% of the muscle is mixed with the alar part of the nasalis m. A part of the deep tissue of the levator labii superioris m. extends to the skin of the nasal vestibule.



Risorius muscles. Three patterns of the risorius muscle, a) platysma-risorius, b) triangularis-risorius c) zygomaticus-risorius

Levator Labii Superioris Alaeque Nasi Muscle (LLSAN)

The levator labii superioris alaeque nasi m. originates from the frontal process of the maxilla and inserts into the upper lip and the nasal ala. The levator labii superioris alaeque nasi m. is divided into superficial and deep layers. The superficial layer proceeds inferiorly to the surface layer of the levator labii superioris m., and the deep layer proceeds even deeper than the levator labii superioris m. The deep and superficial layer of the levator labii superioris m. originates from the frontal process of the maxilla and inserts between the levator anguli oris and the orbicularis oris m.

Modiolus

The modiolus is a fibromuscular structure that decussates between the orbicularis oris m. and the dilators of the lips ending at the lateral border of the cheilion. The modiolus m. lies either superior or inferior to the intercheilion line. It is strongly associated with facial expression, beauty, aging, and formation of the nasolabial fold. In Asians, the modiolus lies 11.0±2.6 mm lateral, 8.9±2.8 mm inferior to the cheilion, and inferior to the intercheilion line. These characteristics are mostly common in Asians, differing from Caucasians whose modiolus lies on or is superior to the intercheilion line.

Muscles that terminate at the modiolus implement formations of subtle and detailed facial expressions. The modiolus m. is a dense, compact, and mobile muscular node formed by a convergence of muscle fibers from the zygomaticus major, the depressor anguli oris, risorius, the orbicularis oris, buccinators, and the levator anguli oris. In 21.4% of Koreans, the modiolus showed tendinous tissue instead of muscular tissue as described above, and this area of convergence consisted of dense, irregular, and collagenous connective tissue

Zygomaticus Minor Muscle (Zmi)
The zygomaticus minor m. originates from the zygomatic bone and inserts into the upper lip. In Korean cases, 28% showed additional fibers inserting into the nasal ala in addition to the upper lip

Depressor Labii Inferioris Muscle (DLI)

The depressor labii inferioris m. originates from the oblique line of the mandible and inserts into the lower lip

Upper Lip Elevators

The shape of the upper lip is directed by upper lip elevators, which consist of the levator labii superioris alaeque nasi, the levator labii superioris, and the zygomaticus minor m. These muscles are used to elevate the upper lip and create smiling or sad facial expressions. Upper lip elevators are categorized into two layers with the levator labii superioris alaeque nasi m. and the zygomaticus minor m. being located on the medial and lateral side, respectively, and partially or completely covering the levator labii superioris, which is located on a deeper layer.These three muscles are localized on the lateral side of the nasal ala. Upper lip elevators are attached to the surface of the orbicularis oris m. and are involved to form the nasolabial fold.

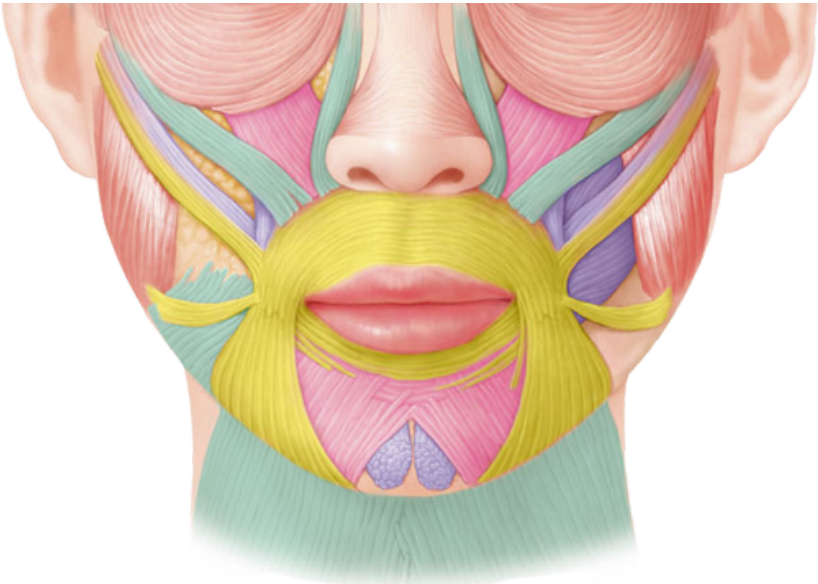
Contracting Muscle of the Chin

Mentalis Muscle

The mentalis m. elevates the chin and the lower lip and provides major vertical support for the lower lip. Resection of the mentalis m. may cause the patient to drool and may affect the denture stability. This muscle is cone shaped with its apex originating from the incisive fossa of the mandible. Its medial fibers descend anteromedially and cross together, forming a dome-shaped pattern. Contraction of the mentalis m. produces a wrinkle in the skin of the mentum.

Layers of the Perioral Muscles

The perioral m. is categorized into four layers according to depth, which is then further specified into three superficial layers and one deep layer.



The layers of the perioral musculature (yellow first layer, blue second layer, pink third layer, purple fourth layer)

Superficial Layer

- First layer

Depressor anguli oris, risorius, superficial layer of the orbicularis oris m., and superficial layer of the zygomaticus major m.

- Second layer

Platysma, zygomaticus minor, and levator labii superioris alaeque nasi

- Third layer

Levator labii superioris, deep layer of the orbicularis oris m., and deep layer of the depressor labii inferioris m.

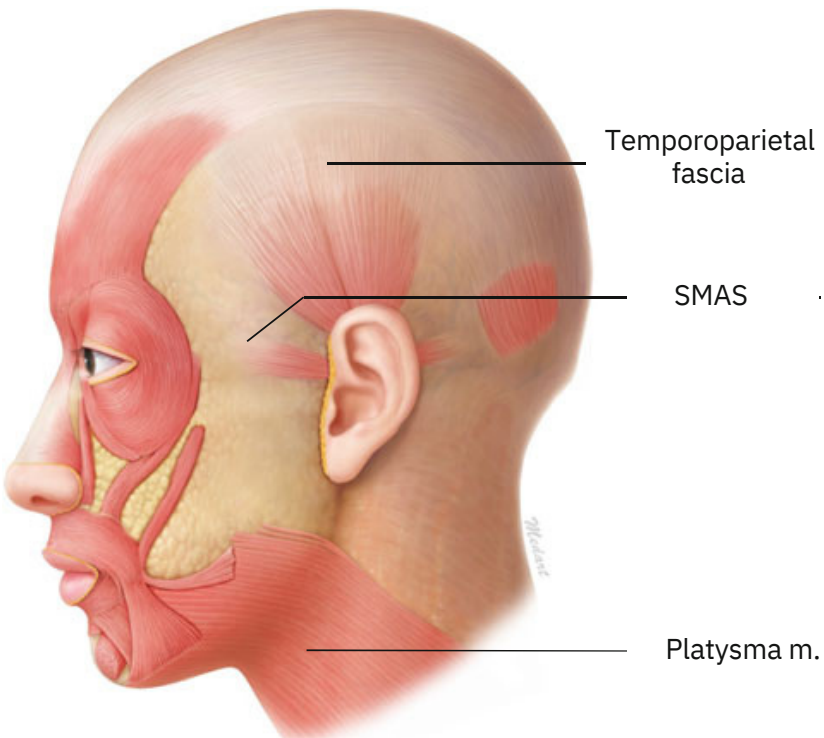
Deep Layer

- Fourth layer

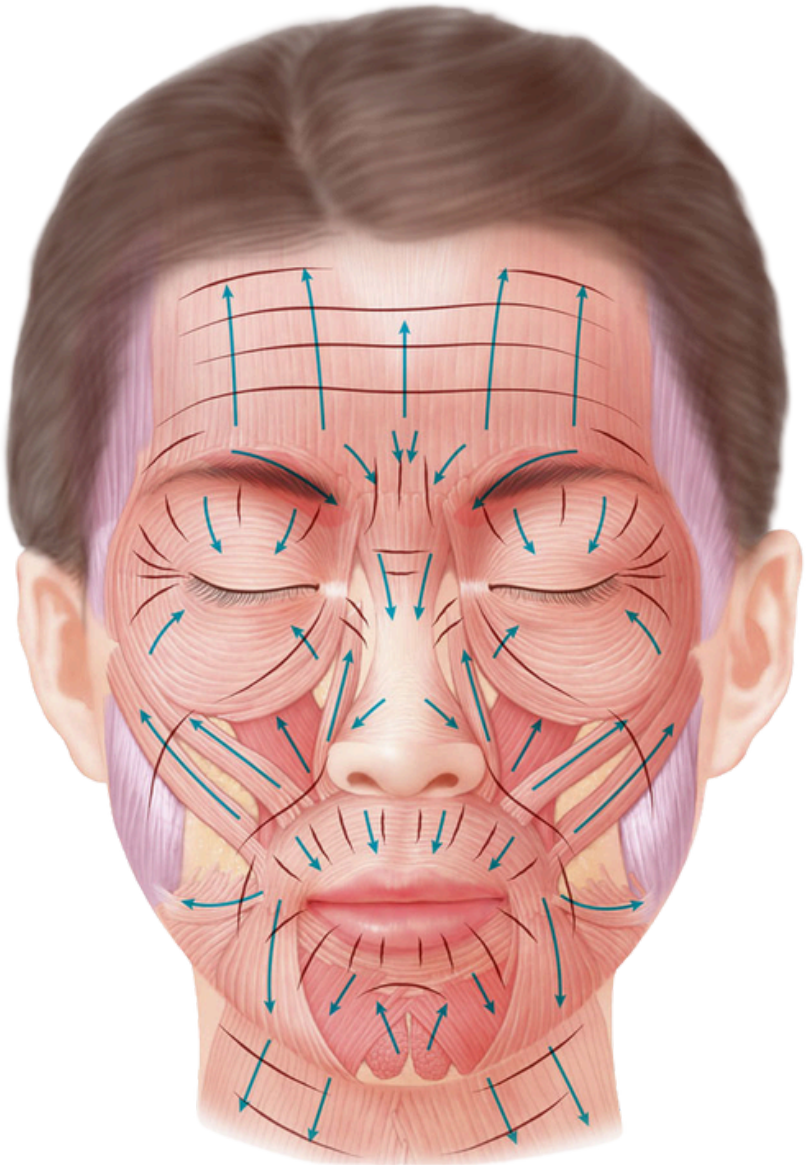
Levator anguli oris, mentalis, deep layer of the zygomaticus major m., and buccinator

Platysma Muscle

The platysma m. attaches to the lower border of the mandible and to the mandibular septum and also merges with the facial m. around the lower lip. It consists of two types of fibers. A flattened bundle passes superomedially to the lateral border of the depressor anguli oris, and the other type remains deep into the depressor anguli oris and reappears at its medial border. Lack of decussation creates a cervical defect, resulting in an elasticity reduction in the cervical skin and giving rise to the so-called gobbler neck deformity with age, Asians experience fewer cases than Caucasians of lacking decussation, which then leads to fewer cases of the "gobbler neck." Platysmal fibers do not merely decussate but also sometimes show cases of interlacing from each side or of one side of the muscle_overlapping and covering the other side



Superficial musculoaponeurotic system (SMAS) illustration.



Facial muscles and their actions (arrows)

FACIAL LIGAMENTS

SMAS LAYER AND LIGAMENTS OF THE FACE

The superficial musculoaponeurotic system (SMAS) is a continuous fibromuscular layer investing and interlinking the muscles of facial expression. It has been found to consist of three distinct layers: a fascial layer superficial to the musculature, a layer intimately associated with the mimic m., and a deep layer extensively attached to the periosteum of the bones of the face. In addition to its usefulness as a deep layer to tighten during an aging face surgery, it serves as a guide to the depth of key neurovascular structures.

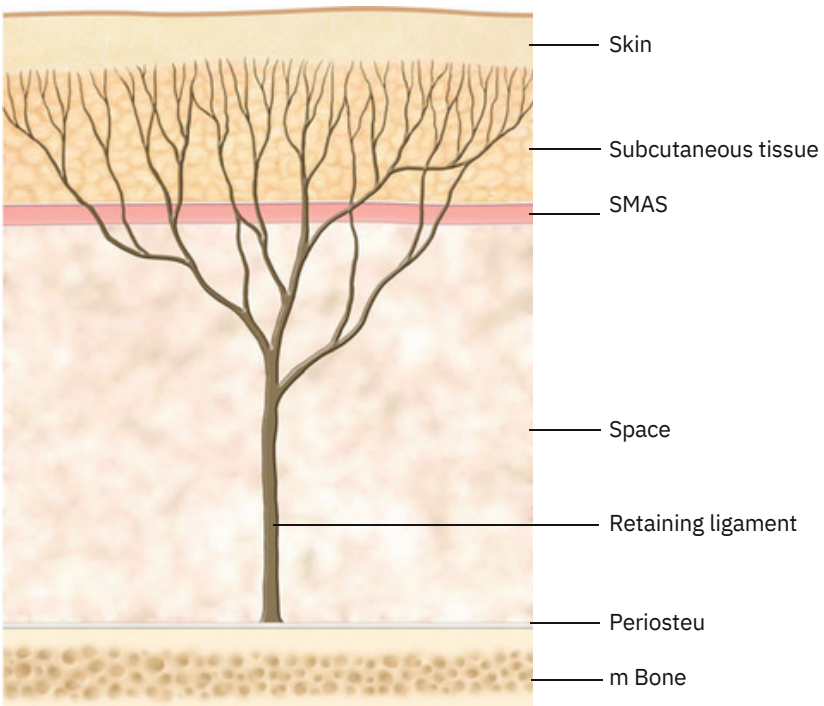
The face, like other body parts, also has several ligament structures, which firmly supports surrounding tissues. This retaining ligament is broadly and firmly attached from the periosteum, or fascia, to the dermis. These strong retaining ligaments in the face can be divided into true (osteocutaneous) and false (fasciocutaneous) ligaments according to its strength, attachment, and function.

The true retaining ligament originates from the periosteum, attaches to the dermis, and gives strong support to the soft tissue. True retaining ligaments consist of the orbicularis retaining ligament, the zygomatic ligament, the zygomatic cutaneous ligament, the lateral orbital thickening, the mandibular ligament, etc.

There are multiple false retaining ligament attachments that exist at sequential facial planes. These attachments emanate from the dermis and attach to the underlying SMAS, but it does not retain strongly. The false retaining ligaments are particularly strong over the forehead, eyes, nose, lip, and chin areas. They are of intermediate strength over the lateral cheek and neck areas and tend to be relatively loose over the medial cheek and temple areas. Therefore, they easily lose elasticity and sag with age, causing changes in facial features due to fat redistribution and drooping. False retaining ligaments consist of the masseteric cutaneous ligament, the platysma-auricular ligament, etc.

Superior Temporal Septum

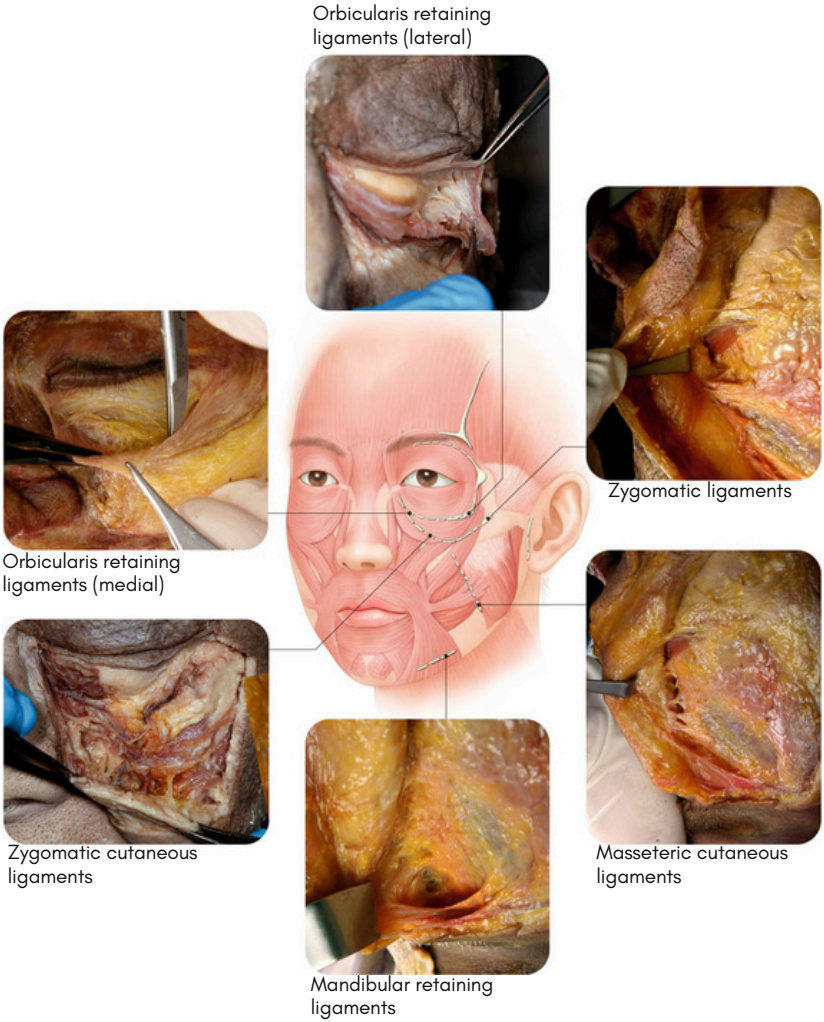
The superior temporal septum's fascia adheres to the superior temporal line of the skull. This structure appears to be merging with the temporal fascia and the periosteum of the skull. This merging ends as a temporal ligamentous adhesion at the lateral third of the eyebrow and occurs 10 mm superior to the supraorbital margin with a height of 20 mm and a width of 15 mm.



Schematic illustration of the retaining ligament from the periosteum to the skin.

Zygomatic Ligament

The zygomatic ligament, also known as the McGregor's patch, is located posterior to the origin of the zygomaticus minor m. This structure is a true retaining ligament that connects the lower margin of the zygomatic arch to the skin.



The retaining ligaments of the face

Zygomatic Cutaneous Ligament

The zygomatic cutaneous ligament originates from the periosteum of the zygomatic bone, proceeds along the lower margin of the orbicularis oculi m., and attaches to the skin on the anterior portion of the zygomatic bone. The soft tissues in this area are maintained by the ligament, which droops with age in the form of a malar mound (or baggy lower eyelid).

Orbicularis Retaining Ligament

The orbicularis retaining ligament is located superiorly, inferiorly, and laterally along the orbital rim. It attaches to the lateral periosteum of the orbit and extends to the deep portion of the orbicularis oculi m.

Lateral Orbital Thickening

Lateral orbital thickening is located on the superolateral side of the orbital margin and originates from the orbital retaining ligament.

Mandibular Retaining Ligament

The mandibular retaining ligament connects the periosteum of the mandible, located right underneath the origin of the depressor anguli oris m., to the skin.

Masseteric Cutaneous Ligament

The masseteric cutaneous ligament is a false retaining ligament originating from the anterior border of the masseter m. This ligament attaches to the SMAS and to the skin covering the cheek. It attenuates with age and causes the SMAS to sag and jowl.

Platysma-Auricular Fascia (PAF)

The platysma-auricular fascia is a compact fi brous tissue located inferior to the ear lobule where the lateral temporal-cheek fat compartment and the postauricular fat compartment merge.

FACIAL VASCULATURE

VASCULATURE

Three major arteries originating directly from the external carotid artery or subsequent branches provide arterial supply to the face: the facial, transverse facial, and infraorbital arteries. The facial artery, which is the largest, crosses the inferior border of the mandible just anterior to the masseter, where its pulsation may be felt, after which it travels in a coiled fashion towards the pyriform fossa. It runs from deep on the mandible, over the buccinator, beneath risorius and zygomaticus major, under or over zygomaticus minor, crosses the nasolabial fold from medial to lateral at the junction of the proximal third after which it becomes the angular artery which anastomoses with the superficial temporal artery (STA).

The ophthalmic artery is the major artery supplying the orbit. Originating from the internal carotid artery in the middle cranial fossa, this artery traverses the optic foramen and subdivides into numerous branches inside the orbital cavity.

The superficial temporal artery represents the final branch of the external carotid artery. This artery arises inside the parotid gland at the point where the maxillary artery branches off the external carotid artery. Bilaterally, this artery supplies a large area of facial skin, including the lateral forehead, the temple, the zygoma, and the ear. One prominent branch that stems from the superficial temporal artery includes the transverse facial artery (also originating from the parotid gland).

The forehead is supplied by the supraorbital and supratrochlear arteries (branches of the ophthalmic artery). The nose has a particularly intricate vascular network of tiny arteries within the alae, tip and columella. Most of this is supplied by the lateral nasal artery (originates from the facial artery) or superior labial artery (also originates from the facial artery). The upper lip is supplied primarily by the superior labial artery, while the lower lip is supplied by three labial arteries. The chin's main vasculature is the mental artery (branch of the inferior alveolar artery.

The majority of veins are located close to the similarly named arteries.

The majority of veins are located close to the similarly named arteries.

After crossing the inferior mandibular border with the facial artery, the facial vein takes a direct path to the medial canthus. The lateral forehead and temporal/parietal regions usually drain via the superficial temporal vein, while the middle forehead and upper eyelid drain via the angular or ophthalmic veins within the cavernous sinus.

Venous drainage of the midface is via the infraorbital vein and pterygoid plexus; certain structures, such as the lips and cheeks drain into the facial vein.

The location, size and origin of the major arteries may vary between individuals and races. With aging, random degenerative changes can occur in individual vessels, including increased diameter, decreased elasticity, and arterial hypertension. These changes can result in elongation and further tortuosity of these arteries.

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Facial Vessels and Their Distribution Patterns

Facial blood vessels are extremely important. As filler injections become more common, blood vessel-related issues, such as skin necrosis and blindness, will become more prominent. Therefore, more in-depth studies on blood vessel pathways in terms of injection techniques are required.

Clinically, facial blood vessels do not follow one specific pattern. Dissections show many variations of this pattern. Furthermore, facial blood vessels contain not only arteries but also veins and their branches. It is impossible to perfectly avoid every single blood vessel during blind injections. However, with enough knowledge of these vessels, it is possible to minimize risks and perform a safe injection.

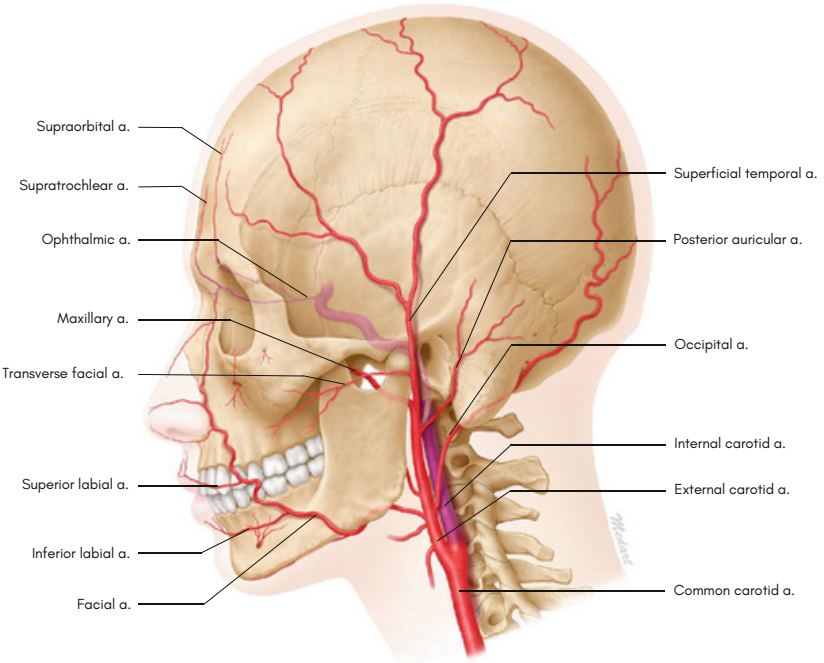
The blood supply of the head and neck is mostly given by the common carotid a. The right common carotid a. and the right subclavian a. are arising from the brachiocephalic trunk. On the other side, the left common carotid a. and the left subclavian a. are arising independently from the aortic arch. At the level of the superior border of the thyroid cartilage, the common carotid a. divides into internal and external carotid arteries. The pulse of the common carotid a. can be felt when touching the anterior border of the sternocleidomastoid m. at the level of the thyroid cartilage.

The internal carotid artery has no other arterial branches except the ophthalmic a. before reaching the brain. The internal carotid a. runs anteromedially through the carotid canal and enters the middle cranial fossa. The internal carotid a. supplies blood to the cerebrum, and a portion enters the orbit area, arrives at the superomedial side of the orbital, and supplies blood to the eye, the orbit, and the lacrimal gland.

The external carotid a. originates from the common carotid a. in the area of the carotid sheath. Although the origin of the external carotid a. lies anteriorly and medially from the internal carotid a., it locates further laterally as it ascends. This artery divides into eight branches.

The facial blood supply is given by the internal carotid a. and the external carotid a. These arteries are accompanied by the corresponding sensory n.

On the superficial layer of the skin, branches of the external carotid a. (facial a., superficial temporal a., facial branches of the maxillary a.) and branches of the internal carotid a. (supraorbital a. branching from the ophthalmic a., supratrochlear a., infratrochlear a.) supply blood to this layer



External and internal carotid arterial system and their branches

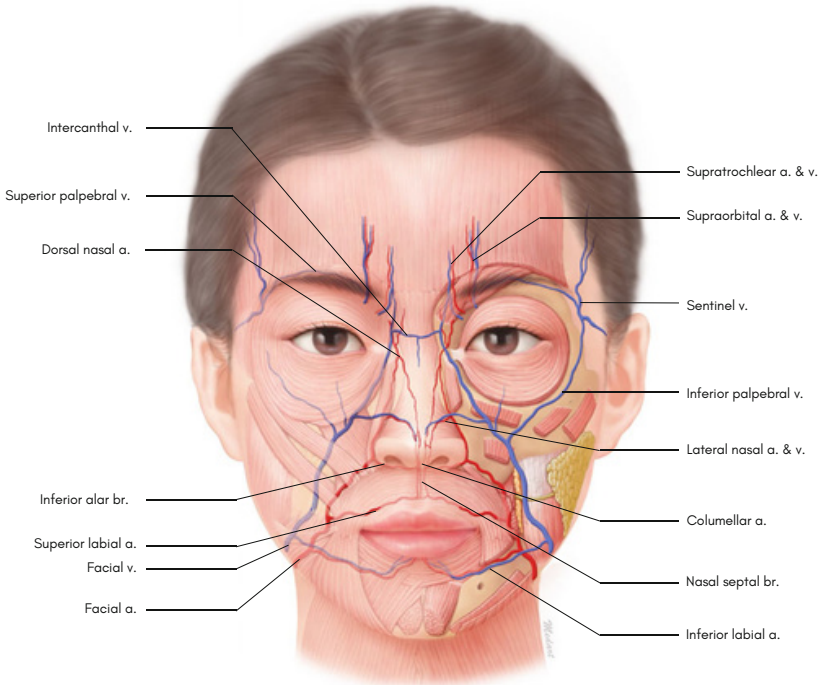
Facial Branches of the Ophthalmic Artery

Supraorbital Artery

The supraorbital a., together with the supraorbital n., originates from the supraorbital notch, or the supraorbital foramen, and supplies the upper eyelid, forehead, and the scalp region.

Supratrochlear Artery

The supratrochlear a. runs more medially than the supraorbital artery and supplies the upper eyelid, the forehead, and the scalp.



General courses and locations of the artery and vein on the face

Dorsal Nasal Artery

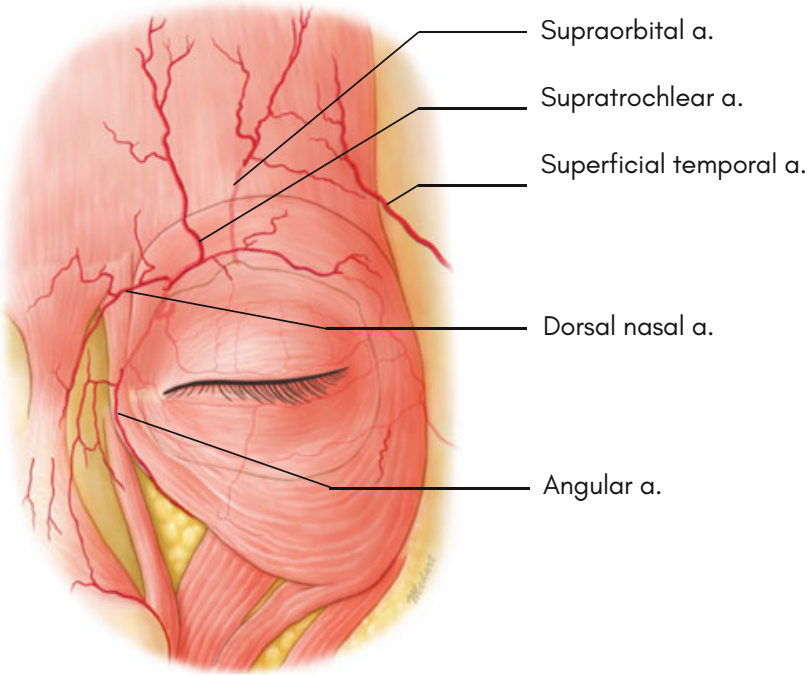
The dorsal nasal a. originates from the medial canthus of the orbit together with the infratrochlear n. and supplies the medial portion of the upper eyelid, the lacrimal sac, and the dorsum of the nose.

Lacrimal Artery

The lacrimal a. is the last, small part of the ophthalmic a. that originates from the lateral side of the supraorbital margin and supplies the lateral side of the upper eyelid.

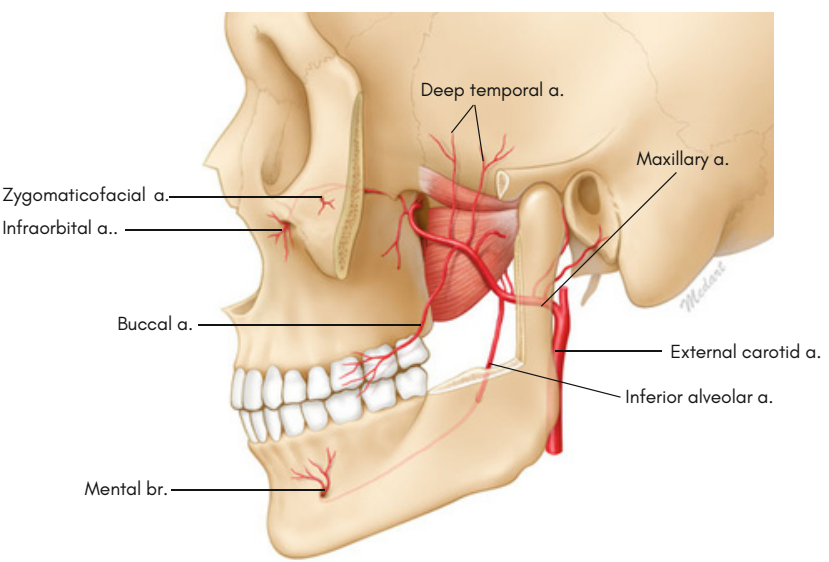
External Nasal Artery

The external nasal a. runs through the junction between the nasal bone and the nasal cartilage. It supplies the intermediate zone of the external nose inferior to the nasal bone.



Periorbital arterial distribution of the ophthalmic artery (internal carotid arterial system) (a, b)

Facial Branches of the Maxillary Artery



Maxillary artery and its branches

Infraorbital Artery

The infraorbital a. exits the infraorbital foramen inferior to the orbit and branches to the inferior palpebral branch, the nasal branch, and the superior labial branch.

Zygomatic Artery

Two branches of the zygomatic a. (zygomaticofacial branch and zygomaticotemporal branch) pass along the zygomatic canal on the lateral wall of the orbit. The zygomaticofacial branch exits the zygomaticofacial foramen and supplies the zygomatic region and the cutaneous layer of the cheek. The zygomaticotemporal branch exits the zygomaticotemporal foramen and supplies the cutaneous layer of the temporal region.

Buccal Artery

The buccal a. runs to the muscle internally between the ramus of the mandible and the masseter m. It branches to the surface of the cheek and supplies the cutaneous and mucosal layer of the cheek and the molar gingiva on the buccal side.

Mental Artery

The mental a. branches from the inferior alveolar a. inside the mandibular canal. It exits the mental foramen along with the mental n. and supplies the chin, the lower lip, and the mandibular incisive gingiva.

Facial Artery

The facial a. branches from the external carotid a., winds through the antegonial notch, passes the masseter m. anteriorly, and runs tortuously to the nasion and the glabella. It is known that the facial a., which runs superomedially through the face, branches to the inferior labial a., the superior labial a., and the lateral nasal a. and terminates as the angular a.

The facial a. is described in many textbooks as running from the mandibular angle to the radix and is in charge of most of the blood supply to the face. The facial a. continues all the way to the angular a. in only 36.3% cases among 91 Korean hemifaces. In other races, the angular a. was observed in 4% of French hemifaces, 12% of Japanese hemi-faces, 22% of Turkish hemifaces, and 68 % of British hemifaces. Although the research presented differences of angular a. occurrences among various ethnicities, the actual cause for that difference is still unclear, because the fractions of populations observed to possess angular a. were quite different between French and British populations despite both of them being Caucasian. What is quite apparent, however, is the fact that general documentation stating that the facial a. proceeds to the angle of the orbit seems erroneous.

Facial a. symmetry is observed in only 30 % of the cases, and regions with sparse blood supply are supplied additionally by branches of the superficial temporal a. (transverse facial a., supraorbital a., supratrochlear a.), branches of the ophthalmic a., and branches of the maxillary a. (infraorbital a., mental a.). The more prominent arteries on the opposite side of the face can also supply these regions.

Facial Artery Branches

Superior, Inferior Labial Branch

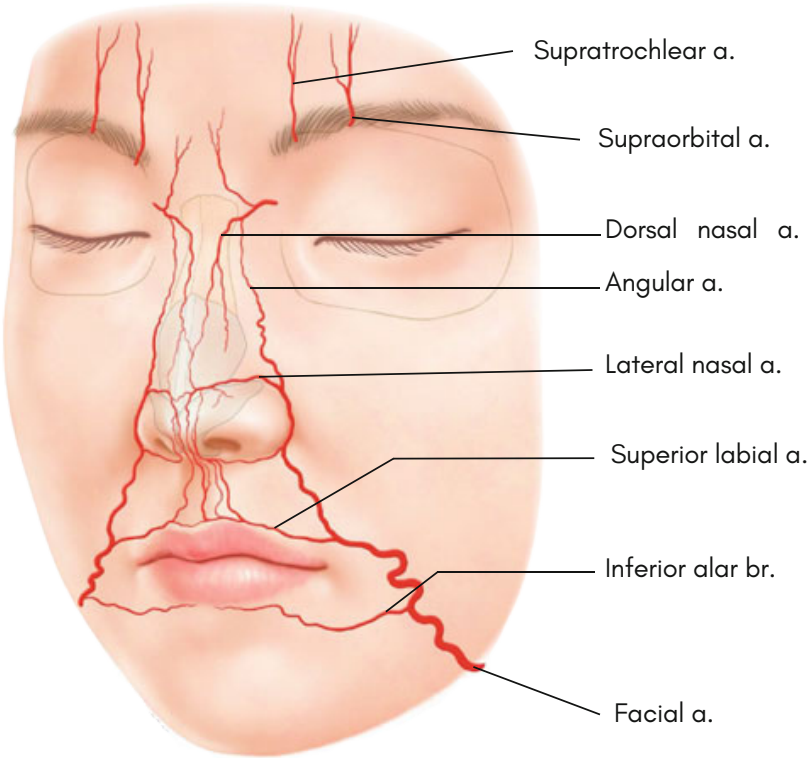
The facial a. proceeds obliquely and superiorly to the angle of the mouth, and branches of the superior labial a. to the upper lip and branches of the inferior labial artery to the lower lip appear.

Inferior Alar Branch

The inferior alar branch divides off from the facial a. immediately adjacent to the nasal ala and runs to the columella. It merges with columellar branches from the superior labial a. and forms an artery that runs through the columella all the way to the nasal tip.

Lateral Nasal Branch

The lateral nasal branch supplies the nasal ala and the nasal bridge,divides lateral to the nasal ala, and runs along the lateral side of the nose. It continues to the nasal branch of the infraorbital a. and the external nasal branch of the ophthalmic a.



General concept about the course of the facial artery. This concept is controversial according to many studies of the facial artery

Angular Artery

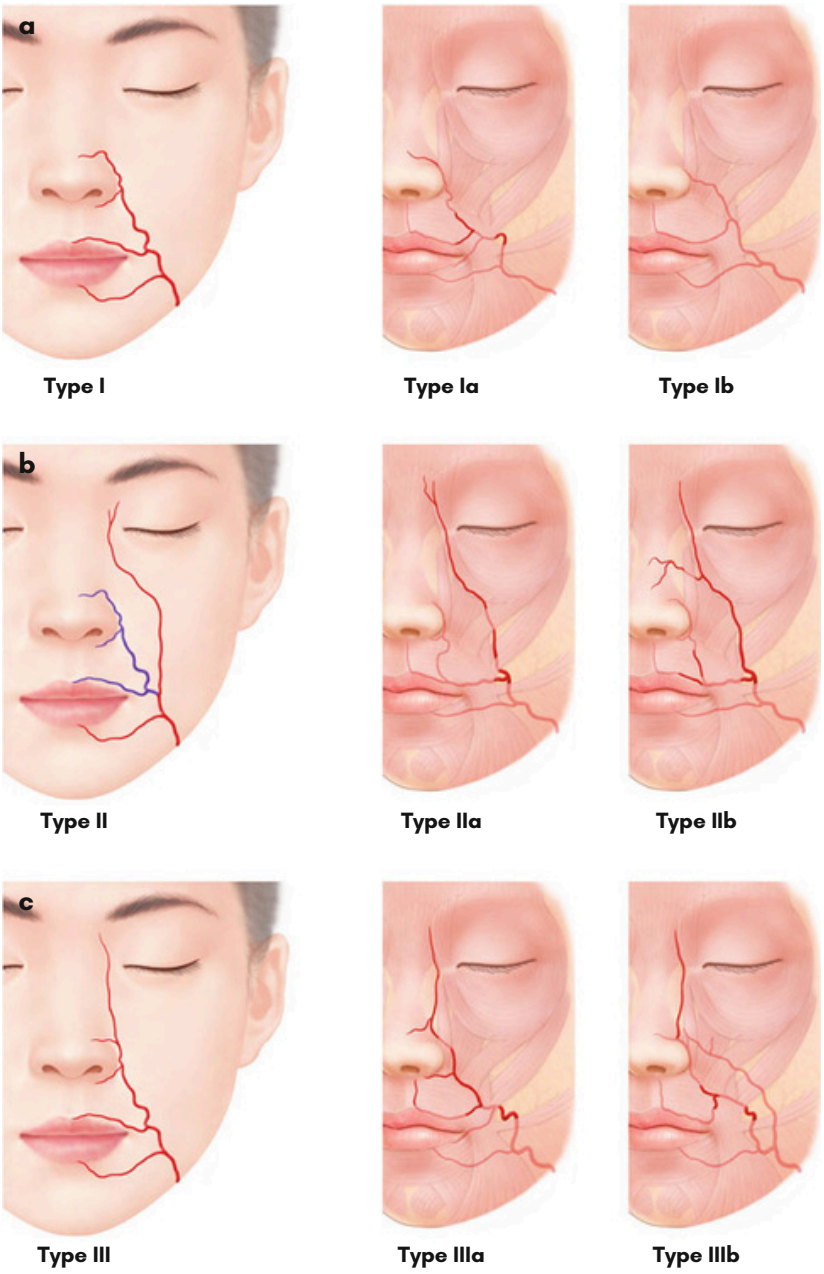
The angular a. is the terminal artery of the facial a. after it branches from the lateral nasal and runs superiorly to the canthus. It terminates at the medial canthus region and branches to the medial side of the eyelid and the nose. The angular a. sometimes branches from the ophthalmic branch rather than from the facial a., but it is observed to be the terminating branch of the facial a. in 51 % of the cases.

Typical Distribution Patterns of the Facial Artery

Branches of the facial a. are categorized into four types depending on their directions, locations, and supplying regions (lower lip, upper lip, nasal, and infraorbital region). The branching pattern can be generally organized into three categories depending on the region: type I (nasolabial pattern; 51.8%), type II (nasolabial pattern with infraorbital trunk; 29.6 %), and type III (forehead pattern; 18.6 %)

Frontal Branch of the Superficial Temporal Artery

The superficial temporal a. is the terminal branch of the external carotid a. that emerges from the facial side between the temporomandibular joint and the ear and runs superiorly to the scalp. It branches to the transverse facial a. immediately inferior to the ear and is located about 2 cm inferior to the zygomatic arch. The superficial temporal a. branches to both the frontal branch and the parietal branch 37 mm superior and 18 mm anterior from the tragus. The frontal branch runs obliquely toward the forehead and has either one branch (94.8 %) or two branches (5.2 %) that approach the frontalis m. past its lateral border and supply the region. The superficial temporal a. passes the lateral side of the head along with auriculotemporal n. It branches to the transverse facial a. approximately 1 cm inferior to the zygomatic arch, runs superiorly, and divides into the frontal branch, which supplies the lateral side of the forehead, and into the parietal branch, which supplies the parietal region. The transverse facial a. runs anteriorly, merges with the branch of the facial a., and supplies the parotid gland and the cheek.



Three patterns of the facial artery (FA). (a) Type I nasolabial pattern, (b) type II nasolabial pattern with infraorbital trunk, (c) type III forehead pattern (Ag angular a., LN lateral nasal a., IA inferior alar br., SL superior labial a., IL inferior labial a., FA facial a.)

Facial Veins

The facial v. follows the same distribution pattern as the facial a. with a few differences. Typically, the facial v. presents a greater amount of pattern variation than the facial a.

Veins with Cutaneous Nerves and Arteries

The facial v. runs in the direction opposite from the corresponding facial a. Veins of the forehead, the scalp, and the upper eyelid run to the superior ophthalmic v. on the orbit. The veins of the upper lip, the lateral side of the nose, and the lower eyelid run through the infraorbital v. to the infratemporal region and the pterygoid plexus.

Facial Vein

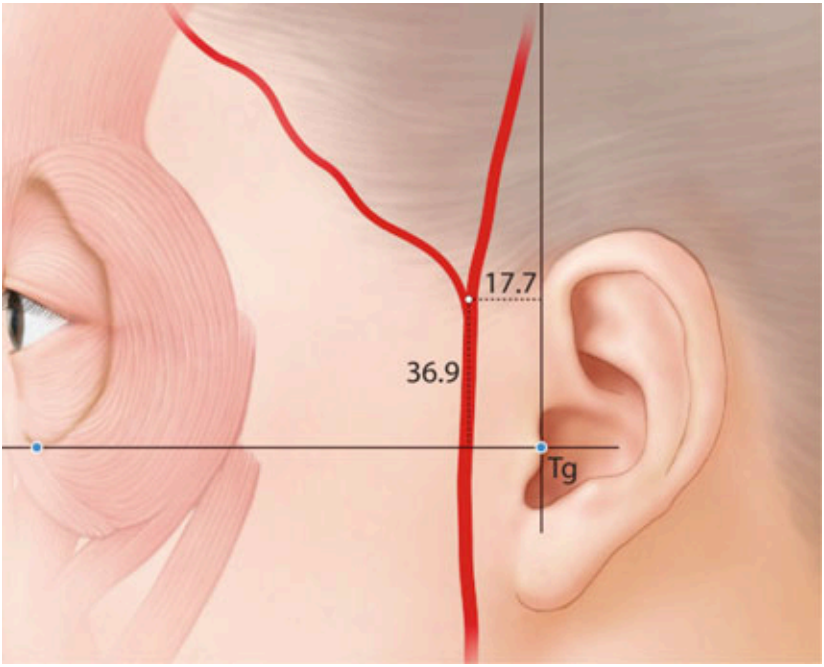
The facial v. parallels the facial a. in most instances. However, it runs more posteriorly than the facial a. and is less tortuous in the opposite direction of the facial a. The facial v. branches as follows.

Angular Vein

The angular v. is formed through the merging of the supraorbital v. and the supratrochlear v. at the canthus. The angular v. branches into two different branches with one flowing into the orbit and continuing to the superior ophthalmic v. and the other proceeding superficially and running inferiorly along the face as a facial v.

Intercanthal Vein

The intercanthal v. has been observed at the glabella and the radix in 71% of the cases and is located along the midpupillary line on the subcutaneous layer. 63.4 % of the cases showed that the intercanthal v. was observed along the line connecting the bilateral canthus, and the other 7.3 % of the cases showed that the vein was observed inferior to the same line. All the observed intercanthal veins run through the more superficial subcutaneous layer rather than the procerus m.



Frontal and parietal branches of the superfi cial temporal artery

Facial Vein

The facial v. obliquely runs posteroinferiorly toward the mandibular angle, receiving many tributaries.

External Nasal Vein

The external nasal v. originates from the lateral side of the nose and connects to branches of the infraorbital v.

Deep Facial Vein

The deep facial v. connects to the pterygoid plexus in the deep layer of the face.

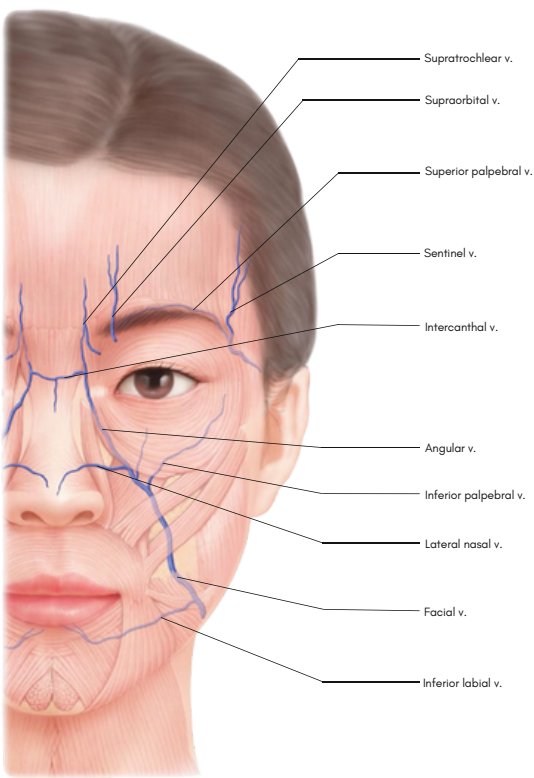
Labial Vein

The labial v. originates from the upper lip and the lower lip. The superior labial v. connects to the infraorbital v.

The inferior labial v. connects to the mental v. The facial v. continues inferiorly along the antegonial notch toward the neck. The facial v., unlike the facial a., runs through the superficial portion of the mandible.

Retromandibular Vein

The superficial temporal v. runs inferiorly, merging with the branch from the parotid gland and exits the lower margin of the parotid gland. The retromandibular v. is bifurcated into the anterior and posterior branch at the mandibular angle. The posterior branch merges with the posterior auricular v. from the posterior portion of the ear and forms the external jugular v. The anterior branch of the retromandibular v. merges with the facial v. at the neck and forms the common facial v. The common facial v. continues into the internal jugular v.



General course of the facial vein and its topographic relationships with the facial muscles

Superficial Temporal Vein

The superficial temporal v. receives the vein branch from the lateral side of the head. It proceeds inferiorly along the anterior side of the ear and enters the parotid gland. The superficial temporal v. merges with the maxillary v. from the inferior portion of the temporal region inside the parotid gland.

Connections of the Vein

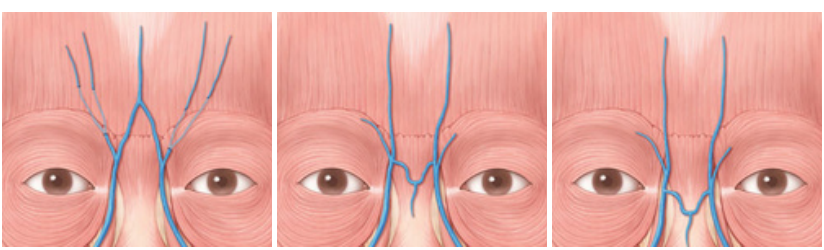
The facial v. lacks valves and is connected to relatively fewer branches. These two following vein connections are extremely important.

Connection Between Facial Vein and Angular Vein

The facial v. passes the angular v. and connects directly to the superior ophthalmic v. The venous blood from the medial canthus flows through the facial v. inferiorly to the neck or through the superior ophthalmic v. to the orbit. The superior ophthalmic v. proceeds into the cavernous sinus with a slow blood flow rate.

Connection Between the Pterygoid Plexus and the Cavernous Sinus

The venous blood from the facial v. flows through the deep facial v. again and into the pterygoid plexus inferior to the temporal region. The pterygoid plexus connects to the cavernous sinus inside the skull.



Venous anastomoses (intercanthal vein) at the glabellar region (c) type I at the glabellar region, (d) type IIa at the level above the intercanthal line, (e) type IIb at the level below the intercanthal line)

FACIAL NERVES

NERVES

Cranial nerve (CN) VII—the facial nerve—is the main motor innervation of the facial muscles, with damage to CN VII being one of the most dreaded (but rare) complications of surgery. After exiting the stylomastoid foramen, an upper and lower division develops as it passes through the parotid gland before travelling to the facial muscles. This nerve harbors significant clinical implications during facial surgery. Another significant clinical consideration during a mandibular block (CN VII), is potential hemifacial paralysis, otherwise known as Bell's palsy.

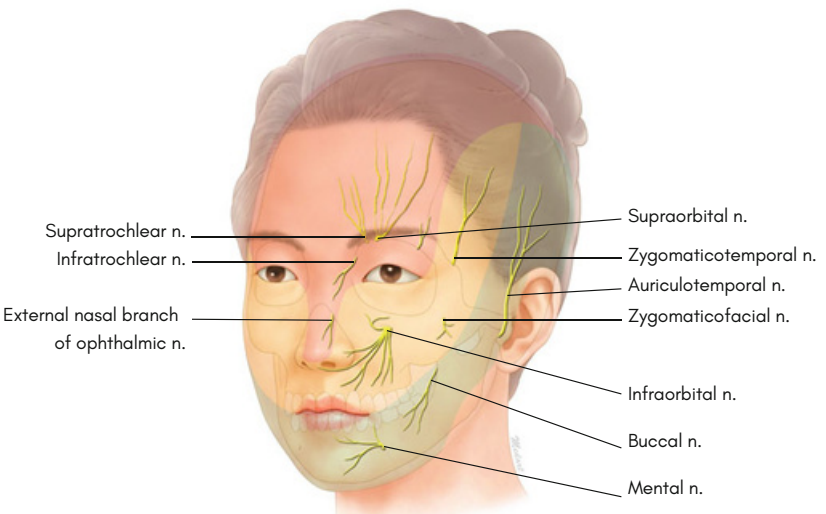
Other important innervations include CN V (trigeminal nerve), which has three branches as well as additional branches from the cervical plexus.

The greater auricular nerve is found approximately 5 cm inferior to the external auditory meatus, running deep within the superficial cervical fascia. The mental nerve, a branch of the inferior alveolar nerve, exits the mental foramen where it can be seen and palpated when the oral mucosa is stretched. This nerve provides innervation to the lower lip and the mandible. The buccal mucosa and the skin on the cheek is innervated by the buccal branch of the mandibular nerve, while the anterior two-thirds of the tongue is innervated by the lingual nerve (a branch of the mandibular division of the trigeminal nerve).

Face transplants have rapidly blossomed into a feasible management for patients with extreme disfigurements. To help repair damaged facial expression muscles and preserve their function, it is vital also to understand that these muscles do not contain proprioceptive receptors, compared with mastication muscles (which are innervated by the trigeminal nerve and thus contain proprioceptors).

Nerves of the Face and Their Distributions

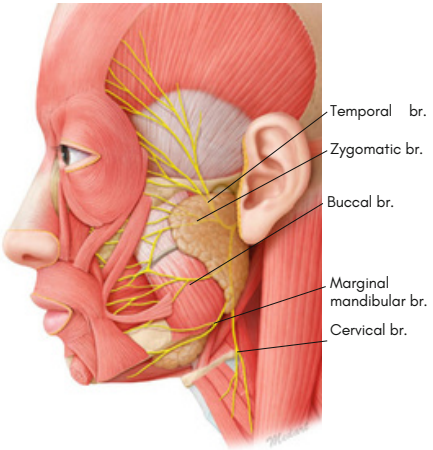
The trigeminal n. and the facial n. are major nerves distributed on the face. The trigeminal n. consists of three parts: the ophthalmic n., the maxillary n., and the mandibular n. The trigeminal n. passes through the foramina of the skull and divides into independent facial sensory nerves. On the other hand, the facial n. has one nerve trunk that passes through the stylomastoid foramen and separates into two divisions (temporofacial and cervicofacial divisions) within the parotid gland. Later, it branches off into five different nerve bundles transmitting motor impulses to facial mm.

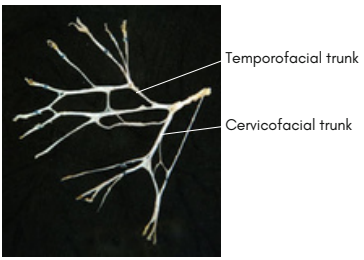


The cutaneous sensory distribution of the face (red zone area of the ophthalmic nerve (V1) branches, yellow zone area of the maxillary nerve (V2) branches green zone area of the mandibular nerve (V3) branches)

Distribution of the Sensory Nerve

- Supraorbital n., supratrochlear n. (ophthalmic n.): forehead, glabellar region
- Infratrochlear n. (ophthalmic n.): glabella, radix
- Infraorbital n. (maxillary n.): external nose, nasal septum, lower eyelid, upper lip
- Buccal n. (mandibular n.): cheek, cheilion
- Mental n. (mandibular n.): lower lip, mentum, cheilion





Trunk of the facial nerve (a , b, c)and its temporofacial (upper) and cervicofacial (lower) divisions

Distribution of the Motor Nerve

The facial n. consists of temporal, zygomatic, buccal, marginal mandibular, and cervical nerve branches that transmit motor impulse to facial and neck muscles. There are several small nerve branches with complicated, random distribution patterns to the muscles. Therefore, it is difficult to determine nerve distribution region of boundaries for each muscle

Upper Face

Distribution of the Sensory Nerve

The upper face includes the forehead, the glabella, the radix, and the upper and lower eyelids. The supraorbital n. distributes to the forehead, the glabella, and the upper eyelid with its long, distinct branch and runs to the forehead and the glabellar region.

Furthermore, the minor branches of the supraorbital n. distribute to the upper eyelid in a triangular pattern. The supratrochlear n. is distributed to the upper eyelid and the medial side of the glabella. The inferior palpebral branch of the infraorbital n. moves superiorly past the infraorbital foramen and is distributed to the lower eyelid in a triangular pattern.

Also, several minor branches of the zygomaticofacial n. become distributed to the inferior and medial side of the lower eyelid.

Distribution of the Motor Nerve

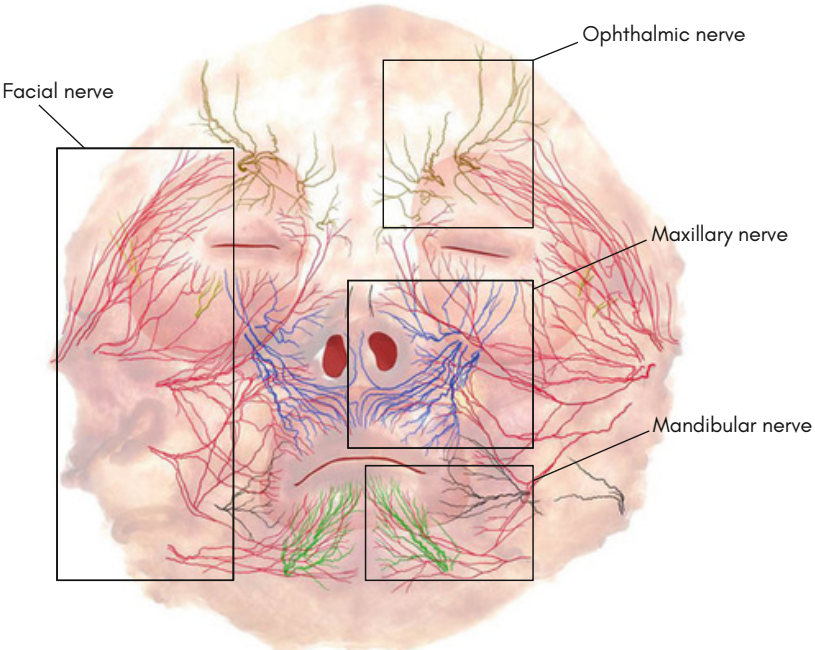
The temporal branch of the facial n. moves superomedially toward the upper eyelid and is distributed to the muscles on the lateral side of the upper eyelid. The zygomatic branch of the facial n. distributes the orbit and the muscles on the lateral side of the lower eyelid as it runs superior to the inferior palpebral branch of the infraorbital n. Generally, the temporal branch transmits motor ability to the frontalis m., the corrugator supercilii m., and the superior portion of the orbicularis oculi. The zygomatic branch is distributed to the inferior portion of the orbicularis oculi m. and to the origins of the zygomaticus major and minor m.

Typically, the buccal branch of the facial n. runs superiorly along the lateral side of the nose to the radix. Therefore, the procerus m., the medial portion of the corrugator supercilii m. on the glabella, and the radix are innervated by the temporal branch and by the buccal branch

Midface

Distribution of the Sensory Nerve

The midface includes the cheek region and the nose. The infraorbital n. of the trigeminal n. plays a vital role in the cutaneous sensation in the midface. The external nose is mostly innervated by the infraorbital n. with the exception of some parts that are innervated by the external nasal branch of the nasociliary n. (from ophthalmic n.). The lateral nasal branch of the infraorbital n. proceeds along the nasal ala with some distributing to the nose tip near the midline. The internal nasal branch of the infraorbital n. is distributed to the mucosal of the nasal septum. The superior labial branch of the infraorbital n., one of the most distinct branches, is distributed to the area that spans from the medial portion of the upper lip to the cheilion. The infraorbital n. is distributed among the general infraorbital region from the infraorbital foramen to the upper lip.



Sensory and motor nerve distribution on the face (V1, ophthalmic nerve; V2, maxillary nerve; V3, mandibular nerve; VII, facial nerve)

Distribution of the Motor Nerve, 1

The temporal branch of the facial n. moves superomedially toward the upper eyelid and is distributed to the muscles on the lateral side of the upper eyelid. The zygomatic branch of the facial n. distributes the orbit and the muscles on the lateral side of the lower eyelid as it runs superior to the inferior palpebral branch of the infraorbital n. Generally, the temporal branch transmits motor ability to the frontalis m., the corrugator supercilii m., and the superior portion of the orbicularis ocuper lip.

Distribution of the Motor Nerve, 2

The buccal branch of the facial n. proceeds medially and has small branches that are dispersed to the cheek. These branches superimpose with the superior labial branch of the infraorbital n.

The buccal branch and the infraorbital n. lie superimposed with each other in the superior 3/4 of the infraorbital region. The buccal branch is distributed to the levator labii superioris alaeque nasi, the levator labii superioris, and the zygomaticus minor m. The buccal branch also is distributed to the zygomaticus major, the risorius, and the superior portion of the orbicularis oris m.

Lower Face

Distribution of the Sensory Nerve

In the lower face, the mandibular n. distributes to the lower lip and to the mentum. The buccal n. proceeds medially along the occlusal plane to the cheilion. The mental n. runs through the mental foramen and is distributed to the lower lip which includes the cheilion and the mandible. The superior labial branch of the infraorbital n., the buccal n., and the angular branch of the mental n. is distributed to the mouth corner. Furthermore, there are nerve plexus formed between the infraorbital n. and the buccal n. and also between the buccal n. and the mental n. superior and inferior to the cheilion.

Distribution of the Motor Nerve

The marginal mandibular branch of the facial n. is distributed to the mentalis, the depressor anguli oris, the depressor labii inferioris, and the inferior portion of the orbicularis oris m. The actual anatomy of the trigeminal n. and the facial n. is quite different from that found in the textbook. The cutaneous n. of the trigeminal n. and the motor n. of the facial n. are not distinguished as some of the few, distinct nerves. Even though some of the major branches can be observed during dissection surgeries with the naked eye, they are intertwined with other small branches such as nets. Therefore, it is best to describe the distribution pattern of nerves with a plane rather than with several distinct lines

FACIAL NERVE BLOCK

NERVE BLOCK

Supraorbital Nerve Block (SON Block)

The supraorbital n. originates from the supraorbital notch, which can be identified on the supraorbital rim. If the supraorbital notch cannot be found externally, it can be replaced by the supraorbital foramen. The supraorbital notch is located medial to the midpupillary line on the frontal bone. Insert the syringe immediately inferior to the eyebrow and inject anesthetics proximal to the supraorbital notch. It is necessary to take caution to avoid injecting the anesthetics into the orbit. If the lateral branch has not been anesthetized with a general infraorbital nerve block, it is suggested to perform additional anesthesia by inserting the syringe 1 cm superior to the orbit toward the medial portion of the eyebrow.

Supratrochlear Nerve Block (STN Block)

In 30 % of cases, the supratrochlear n. arises together with the supraorbital n. from the supraorbital notch and can perform nerve blocks along with SON blocks. However, in the majority of cases (70 %), the supratrochlear n. originates separately from the frontal notch, which requires an injection 15 mm lateral from the facial midline, which can be approximated by placing the index finger on the midline of the forehead. In this case, an additional injection is required

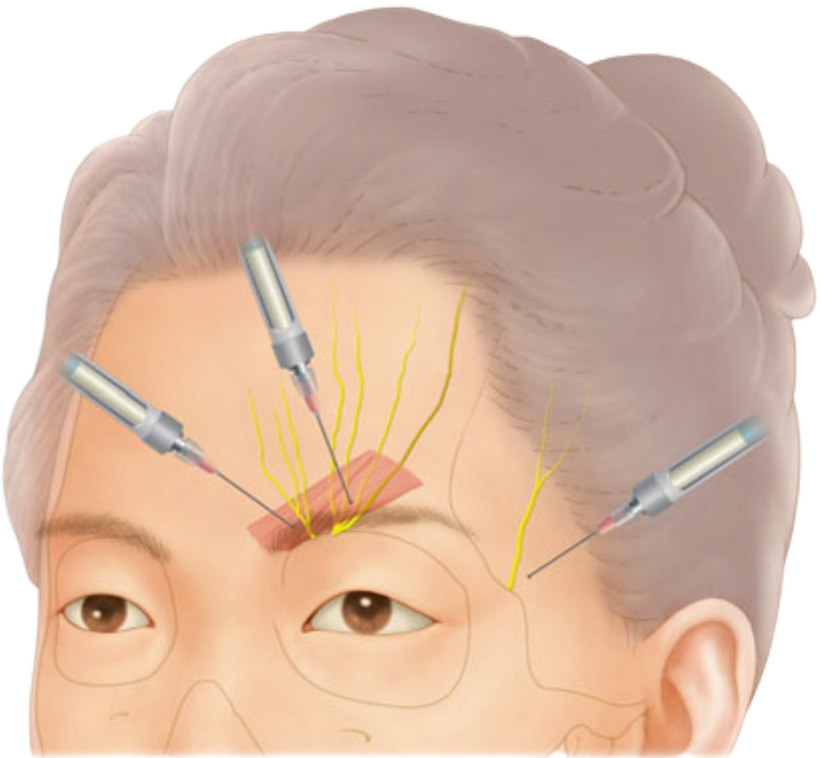
Infraorbital Nerve Block (ION Block)

The infraorbital nerve block is an extremely useful technique to use in aesthetic surgery procedures as both intraoral and extraoral approaches could perform effectively. Both approaches target the infraorbital foramen, which the infraorbital n. passes. The infraorbital foramen is located on the upper third where the line between the nasal ala superior to the vertical line passing the cheilion and the point at the same height as the infraorbital margin is divided into three sections.

In the extraoral approach, inject anesthetics targeting the location of the infraorbital foramen as described above. However, the transcutaneous, nasolabial approach of approaching from the marionette line rather than by vertical insertion also exists.

This approach injects at the site where the superior portion of the marionette line and the alar groove meet to form the upside-down V-shape and then runs superolaterally. The transcutaneous nasolabial method allows for a more intricate approach to the infraorbital foramen.

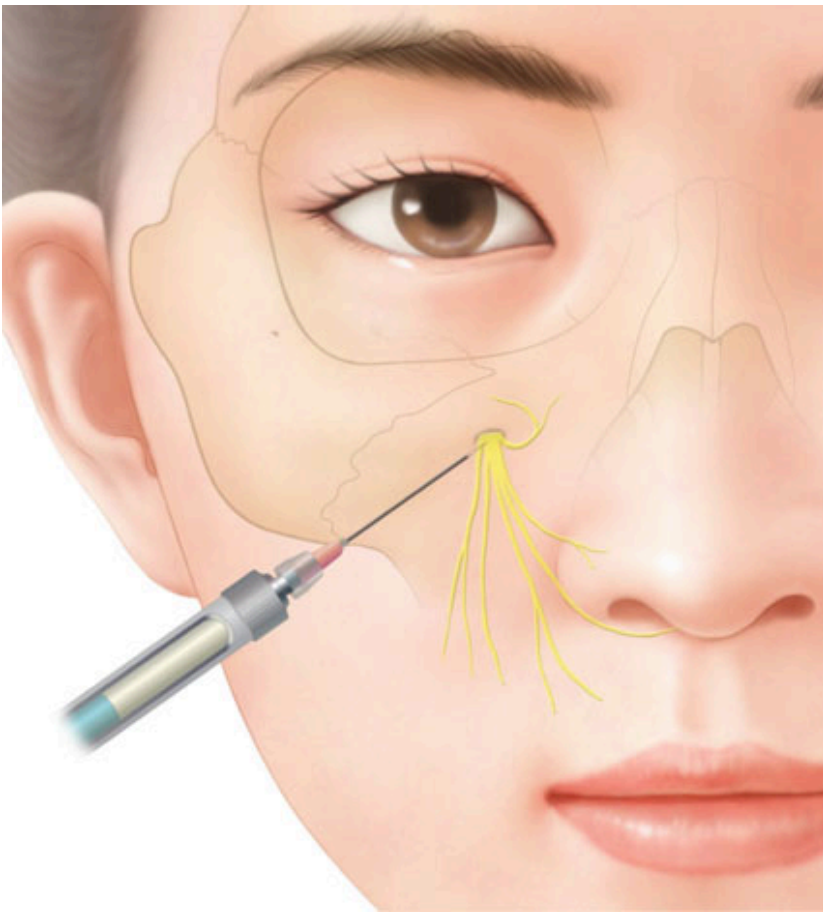
In the intraoral approach, place the syringe parallel to the longer axis of the maxillary second premolar and inject the needle slowly and superiorly. Inject anestetics when the target is located. Both approaches require caution to avoid injecting the anesthetic inside of the orbit. In such cases, diplopia may occur.



Supraorbital and supratrochlear nerve block

Zygomaticotemporal Nerve Block (ZTN Block)

The meeting point of the frontal bone and the zygomatic bone is presented as an eminence point lateral to the eyebrow. The zygomatico temporal n. originates laterally to this region and innervates the lateral portion of the eyebrow and the glabellar region. However, facial landmarks are unclear. Therefore, a nerve block does not always perform well.



Extraoral (a)and intraoral (b) approaches for the infraorbital nerve block

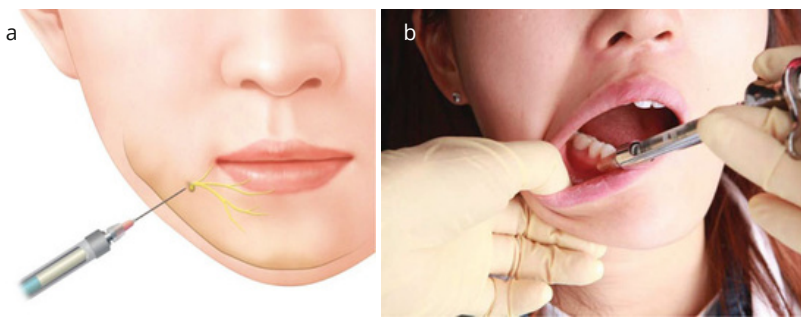
Mental Nerve Block (MN Block)

Similar to the infraorbital nerve block, a mental nerve block can also be completed via the extraoral or the intraoral approach. Both approaches target the mental foramen 2cm vertically inferior from the cheilion. For the extraoral approach, inject the syringe posterior to and superomedially while targeting the mental foramen.

In the intraoral approach, inject slowly, inferiorly, and posteriorly at the mandibular second premolar region.

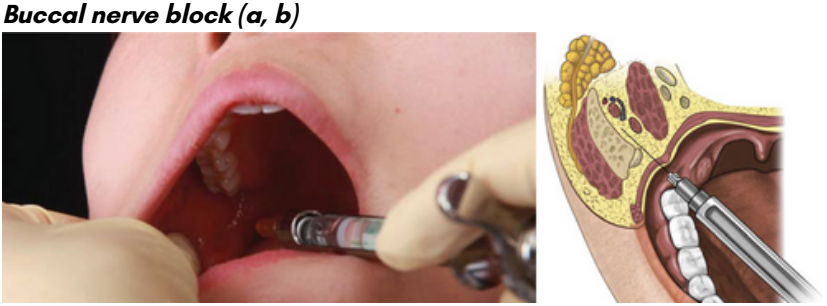
Buccal Nerve Block (BN Block)

The buccal nerve enters the oral mucosa near the maxillary second molar, its main trunk running medially. As it proceeds medially through the dentition, the main trunk of the buccal n. lies in a slightly inferior position. The main trunk of the buccal n. supplies the entire buccal area including the mucosa and skin of the lateral area of the mouth corner. The main trunk gives off some branches not only near the main trunk running inferomedially, but also in the other regions. The buccal nerve block should be performed with a needle approaching the buccal aspect of the mandibular second molar. After placing a needle parallel to the occlusal plane, inject the anesthetic slowly along the buccal aspect of the mandibular second molar or oblique line of the mandible



Extraoral (a)and intraoral (b)approaches for mental nerve block

Buccal nerve block (a, b)



Inferior alveolar nerve block (a, b)

Inferior Alveolar Nerve Block (IAN Block)

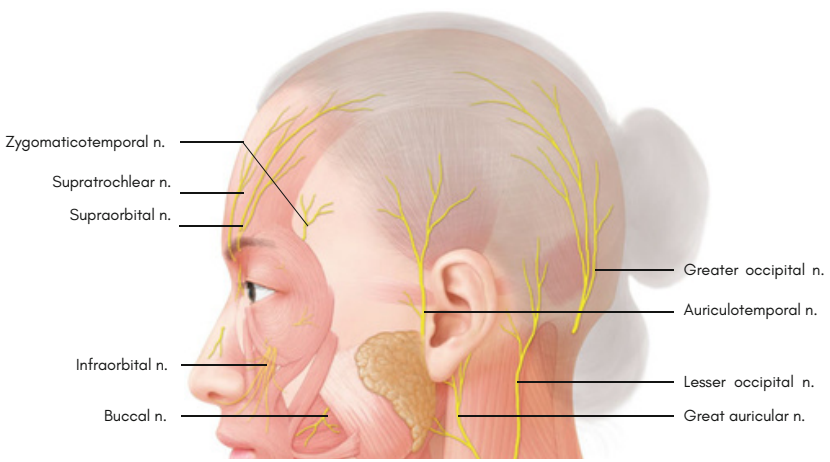
In order to completely anesthetize the skin on the lower chin, it is necessary to intraorally approach the inferior alveolar n. Slowly inject a long needle 1 cm superior to the occlusal plane of the fi rst premolar on the opposite side of the target toward the central point on the retromolar triangle. If the needle comes into contact with the ramus of the mandible, pull it back slightly and inject anesthetics

Auriculotemporal Nerve Block (ATN Block)

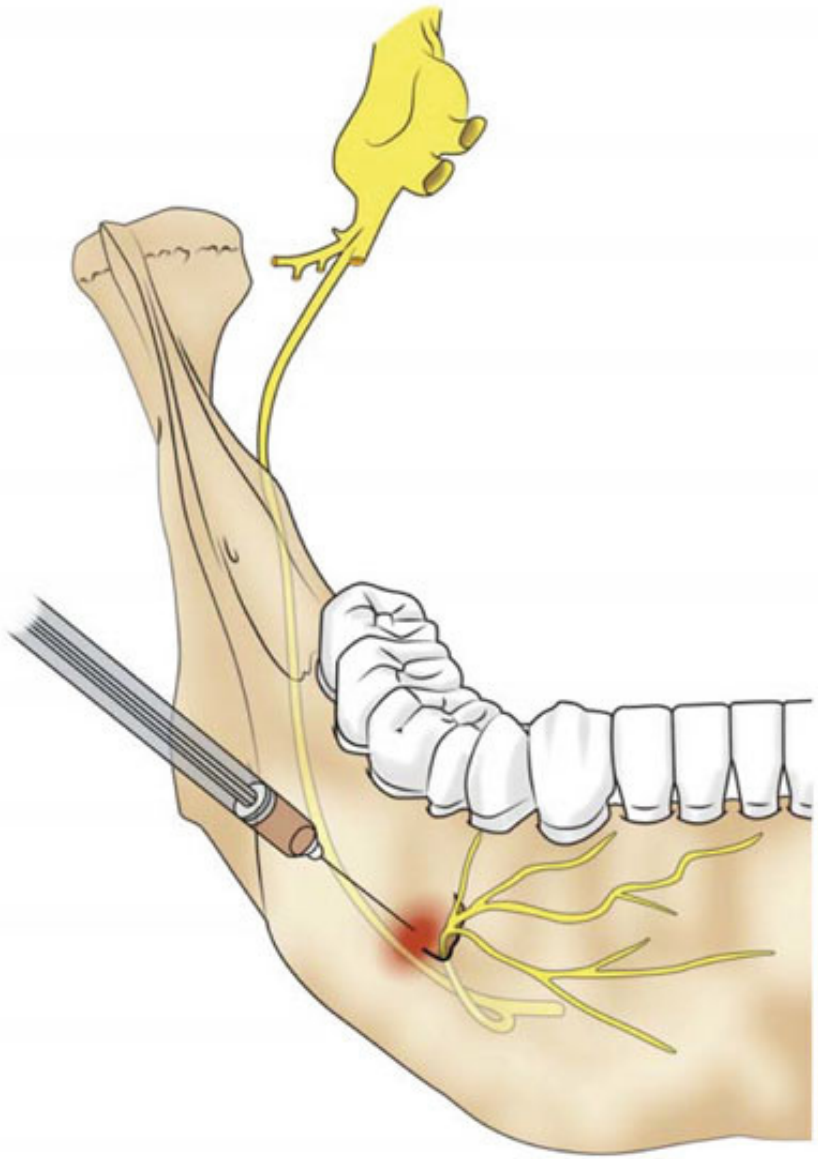
For an auriculotemporal nerve block, inject 2 cc of anesthetics anterior to the tragus. If the auriculotemporal n. is blocked, the sensation of the tragus, the anterior auricle, and the external auditory meatus also become blocked. Anesthetizing other parts of the auricle requires a great auricular nerve block.

Great Auricular Nerve Block (GAN Block)

The great auricular n. proceeds superiorly along the anterior surface of the sternocleidomastoid m. Place a hand on the patient's temple in order to distinguish the sternocleidomastoid m. and mark its boundary. Then inject the anesthetic 6.5 cm along the line from the external acoustic pore to the midpoint between the boundaries of the sternocleidomastoid m.



Topographic anatomy of peripheral sensory nerve branches of the head and neck



An illustration showing the anesthetized area for Extraoral (a)and intraoral (b)approaches for mental nerve block

SKULL AND FACE BONES

BONES

Youthful features have been said to be optimally present at a point in time when a specific set of skeletal proportions are ideal for their soft-tissue envelope. The skeletal framework forms the basis on which unique facial characteristics are built, rendering underlying bone vital in providing and preserving ideal soft tissue relationships.

Important facial bony constituents include the frontal, maxillary, zygomatic, palatine, nasal, temporal, lacrimal, ethmoidal and mandibular bones. Bone provides structural support and attachment sites for the muscles of facial expression and mastication, and also protects certain structures such as the eyes.

The facial skeleton undergoes both expansion and selective resorption throughout life, with the pyriform and orbital apertures being particularly susceptible to age-related resorption. Maxillary recession and a 10° decrease in the maxillary angle have been noted after 60 years of age. Midface skeletal involution also occurs from the sixth decade, occurring more frequently in women than men. Skeletal regression of particularly the inferolateral orbital rim and alveolar ridges, contributes to loss of midfacial support and also loss of overall facial height.

Age-related changes within the nasal aperture, paired nasal bones, and ascending processes of maxillae may lead to prominent changes, including nasal lengthening, sagging of the tip, and posterior displacement of the columella and lateral crura.

Selective resorption of the upper jaw may lead to a subsequent loss of dentition, with Bartlett et al. demonstrating that decreasing height of the maxilla and mandible correlate strongly with eventual loss of dentition.

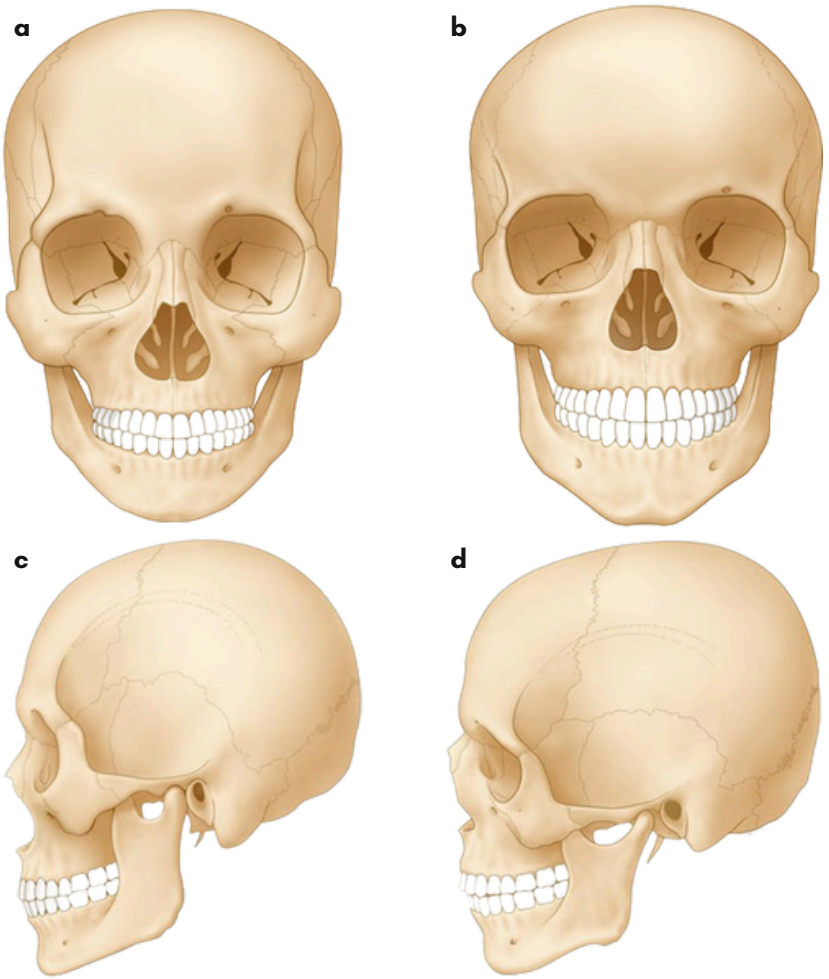
Loss of teeth generally affects the mandible more than the maxilla, with women at a higher risk of this loss.

Individuals with prominent bony features, including a supra-orbital bar, strong cheekbones, and prominent jawlines have been said to age more favorably.

CHARACTERISTICS OF SKULL AND FACE

Caucasian heads are dolichocephalic and mesocephalic shaped. However, Asians, including Koreans, show brachycephalic head shapes.

Brachycephalic shaped heads indicate that the head width is relatively large in comparison to its length. Similarly, Asians possess relatively fl at faces. They are particularly characterized by a protruding zygomatic bone in the anterolateral direction and a pronounced mandibular angle.



Anthropological difference of the skull between Asian (Korean) and Caucasian ((a, c)Korean skull, (b, d)Caucasian skull)

SURFACE LANDMARKS

FACIAL AND SKULL SURFACE LANDMARKS

1.Facial Surface Landmarks According to Anatomical Labels

2. Surface Landmarks of the Skull

It is best to be acquainted with foramens of the skull, which serve as landmarks from which major vessels and nerves exit.

(a) The frontal notch and the supraorbital foramen: The frontal notch medial to the eye could feel near the glabella along the supraorbital margin, and the supraorbital foramen could feel slightly lateral to the frontal notch. The supratrochlear a. and supratrochlear n. pass through the frontal notch, and the supraorbital a. and supraorbital n. pass through the supraorbital foramen.

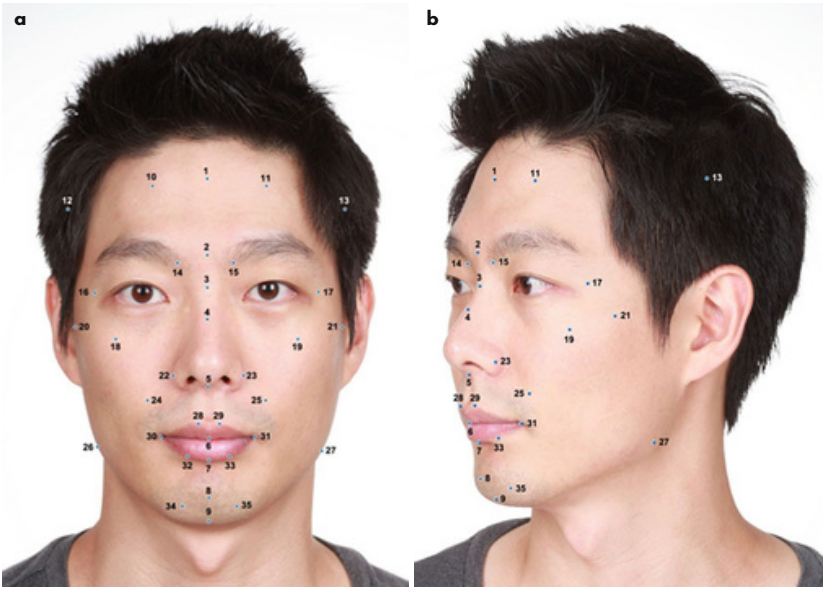
(b) Infraorbital foramen: The infraorbital foramen is located on the upper third of the line connecting the infraorbital margin to the nasal ala. The infraorbital foramen locates medial to the vertical line connecting the pupil and mental foramen. The infraorbital a. and infraorbital n. exit through the infraorbital foramen

(c) Mental foramen: The mental foramen is located along the same line used to locate the infraorbital foramen 2 cm inferior to the oral commissure. The mental n. exits through the mental foramen

3. Surface Anatomy

4. Actions of the Facial Muscles and Formation of Creases

There are various muscles in the face that participate in the formation of facial expressions and facial creases. The following three points should be considered.



Surface landmarks of the face ((a)frontal view, (b)oblique view)

1. First, there are origin and insertion points for the facial m., and the muscle contracts toward the origin in order to produce facial expressions and creases. Therefore, it is necessary to understand each muscle's movement vectors and their actions.

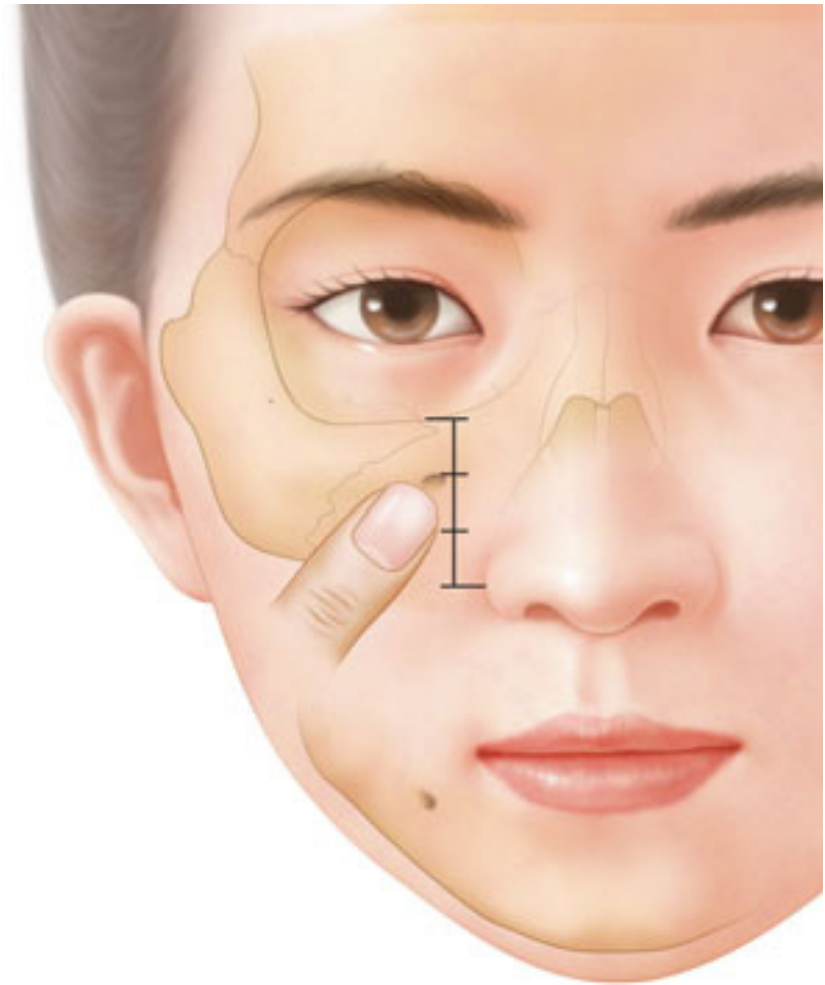
2. Second, all the wrinkles or furrows are created perpendicularly to the muscle vectors.

3. Third, there are many instances when multiple muscles overlap. Furthermore, multiple muscles are involved in producing one's facial expressions. Therefore, each muscle's movements cannot be correlated to one facial expression.

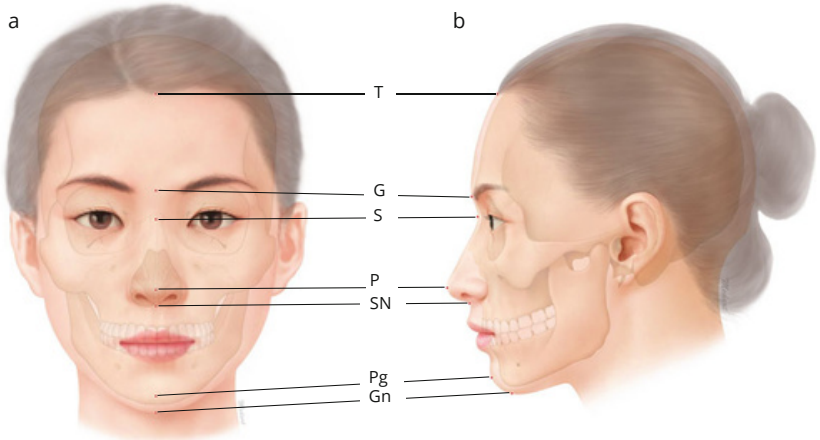
4. Fourth, not all muscles lie on the same plane. There are about 3-4 layers of depth with different muscles in each layer, which affect injection depth.

Muscle size and location vary among individuals. For example, some possess a thicker corrugator supercilii m., while others possess a wider depressor anguli oris m.

It is necessary to observe a patient's various facial expressions in order to investigate the strength and area of each muscle.



Identification of the supraorbital notch (or foramen), infraorbital foramen, and mental foramen



Major anatomical landmarks of the face (a) frontal view, (b) lateral view) (T trichion (hairline), G glabella (most anterior projection of the forehead), S sellion (the deepest point of the nasofrontal concavity), P prona-sale (apex nasi, nasal tip), SN subnasale (the point at which the nasal septum merges), Pg pogonion (the most prominent point of the soft tissue of chin), Gn gnathion (the lowest part of the soft tissue of the chin)

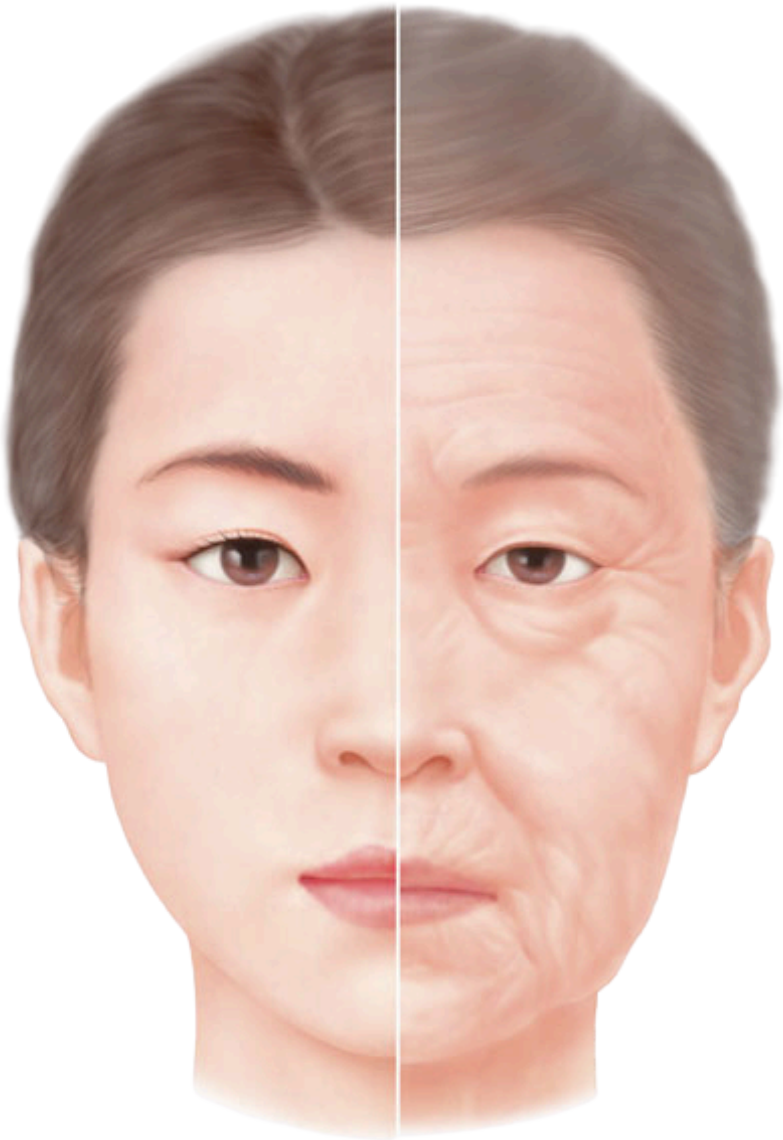
CONCLUSION

The face is unique in its profound ability to communicate, express emotion and masticate. As a result of this intricate functionality, it is imperative that medical practitioners have an insightful understanding of applicable anatomy. Each facial layer is morphologically and clinically distinct and may be differentially affected by the aging process. This layered structure provides an intricate canvas, adding to the functional and artistic imagery required during aesthetic treatments.

By first breaking the anatomy down into basic layers, it is easier to visualize the integral structural an functional components before attempting to brainstorm novel aesthetic solutions.

THE AGING PROCESS

ANATOMY OF THE AGING PROCESS
AGING OF SKIN, SOFT TISSUE, AND BONE



Changes of the facial aging

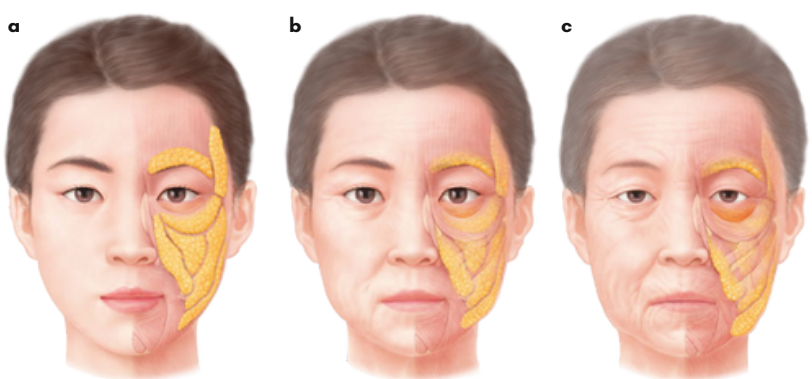
THE COMPLEX CHANGES OF THE FACIAL APPEARANCE WITH AGING

In life, the aging process comes naturally. Research on the aging process increased as people's average life span became longer, and people started to pay greater attention to their quality of life.

Understanding the characteristics of aging is the key foundation for filler and botulinum toxin treatment

All tissues change characteristics with age. As apparent in the illustration tssues are subject to atrophoderma, redistribution, and sagging. The skin loses some collagen and elastin in the dermis, and the dermis loses some hyaluronic acid, becoming dry, inelastic, and wrinkly.

Facial aging is a complex, multifactorial process involving multiple facial layers. Changes in the skin, skull, and soft tissues play contributory roles. Loss of collagen and elastin, combined with epidermal thinning, contributes to the appearance fine rhytides. Distributional changes in the superficial and deep fat pads, in addition to bone remodeling, constitute key morphological factors and result in the characteristic inverted heart shape of the aging face. Understanding these multifactorial aging pathways facilitates effective aesthetic treatments.



Processes of the facial aging in 30's (a), 50's (b), and 70's (c)

AGING PROCESS OF THE FACIAL TISSUE

The anatomical structures of the face related to aging comprise of the facial bone, fat tissue, fibrous connective tissue, and facial muscles.

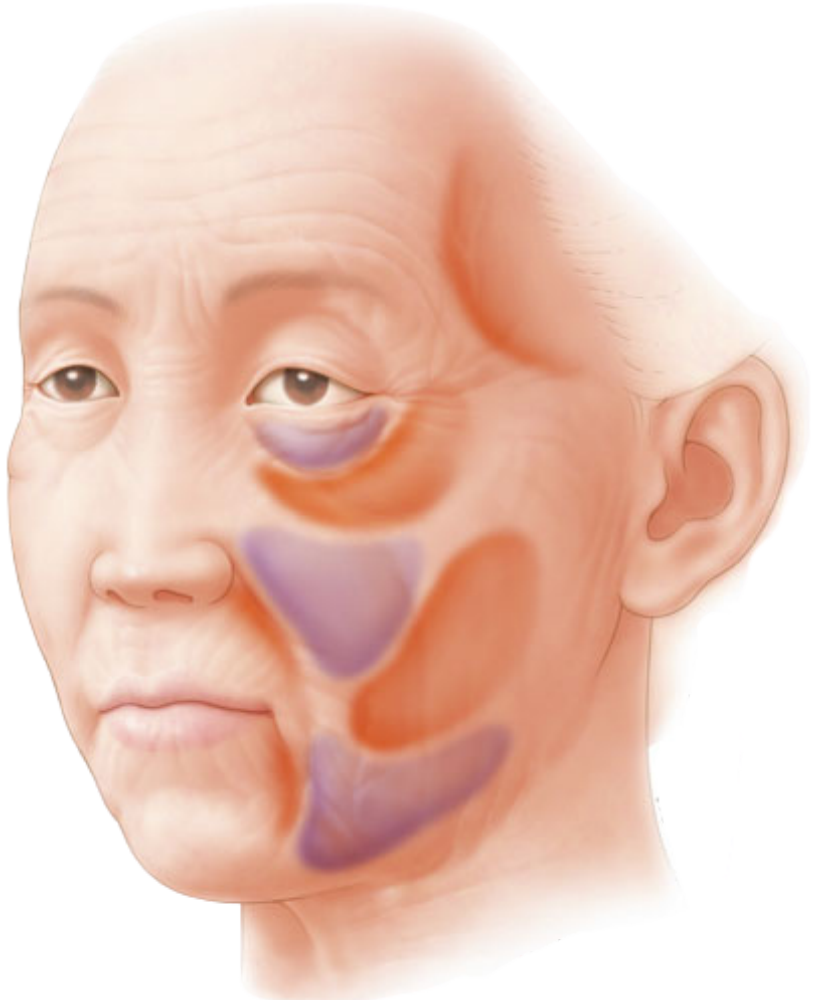
The bony tissue is a structure that forms the basic frame of the face and bone remodeling goes throughout lifelong period. With aging, bone resorption is accelerated and morphologic changes take place in the marginal area of bones, such as the orbital rim, maxilla, and the mandible. Thus, the orbital rim enlarges, the maxilla shortens, and the length and height of the
mandible reduce

Fat tissue shows different aging processes between superficial and deep fat of the face. In the superficial fat, drooping appears due to gravity. In the deep fat, relocation and atrophy take place due to the unbalanced change of the volume of fat compartments. Drooping of the fat tissues is presented as jowl or the deepening of the nasolabial fold caused by the drooping of the superficial fat of the chin and cheek. The relocation and atrophy of fat appear as hollow cheek.

Among the fibrous connective tissue, the thick fibrous connective tissue that has high density and strongly holds facial muscle is called the retaining ligaments. The facial musculature is a thin layer that lies between the superficial fat and deep fat of the face. Fibrous connective tissue loses its elasticity when proteins such as collagen and elastin are degraded. Subsequently, dermal thickness reduces and membranous structures such as septum and SMAS shift downward.

However, retaining ligaments have two sides of aging. Retaining ligaments make the wrinkle look deeper around the boundary of the fat compartments. This is because the retaining ligaments have a function of resisting against drooping of other tissues. On the other hand, when aging proceeds and even the retaining ligament loses its elasticity, fat protrusion and drooping caused by gravity accelerate even more. An example of the former case is tear trough caused by orbicularis retaining ligament and of the latter case is palpebromalar groove and festoon caused by septal fat protrusion.

The aging of facial muscle becomes permanent through reduced elasticity of the muscle itself and through repetitive movements over a long time. A typical example of reduced muscular elasticity is the gobbler neck deformity caused by the laxity of platysma. Examples of muscle fixation or adhesion with aging are deep skin wrinkles such as horizontal forehead lines, crow's feet, and horizontal upper lip line.



The aging changes of the soft tissue of the face. Brown-colored areas indicate the remarkable volume loss with age. Purple-colored areas show the protrusion and drooping of the fat tissues

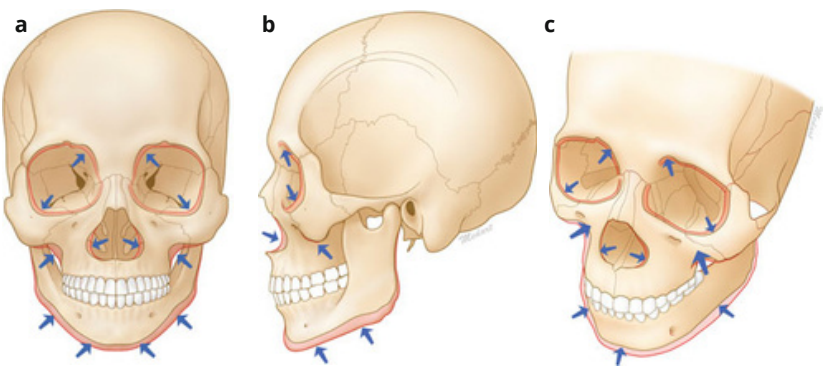
The main function of the facial skeleton is to protect the brain and important sensory organs of smell, sight, and taste, and to provide a foundation for the face. The skull is subdivided into two main parts: the cranial vault, which protects the brain and houses the middle and inner ear structures, and the facial bones, which form the support for the soft tissues of the face, the nasal cavity, the eyeballs, and the upper and lower teeth.

The adult skull comprises 22 separate bones, of which only one, the mandible, is mobile and not fused as a single unit. In order to understand the aging process of the skeletal base, it is of great importance to know the relationships between the different bones, transitions, and landmarks.

The face may be divided into thirds (upper face, mid-face, and lower face) in order to identifying important bony and soft tissue landmarks

The upper face consists of mainly the frontal bone, which forms the upper third of the anterior adult skull giving the forehead an aesthetically pleasing curvature. The frontal bone can be divided into three parts:

1. Squamous part of the frontal bone
2. Glabella and nasion
3. Supraorbital ridge



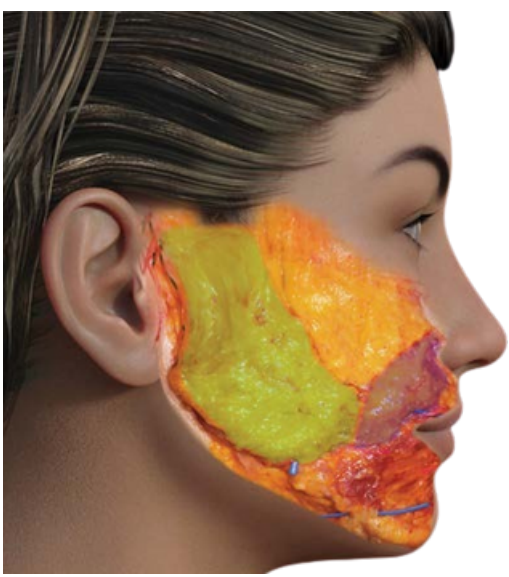
Bone resorption pattern of the face with aging ((a) frontal view, (b) lateral view, (c) oblique view)

There is no clear understanding as to which aging changes occur in the cranium and the upper face.

Soft tissue changes in the aging upper face are also of note. A well-accepted theory is that of volume loss due to lipo and muscle atrophy

Aging of the periorbital area progresses with a complex aging procedure of the bony orbit, orbicularis retaining ligament, superfi cial and deep fat, and orbicularis oculi muscle. When young, tear trough appears due to the orbicularis retaining ligament located in the medial aspect of the orbit. With age, the septal fat above the orbicularis retaining ligament goes through protrusion. Then the tear trough deepens, and margins are connected and form a nasojugal groove. The repetitive movement of the orbicularis oculi muscle at this state forms crow's feet on the lateral orbital rim. When aging further progresses, the orbital rim is absorbed and the bearing capacity, elasticity of ORL, is reduced. Consequently, festoon appears due to septal fat protrusion.

Aging of the perioral area progresses with a complex aging procedure of the maxilla and mandible, mandibular ligament, superficial and deep fat, and perioral muscle.



Midfacial fat.

The midface is a merging of the following bony structures: nasal, lacrimal, ethmoid, maxillary, zygomatic, and palatine bone. The main function of the midface is to house the eyeballs within the orbit and the teeth within the maxilla, which then transmits masticatory forces to the skull base. The midface also provides a scaffold for the main facial tissues.

One of the key points in midface analysis is the bizygomatic distance, or most exterior bilateral point of the zygomatic arch, which is widest part of the face. The midface anthropometric measurement land-marks are the maxillary angle (the angle between the sella-nasion and the line between the superior and inferior maxilla) and the pyriform angle (nasal bone to lateral inferior pyriform aperture, divergent from the sella-nasion line)

Facial skeletal aging is most prominent in the mid-face, but the rate of bone resorption is not uniform; the maxilla is more prone to bone loss compared to the zygoma. Therefore, it is helpful to analyze the important midface features separately (orbit, maxilla, pyriform aperture, and zygomatic arch).

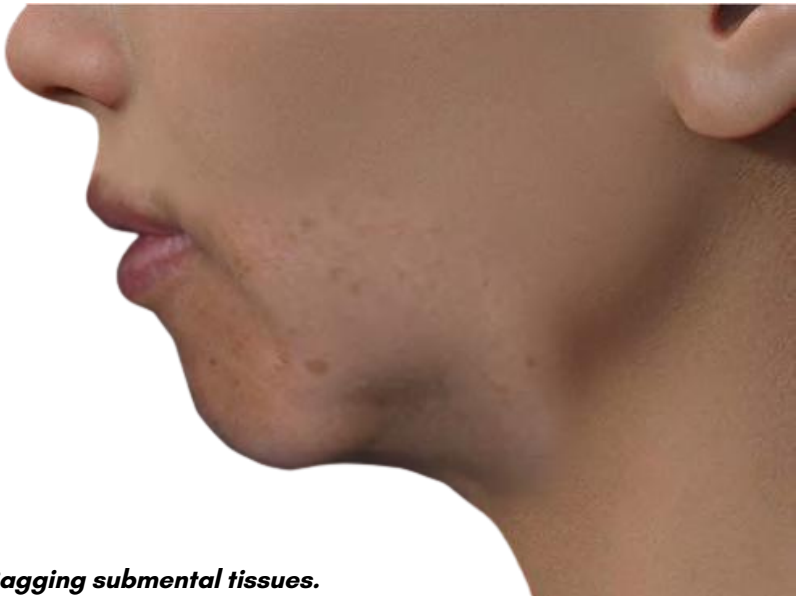
Maxillary recession is most evident in the anterior aspect. Studies found a decrease of the maxillary angle of 10° between the young (<30 years) and old populations (>60 years). This finding can be causative for the loss of support of the inferior orbital rim and loss of projection of the maxilla. The zygomatic arch suffers a posterior and anterior remodeling, which leads to increased temporal hollowing.

In the lower face, there is only one bone, the mandible, which carries the lower dentition. Important anatomical landmarks are the pogonion, the most anterior point of the chin, and the gonial angle, located at the posterior border at the junction of the lower border of the mandibular ramus.

There are numerous controversial theories regarding aging changes of the mandible. Bony changes lead to loss of soft tissue support and therefore changes in facial aesthetics. Decreased mandibular height and length, and an increase in the mandibular angle, contribute to a loss of definition of the jawline and development of jowls.

When young, nasolabial fold appears at the border of the upper lip and superficial malar fat compartments. As the body ages, the nasolabial fold deepens since the superficial malar fat above the nasolabial fold droops down and the cheek becomes hollow.

People with active movements of DAO may form labiomandibular fold on medial boundary of DAO. The labiomandibular fold could connect with the nasolabial fold.



Sagging submental tissues.

When aging progresses, the superficial buccal fat starts to droop and a pit appears in the mid-chin because the mandibular ligament holds the buccal fat.

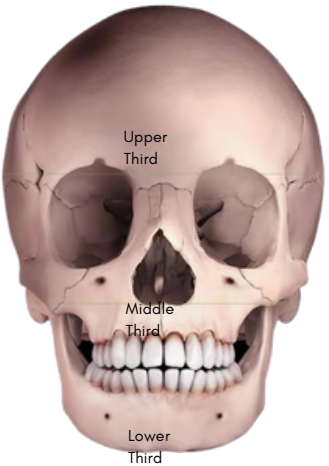
if the elasticity of the mandibular ligament reduces and mandible absorption accelerates the loss of support, jowling may intensify.

When old, not only the mandible and maxilla are absorbed, but the alveolar bone is absorbed intensely. Since the absorption of the maxilla is faster than the mandible, the chin may seem protruded. Since the tooth and alveolar bone are lost and not there to locate the perioral muscle, the perioral muscle and lip contract and fine wrinkles appear at the perioral area.

Loss of maxillary support and projection and the mandible will result in morphological changes, and soft tissue changes also contribute to the saggy appearance in older individuals.

With aging, there is deflation and loss of the normal anatomic subcutaneous facial fat compartments, which give the appearance of increased skin laxity or prominent folds around the nasolabial region, peri-orbital region, and jowl.

One can use the deep and superficial fat pads as a map for facial aging: the deep fat pad of the periorbital area is affected first (the transition between the medial suborbicularis oculi fat and the superior edge of the malar fat pad is lost), which creates a concavity between the thin medial eyelid skin and thicker cheek skin, resulting in a tear trough deformity.



Subsequent further deflation of the deep medial cheek fat leads to ptosis of the overlying superficial malar fat pad and further deepening of the tear trough deformity with hollowing of the centromedial cheek. However, there is not only volume loss but also hypertrophy.

Together with volume loss in fat pads, there is also lack of support and stability of the ligaments because of the repositioning of their points of origin, followed by ligamentous weakening due to continuous stretching.

Ligaments function as a hammock for the fat compartments and promote the appearance of sagging when there is a lack of structure.

BOTULINUM TOXINS

MOLECULAR STRUCTURE AND MODE OF ACTION

Clostridium botulinum is an anaerobic, gram-positive bacterium that secretes an extremely large neurotoxic molecule (900 kDa), which produces food poisoning or botulism. It is now also used in medicine to treat diseases according to Paracelsus's paradigm that the difference between a poison and a drug lies in the dose.

Although seven different serotypes of the bacterium (A to G) are known, type A is the one mostly used for the production of clinical formulations.

Type B also has clinical applications for those patients who may have developed clinical resistance to type A, but this is seldom the case in aesthetic treatments.

Of the 900 kDa natural molecule, only the central 150 kDa segment (the neurotoxic core) is responsible for its biological activity. The surrounding portions have no pharmacological activity and simply act as a protective shield ensuring unchanged toxin absorption from the host's gastrointestinal tract. These surrounding molecules are named accessory proteins and are both hemagglutinin and non-hemagglutinin in nature. Once the toxin has entered the host by ingestion or injection, the biological role of the accessory proteins is largely terminated and the 150 kDa neurotoxin comes into play. Here again, the structure is complex and each portion of the segment plays a relevant role.

The 150 kDa neurotoxic protein is divided into a 100 kDa heavy and a 50 kDa light chain. The heavy chain has a two-fold action. The first part (binding domain) links to the specific receptors at the level of the axonic presynaptic endings; the second (translocation domain) then carries the light chain through the membrane and into the actual nerve ending. Once there, the light 50 kDa chain accomplishes its task by cleaving a group of proteins, named SNARE, which are responsible for the release of the neurotransmitter acetylcholine. Inhibition of acetylcholine release impairs muscular contraction and flaccid paralysis ensues.

The effect of botulinum toxin is only temporary, and within a few months neuromuscular efficacy is spontaneously reestablished.

The details of this process have not been fully elucidated. For the purposes of this guide, however, it should be made clear that regeneration always and completely occurs. Interruption of neuromuscular transmission is not the only medical application of the drug. Indeed, receptors for botulinum toxin are widespread in the human body and reach far beyond only neuromuscular junctions. Accordingly, the indications for the treatment have widened tremendously since its introduction 30 years ago. However, it is beyond the scope of this chapter to examine the present status of botulinum toxin in medicine, and from here onward, only aesthetic applications will be considered.

THE MUSCLES OF FACIAL EXPRESSION

With few exceptions, these muscles, often defined as mimetic, are the target of our treatments. Their role is to animate the face, thus allowing communication of feelings and emotions to the outside world. Most mimetic muscles feature a bony origin and a cutaneous insertion. During muscle exertion, the skin is tractioned toward the skeletal origin, causing wrinkles, lines, and furrows to appear on the surface with repeated contraction. Other mimetic elements have both origin and insertion on the skin, but here again, contraction generates surface irregularities. Muscles seldom function independently and more often interplay synergistically and variably with other muscles. This determines the unlimited nuances of facial expressions. Mimetic muscles are divided into two functional groups: elevators and depressors.

How does botulinum toxin step into the field?

The answer is simple. In youth, the skin has full elastic properties and recovers promptly after each contraction, and the wrinkles and lines generated by the muscles of facial expression are accurately defined as dynamic. Over time, however, skin elasticity, strength, and resistance decrease, and muscle action eventually produces wrinkles, lines, and furrows that remain at rest. A static component thus becomes increasingly visible, harboring increasing aging.

Botulinum toxin treatment of mimetic muscles, also termed chemodenervation, requires a thorough knowledge of muscle anatomy. While routine aesthetic practice may require treatment of 20 to 30 muscles, treatment of patients with facial palsy may require treatment of additional muscles. The closely adjacent facial muscles lie mostly within the superficial muscolar aponeurotic system (SMAS) of the face and have a distinct three-dimensional structure and orientation. Precise injections of the drug are therefore essential in maximizing efficacy and minimizing complications. The dose is equally important. The injected units should ideally saturate the specific target receptors, leaving no unbound molecules free to move to nearby unwanted muscles. The clinician needs to understand that delivering suboptimal doses will negatively affect duration of result, although not giving rise to complications. Conversely, higher-thanrecommended doses may significantly increase the rate of side effects.

Precision in botulinum toxin treatments is key to success. The injector must know the exact localization and depth of the target muscles and the ideal dose for combining efficacy and result. Another extremely important safety-related issue is diffusion rate. Ideally, the formulation with the lowest diffusion potential would guarantee the highest safety margin. In Europe, North America, and many countries, only three different formulations are available. Each of them has a robust literature claiming similar or better performances than its competitors, including the lowest diffusion profile. However, most of the papers are company sponsored and may lack objectivity. Although highly experienced injectors all have personal preferences, it is fair to state that these are often based on subjective impressions only and that to date there is no clear-cut superiority of one formulation over the others in terms of general performance. This is especially true when it comes to the diffusion rate. As long as a licensed product is preferred and correctly administered, efficacy and safety are guaranteed.

RECONSTITUTION

Presently, all licensed formulations are supplied as powders and require reconstitution with saline immediately prior to use. Although physicians are free to dilute according to personal preference, there are well-tested and universally accepted company recommendations. In Europe, licensed formulations are marketed in 50 U vials (Allergan's Vistabel and Merz's Bocouture) and in 125 U vials (Galderma's Azzalure). This raises the vital point that botulinum toxin units are not interchangeable between companies because the biological potency assay tests differ. This is an unfortunate confusion factor for novice injectors. However, there is general agreement that Allergan and Merz dosage units are reasonably comparable (1:1), and that Galderma units are equivalent to Allergan and Merz units in a 2.5:1 ratio. Thus, the Azzalure vial containing 125 U is more or less equivalent to the 50 U vial of Vistabel and Bocouture. Company recommendations from Allergan and Merz suggest diluting the 50 U vial with 1.25 mL of plain saline. This yields 4 U per 0.1 mL of solution. Galderma, on the other hand, has historically recommended a dilution of 0.63 mL per 125 U vial, thereby rendering a solution twice as concentrated as its competitors. It is beyond the scope of this chapter to discuss this rationale. It is fair to state, however, that the double concentration requires considerable extra care for dose precision and may thus obviate an otherwise excellent product. Recent literature has compared the efficacy and safety of Azzalure diluted at 0.63 mL versus 1.25 mL. The results with the 1.25 mL dilution have been as good, if not better, than with the double concentration. This allows the injector to dilute all three formulations with 1.25 mL of saline and to consider the potential of the resulting solution comparable to the others.

A few words should also be spent discussing injection pain. Although generally well tolerated, delivery of the drug may be unpleasant. In part, this is due to the needle, and the thinnest possible gauge size will help to minimize this component. While many injectors use a 30G, the 31-33G sizes may be preferable, although they dull quickly and require frequent changes. However, it is the solution that is mostly responsible for pain during injections. Some authors recommend nerve blocks, but these are possibly more aggressive than necessary.

If available, preserved saline (NaCl 0.9% + benzyl alcohol 0.9%) should be used for dilution because the solution is almost pain-free without losing any of its pharmacological properties. Another option is to use plain saline + lidocaine for dilution. This will not impair the effect of the treatment and does reduce pain to some extent; however, preserved saline seems to be the best option. Although companies recommend keeping the reconstituted solution at 4°C and injecting within 24 hours, both clinical experience and significant literature suggest that the solution remains active for weeks.

MANAGING PATIENT EXPECTATIONS

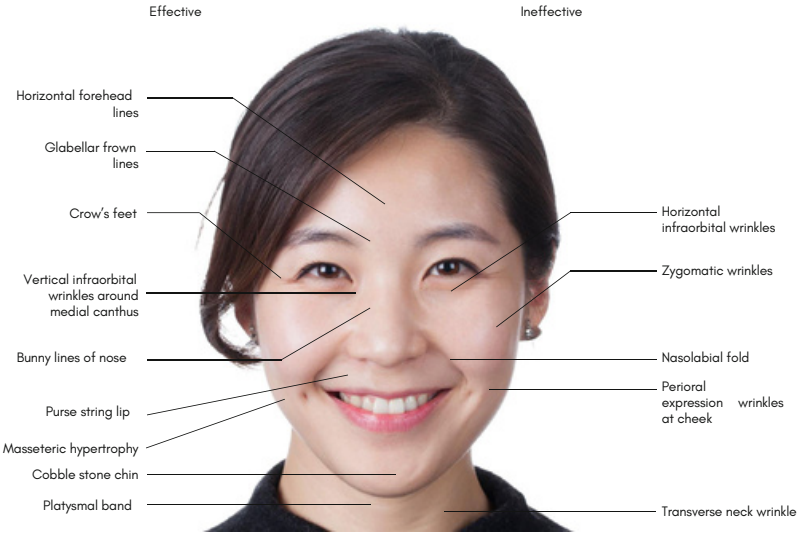
Careful patient assessment clarifies both the possibilities and limitations of individual treatments. Matching clinical assessment with realistic patient expectations is key to successful practice as careful pre-treatment explanation will be generally well accepted, while belated explanations may be construed as excuses. Although botulinum toxin may lead to drastic improvement of facial lines, there may not always be complete eradication. Repeated treatments have been documented to enable longer-lasting results. Patients with very deep or resting frown lines should be realistically informed during initial consultation, and adjunctive treatments, such as careful intradermal injection of hyaluronic acid microdroplets, discussed upfront. Honest and straight-forward physician information helps to establish trust and develop sustainable patient relationships.

EFFECTIVE VERSUS INEFFECTIVE INDICATIONS OF BOTULINUM TOXIN FOR WRINKLE TREATMENT

A wrinkle, or rhytide, is categorized into dynamic and static conditions. Static wrinkles can be further divided into either fine lines or furrows/deep furrows. Dynamic wrinkles can be seen at the glabellar region when frowning or around the periorbital regions when smiling. Glabellar furrows and nasolabial folds are representative cases of deep furrows. The process of the wrinkle formation begins from dynamic wrinkles, and then the dynamic wrinkles become fi xed as fi ne lines. With age, fi ne lines transform into deep furrows accompanied by a gradual loss of the underlying soft tissues.

Although botulinum toxin is often believed to be the mainstay of wrinkle treatment by general people, it cannot be applied to the treatment for all wrinkles. Main indications of botulinum toxin for wrinkle treatment are dynamic wrinkles such as forehead lines, glabellar frown lines, periorbital wrinkles, wrinkles on dorsum of the nose, fine lines around the lips, and the platysmal bands by paralyzing the underlying facial mimetic muscles. However, it is ineffective in treating furrows. Therefore, filler injections should be recommended for the treatment of furrows, including nasolabial folds, the marionette lines, and glabellar frown lines.

Some static wrinkles, such as horizontal necklines and bracelet lines, are not amenable to treatment by botulinum toxin because they are innate lines since birth. On the other hand, fine lines, which result from habitual contraction of the underlying facial mimetic muscles, can be improved with botulinum toxin injection to some extent. The representative areas where the toxin treatment can bring about improvement of fine lines include forehead, glabella, and lateral canthal area. The mechanism of botulinum toxin for improving fi ne lines can be hypothesized in many different ways. Firstly, it can weaken the muscle tone of underlying facial muscles attached directly to the skin. As a result, the fine lines in repose seem to improve. Secondly, the paralysis of the facial mimetic muscles attached to dermis leads to lymphatic insufficiency, and subsequent dermal edema in the skin may happen. The consequence can improve fine wrinkles and facial pores.



Botulinum toxin is ineffective in some types of dynamic wrinkles. The representative examples are transverse infraorbital wrinkles and zygomatic wrinkles which are caused by the mouth corner elevators while smiling. The botulinum toxin injection should be also avoided in the area of cheek and nasolabial folds where dynamic wrinkles are deepened during facial expression. If the botulinum toxin is injected onto these mouth corner elevators such as zygomaticus major muscle, awkward and asymmetrical smile may occur. This kind of dynamic wrinkles could be improved only by multiple intradermal injections of hyaluronic acid filler.

ABSORBABLE SOFT TISSUE FILLERS: CORE CHARACTERISTICS

INTRODUCTION

The increasing popularity of minimally invasive cosmetic procedures in recent years has caused a surge in the use of soft-tissue fillers. Minimally invasive cosmetic procedures have increased 300% from 2000; among the most utilized minimally invasive cosmetic procedures, soft-tissue fillers rank second, behind neuromodulators.

Hyaluronic acid (HA) products are currently the most utilized soft-tissue fillers. Adequate working knowledge of individual products, injection techniques, and anatomic principles is vital to improve outcomes and to prevent and minimize complications.There is a plethora of literature detailing the use and safety of soft-tissue fillers. Each company is keen to highlight unique and proprietary characteristics which set their product apart from the competitors. The sheer choice and volume of different soft tissue fillers flooding the market may be overwhelming for novice and even seasoned injectors.

The aim of this chapter is to allow an unbiased description of common HA and non-HA absorbable soft-tissue fillers as a rough guide for decision making. This chapter is not intended to be exhaustive as this would be beyond the scope of this book in which we endeavour to cover “the essentials for injections”. Basic rheology, HA manufacturing processes and short review of injections and layering principles by region are discussed with a focus on patient safety.

Prevention of complications and management of paramount importance when utilizing soft tissue fillers are covered in a different section of this guide.

HYALURONIC ACID FILLERS

HA is a naturally-occurring component of the extracellular matrix. It is a glycosaminoglycan (GAG) polymer consisting of repeat disaccharide units of glucuronic acid and N-acetylglucosamine. Approximately 50% of the body’s total HA is in the skin.

HA acts as a scaffold for the extracellular matrix, providing rigidity, hydration and turgor whilst allowing cellular movement and regeneration. It is also important in protecting the skin from free radical damage, particularly against UVA and UVB. HA is rapidly metabolised in the tissues, with one third of total body HA being turned over daily

HA from animal sources generally has longer polymer chains than in those from bacterial sources (usually from the Streptococcus equi species); they are forbidden in Europe as soft tissue fillers. Bacterial fermentation is generally preferred as a source for HA because it is less likely to be antigenic, being free from foreign proteins and easier to purify

(a) (b) (c)

(d)

Crosslinking HA polymer chains transform the HA solution (a) into a gel (c). Crosslinker molecules (b) bind individual HA polymer chains to create a network (c), which manifests macroscopically as a gel mass (d).

HA SOFT TISSUE FILLERS

HA soft tissue fillers consist of long chains of hyaluronic acid. Most dermal filler products will consist of HA crosslinked with a chemical such as 1,4-butanedioldiglycidyl ether (BDDE) for Restylane®, Belotero®, Teosyal®, Hyabell®, Stylage® and Juvéderm® and suspended in a physiological or phosphate-buffered solution .
The process of crosslinking adds a molecule to link the polymer chains to each other, thus modifying their physical properties to make them longer-lasting and less likely to be degraded. The most commonly used crosslinker is 1,4-butanediol diglycidyl ether (BDDE); BDDE has a significantly lower toxicity than other crosslinking agents (e.g., divinyl sulfone or formaldehyde) and is biodegradable.The product is then processed "sized" into smaller, crosslinked domains to allow for injection through a needle into the skin as a homogeneous gel or a suspension of particles in gel carriers.

Variability in methods used to manufacture HA fillers have given rise to differences in properties such as degree of cross-linkage, particle size and concentration. These properties are vital in determining the clinical performance of the filler.

The HA filler manufacturing process usually consists of the following steps:

- Dilution of HA powder in basic medium
- Mixing of BDDE (ratio is important for quality)
- Cross-linking reaction (heating)
- Dilution of gel in basic medium (addition of lidocaine)
- Purification
- Mixture of purified cross-linked (and sometimes uncross-linked HA)
- Degassing, then filling the syringes
- Gel sterilization
- Blistering and packaging

HA fillers can be classified according to their particulate forms: either monophasic or biphasic gels. Monophasic gels consist of a single 'phase' of HA.

They can be either monodensified HA is mixed and cross-linked in a single step (e.g., Juvéderm and Teosyal) or polydensified HA goes through two stages of cross-linking (e.g., Belotero). Biphasic gels such as Restylane and Perlane consist of two 'phases' of HA cross-linked HA of a specific size which is then suspended in non-cross-linked HA acting as a carrier.

Controversy remains over the relative clinical effectiveness of monophasic vs biphasic hyaluronic acid fillers; it is likely that no single method is superior to another, rather that the different physical properties of dermal fillers are more suitable for different clinical
indications.

DERMAL FILLER RHEOLOGY

"Rheology" is the study of the physical characteristics that influence the way materials behave when subject to deforming forces. Once injected, fillers are subject to shearing, vertical compression and stretch from muscle movements, compression, and gravity.

It is our role as practitioners to understand the way fillers will behave when injected into a particular area or layer of the skin and to choose the most appropriate dermal filler to achieve the desired aesthetic result. Fillers used to treat different parts of the face have very different desirable qualities. For example, when treating the deep supra-periosteal layers of the chin or jawline, it is important that the filler gives good volume and projection without spreading through the tissues. Conversely, when injecting into superficial dermal layers, it is important that fillers can easily spread through the tight connective tissue in order to sit smoothly in the upper layers of the skin.

A number of factors affect the physical characteristics of HA dermal fillers. These include:

• Elastic modulus (G'): The ability to recover the original shape after shear deformation. Elasticity is the ability of a material to return to its original shape after being deformed.

• Viscous modulus (G″): The inability to recover the original shape after shear deformation. Viscosity is a measure of the resistance of a fluid, which is being deformed by either shear or tensile stress.

• Complex modulus (G*): The total ability of material to withstand deformation. It is defined as the sum of the elastic modulus (G′) and viscous modulus (G″).

• Tan δ: The ratio between the viscous modulus and the elastic modulus corresponds to the loss tangent (loss factor) tan δ, and thus describes the ratio between the elastic and the viscous share of a polymer fluid. If the loss tangent δ is greater than 1, the material is predominantly viscous, and if it is smaller than 1, the material is predominantly elastic.

• Cohesivity: The cohesivity of a filler is the strength of the cross-linking adhesion forces that hold the individual HA units together. Cohesivity is determined by the concentration of HA and the degree of cross-linking. High cohesivity helps the filler maintain vertical projection. A gel must be cohesive in order to avoid any migration. Visco-elastic properties and crosslinking determine how cohesive the gel is. A gel with low cohesivity is suitable for delicate and superficial treatments. A gel with high cohesivity has more volume and lifting capacity for structural contouring. The extrusion force of the gel is also important and differs amongst various fillers.

• Lift Capacity: The lift capacity of a filler is its ability to oppose deformation and flattening and affects its suitability for different applications, whether for more superficial correction of fine lines or deeper use for wrinkles and folds, volumizing, and contouring. G′ has usually been previously used to predict and describe the lift capacity of a filler, but lift capacity has also now been treated as a function of both elastic modulus (G′) and gel cohesivity and will differ among products using proprietary manufacturing processes.

• Resistance to Deformation: This phenomenon the ability to mould a filler in order to achieve a desired effect is important for the clinician. The resistance to deformation of a filler is a function of the physical properties of its chemical composition, including cohesivity.

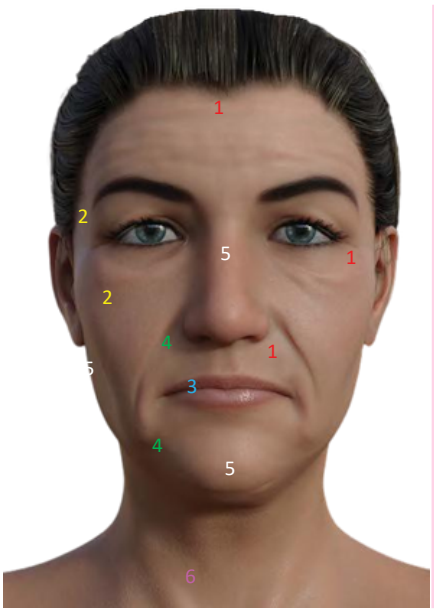
• Tissue Integration: Tissue integration the way the filler integrates with or distributes into surrounding tissue is an essential parameter for clinical and marketing purposes and also by which to assess products under development . When injected, HA fillers tend to spread within the reticular dermis and to distribute between the dermal fibers, but different HA dermal fillers behave differently if different crosslinking technologies have been used to create specific viscoelastic properties. Several studies have investigated the behavior of different crosslinked dermal fillers, whether injected intradermally or in the subcutaneous layer. Histology is usually employed to give a qualitative measure of tissue integration.

Rheology is not sufficient by itself to completely understand filler performance but it is useful for an evaluation of different fillers of similar composition; animal models have been developed to allow such comparative evaluations among fillers. The results indicated that biological interaction has an important role to play in the filler's clinical performance.

HA fillers can be dissolved with hyaluronidases, increasing their "safety" when compared to non-HA fillers. HA levels are determined by the balance between enzymes that create it (synthase HAS1, HAS2 and HAS3) and those that break it down (hyaluronidases HYAL1, HYAL2 and HYAL3). Hyaluronidases are enzymes licensed for enhancing penetration of subcutaneous or intramuscular injections, local anaesthetics and infusions and reduce swelling. However, they are also widely used "off-label" in aesthetic medicine to dissolve hyaluronic acid fillers. The enzymes can be classified by their mechanism of action: mammalian (endo-Beta-N-acetylhexosaminidase), leech/hook-worm (endo-Beta-D-glucuronidase) and microbial (hyaluronate lyase). The most commonly-used preparation in the UK is Hyalase, derived from sheep; however, microbial and human hyaluronidases appear to have advantages in terms of safety and reduced immunogenicity.

CHALLENGES IN FILLER CHOICE IN FACIAL AESTHETICS

Harmony in facial rejuvenation and replenishment requires an understanding of the complex anatomy and dynamic structure of the face and its evolution during the facial ageing. Fillers implanted in the face area subjected to forces such as lateral shear and compression/stretching from intrinsic tissues and extrinsic sources. The challenge in the development of novel fillers will consist of optimizing their tailoring for the ideal mechanical properties for each specific indication and facial region;



1 **Forehead, Fine Lines, Periorbital, and Perioral**
Low viscosity
Low-medium elas city G'
Low cohesivity
2 **Mid-face, Temple, and Lateral Brow**
Low-medium viscosity
Medium-high elas city (G')
Medium-high cohesivity
3 **Lips**
Low viscosity
Low-medium G'
Low-medium cohesivity
4**Lower Face(marione e lines, nasolabial folds, accordion lines)**
Medium viscosity
Moderate elas city G'
Low-medium cohesivity
5 Nose, Chin, and Jawline
Medium-high viscosity
High elas city, high G'
High cohesivity
6 **Neck and Décolletage**
Very low-low viscosity, low elas city G'
Low cohesivity
Non or low crosslinked fillers (skin boosters)

Basic soft tissue filler rheology.

Area: Forehead, Fine Lines, Periorbital, and Perioral

Aim: To restore volume in intradermal and subdermal planes.

Filler properties: Allows easy moulding and spread of product for smooth effect, non-bulking.

Rheology: Low viscosity; Low-medium elasticity, G′; Low cohesivity with low-to-medium G*. For the tear trough it is important that there is minimal "water attraction."

TECHNOLOGY OF COMMON ABSORBABLE FILLERS

This list, derived from articles indexed in PubMed and from company publications, is by no means exhaustive.

Cohesive Polydensified Matrix (CPM) (Merz-Anteis, Belotero Range)

CPM is based on a dynamic crosslinking process, with two additional steps in the crosslinking process. Introducing an additional amount of HA produces a monophasic polydensified gel that combines a high level of crosslinked HA; and continuation of the crosslinking process leads to a lighter level of crosslinked HA, in a cohesive matrix

Interpenetrating Network Like (IPN-Like) (Vivacy, Stylage Range)

Two or more monophasic HA gels are separately crosslinked and then mixed together, forming an interpenetrating network. An antioxidant —mannitol— is added to protect the HA chains from oxidative stress

Non-Animal Stabilized Hyaluronic Acid Technology (NASHA) (Galderma, Restylane Vital and Vital light, Restylane Lyft/Lyps/SubQ)

A small amount of BDDE is added between polysaccharide chains in specific conditions to allow the formation of a complex matrix of HA gel. A sizing process generates the HA gel particles that are then suspended in a fluid phase. The resulting product range is based on the particle sizes adapted to specific clinical indications of the final product

Optimal Balance Technology (OBT)/ XpressHAn Technology (Galderma, Restylane Refyne/Defyne/ Volyme)

The same concentration of hyaluronic acid is maintained in the entire range, but with different degrees of crosslinking and different sizing for the gel calibration that allows it to be injected through a fine needle.

Resilient Hyaluronic Acid (RHA) (Teoxane, Teosyal RHA Range)

RHA range is characterized by a gel with long HA chains stabilized with a low amount of BDDE while minimizing the degradation of the HA during the process. The products in this range differ in the degree of crosslinking as well as their HA concentration. The products allow "strength" for higher G′ and "stretch" for lower G′

Vycross Technology (Allergan, Juvéderm Vollux, Voluma, Volift/Vollure, Volbella, Volite)

This technology uses a proprietary combination of lower (between 0.5 and 1 MDa) and higher (>1 MDa) molecular weight HA to improve the crosslinking efficiency between HA chains. The increased proportion of lower molecular weight HA allows a higher concentration during the crosslinking and a higher efficiency of the reaction

Monophasic Particle Technology (MPT) (Adoderm, Varioderm® Range, Hyabell® Range)

This technology approach permits to customize the degree of cross-linking (going up to 80% of efficient connections between the molecules of HA chains) and concentrations. Main characteristics of these gels are the difference in concentrations, its balance on rheological values while granting a soft and homogenous extrusion force

ProfHilo NAHYCO™ technology (IBSA)

This is a slowly degraded HA without chemical modification, available as intradermal injectable formulations with intended enhanced injectability, longer duration, and high biocompatibility. The hybrid cooperative complexes are supposed to deliver a higher HA amount than the unmodified HA products. The formation of the hybrid cooperative complexes is characterized by a drop in dynamic viscosity that, in clinical practice, allows the clinician to inject very high concentrations of HA.

These formulations can be defined “physical gels”, in which interactions between long and short HA chains were made, without changing the disaccharides unit structure and without introducing other “chemical compounds”. A key feature of the hybrid cooperative complexes is claimed to be prolonged stability to enzymatic attack, despite the absence of chemical cross linking.

Neuvia Organic (Intense, Intense IV, Intense Lips, Intense Rose)

IPN stands for Interpenetrating Polymer Network, a technology to combine two different polymers HA (from Bacillus subtilis) and PEG as the cross linker in one network to obtain a 3D hydrogel matrix.

Neauvia Organic Stimulate is intended to offer not only filling effect but also biostimulation. It consists of HA with 1% of calcium hydroxyapatite (8-12 microns).

NON-HA FILLERS

Poly-L-Lactic Acid (PLLA)

PLLA is an absorbable polymer which stimulates fibroblast production and generation of collagen; results usually last for around two years. For optimal results, multiple treatment sessions are often required. The main concern with PLLA is a delayed development of palpable nodules. However, a study by Woerle et al. on 300 patients followed-up over five years reported that with adequate dilution, longer hydration time, addition of lidocaine and proper handling of the vials, the incidence of nodule formation is below 1%. Similar recommendations were made by Alessio et al. The most widely known filler that contains PLLA is Sculptra (Dermik Laboratories), which was approved by the FDA in 2004 for the correction of facial lipoatrophy in patients with HIV.

Calcium Hydroxylapatite (CaHA)

Radiesse (MERZ) is the only CaHA filler approved by the FDA; it was approved first in 2006 for the correction of facial lipoatrophy in patients with HIV, and for moderate wrinkles and skin folds. Radiesse is composed of 30% calcium hydroxylapatite microspheres suspended in a 70% gel carrier. It is a synthetic compound, similar in structure to bones and teeth. Radiesse is non-immunogenic, hence does not require patch testing, and is fully degraded and excreted by the body. The corrective results last for approximately 12 months.

When used in hyperdiluted form (i.e., 1.5 mL of product plus ≥1.5 mL of diluent), Radiesse has a minimal or absent immediate volumizing effect due to carboxymethylcellulose gel dispersion, generating only long-term tissue remodeling by the CaHA micro-spheres and allowing its injection more superficially for dermal rejuvenation and the treatment of larger areas such as neck, décolletage, buttocks, thighs, arms, abdomen, knees, and elbows.

Polycaprolactone (PCL)

Ellansé (Sinclairpharma) is CE marketed as a biodegradable collagen stimulator, composed of microspheres of a bioresorbable polymer, polycaprolactone (PCL), in an aqueous carboxymethyl cellulose (CMC) gel carrier. PCL biodegradation occur via hydrolysis of the ester linkages that are totally eliminated from the body. Four versions are available: Ellansé-S (short, S version), Ellansé-M (medium, M version), Ellansé-L (long, L version), and Ellansé-E (extralong, E version), with expected in vivo longevity of 1, 2, 3 and 4 years, respectively.

SUMMARY

Hyaluronic acid is a natural constituent of human skin present in abundance. HA is transformed into soft tissue filler for aesthetic treatments by stabilisation with cross-linking proteins, usually 1,4-BDDE. This makes HA more resistant to degradation and therefore enables it to last several months in the skin.

HA fillers are characterized through a combination of rheology, animal, or clinical performance evaluations to help the clinician better understand the relative performance attributes of different fillers when used in a biological environment.

Emerging technologies used for manufacturing HA dermal fillers with different rheological properties are continuing to come onto an expanding aesthetic market.

Non-HA fillers are intended to have certain advantages such as a longer duration of effectiveness and generation of collagen in the skin but cannot be dissolved with hyaluronidase.

Both novice and seasoned injectors must remain on top of their game to keep up with these technologies, allowing them to choose the filler with the most optimal properties for the patient's indication, providing safe and effective treatments.

COMPLICATIONS OF ABSORBABLE FILLERS

COMPLICATIONS OF ABSORBABLE FILLERS

Injectable fillers currently constitute the second most commonly performed aesthetic procedure after botulinum toxin. The tremendous market expansion, coupled with new treatment paradigms and inadequate control of both products and injectors, has heralded a concerning increase in serious adverse events. Complication recognition and management have become the most significant unmet needs for filler treatments.

A multitude of soft tissue fillers are currently available for facial aesthetic indications, ranging from autologous fat, polymethylmethacrylate, calcium hydroxylapatite, poly-L-Lactic acid, polycaprolactone, and hyaluronic acid (HA). Because HA fillers have the powerful advantage of being completely removable by the use of hyaluronidase, depending on approval in specific countries, they are referred to as reversible and are currently the most widely used dermal fillers. For this reason, this chapter is aimed primarily at complications arising from the use of HA fillers.

The optimal approach to filler complications lies in having practical strategies for their prevention, as well as the insightful knowledge required for timely diagnosis and treatment.

PREVENTION

All injectors should work with an operative strategy aimed at reducing the risk of complications. Careful pre-consideration should be given to the possible confounding factors specific to each procedure. Although by no means comprehensive, the following 10-point plan may be used as a simple pre-injection checklist.

History and selection: It is advisable to invest time in a pretreatment consultation, with elucidation of skin conditions, systemic disease, medications, and previous procedures. The treatment plan should ideally be structured over time with due consideration given to pending medical procedures, dental visits, and immunizations. These steps aim to limit inflammatory reactions or hypersensitivities due to a heightened immune system.

Skin barrier disruption due to inflammatory or infective conditions may persist for 3–4 weeks after apparent clearing of skin conditions, thus allowing the penetration of infective agents. Acne, rosacea, and dermatitis should be adequately treated, with an additional 3–4 weeks allowed for repair of optimal barrier function before filler treatments are performed. Increased numbers of resistant P. acnes at the edges of topically treated acne areas are thought to play a role in the formation of biofilms via the toll-like receptors (TLR-2), and the "safe distance" for filler placement relative to an area of acne is unknown.

Ascertain the use of current antibiotics and indications thereof as patients with remote infections involving the urinary tract, sinuses, intestinal tract, and oral cavity are best deferred for treatment. Hematogenous spread of normally non-virulent bacteria may lead to binding to the toll-like receptors (TLRs) with possible triggering of an immune response and formation of late-onset nodules many months later. Prophylactic antivirals are advised to prevent virus reactivation if there is a history of herpes simplex infection in the intended area of injection.

Dental procedures, visits to the oral hygienist, and tooth bleaching/whitening are best avoided during the 2–4-week period before and after filler treatment to reduce the risk of hematogenous bacterial seeding and potential development of biofilm.

Filler treatments are contraindicated in active autoimmune diseases such as systemic lupus erythematosus, rheumatoid arthritis, mixed connective tissue disease, and Hashimoto's thyroiditis, but may be performed in burnt out conditions such as end-stage morphoea.

It is preferable to avoid injecting patients with multiple, severe allergies and a history of anaphylaxis. Drug allergies might also preclude optimal management of complications, should they arise. Knowledge of previous surgical and nonsurgical cosmetic procedures is vital as these could cause anatomical repositioning of structures and fixation and scarring of underlying vasculature, thus facilitating intravascular placement. Knowledge of the types and location of previously injected products may help to prevent compatibility issues with minimally degradable fillers.

Patients should be given a pre-filler checklist as an exclusion questionnaire in order to emphasize the importance of having no infective or inflammatory conditions (cutaneous or systemic) at the time of treatment. The checklist should ideally include a list of common anticoagulating compounds (medications and foodstuffs) for avoidance in the week before treatment (aspirin, nonsteroidal anti-inflammatories, salmon oil, vitamin E, gingko biloba, alcohol, dark chocolate, grapefruit, etc.).

POST-TREATMENT CHECKLIST

• The patient should be furnished with written post-treatment instructions and contact numbers. Common-sense advice such as washing the face with uncontaminated water, using a new lipstick, and applying uncontaminated facial products should be given.
• The injector/clinic should be available via phone for 48 hours post-procedure.
• It is good practice to have a staff member call the patient the next day.

2. Assessment: Consider the intricacies of ethnicity, gender, and generational needs in order to construct an applicable treatment plan.

3. Informed consent: Signed informed consent is crucial in creating awareness of the potential risk of filler-induced complications. Written preand post-treatment instructions help to establish realistic expectations and minimize legal repercussions.

It is wise to obtain informed consent for both the procedure as well as the management of inadvertent complications, should they arise, in order to expedite efficient management. This includes the discussion of possible, albeit rare, ophthalmic complications

4. Reversibility is a powerful advantage when using HA products. Practical knowledge pertaining to locally available hyaluronidases and their effect on locally available HA fillers is of paramount importance as the required dosages may differ. Certain products require more massage than others in order to dissolve adequately.

5. Product characteristics such as HA concentration and proprietary crosslinking should be understood in the context of ideal depth, placement, and duration.

The hygroscopic nature of HA is an important determinant of product-related swelling and needs to be differentiated from procedural swelling. The HA concentration and extent of crosslinking determine the product's characteristics (viscosity, elasticity, resistance to degradation, G′ [elastic modulus], G″ [viscous modulus], and Tan Delta) and ultimately its clinical efficacy and ideal depth of placement.

6. Product layering over late or minimally degradable fillers is discouraged, although layering of HA filler over other HA fillers is generally deemed acceptable. Late or minimally biodegradable fillers may be provoked into reactivity when a second filler such as HA is layered over them, potentially inducing long-lasting complications such as foreign body granulomas. Although HAs remain the most compatible fillers, it is wise to be cautious when considering cross-brand layering.

Accurate knowledge of the types and locations of previously injected products may help to prevent compatibility issues with minimally degradable fillers, and filler types should be meticulously documented after each treatment.

7. Photographic documentation (preand post-procedure) is vital for patient monitoring, medio-legal purposes, and as a tool for self-education.

8. Procedural planning and aseptic technique are pivotal in avoiding complications, and it is essential to prevent breaching of the clean workspace. Consider the following:

• Have everything at hand to reduce breaks in the aseptic field and the concomitant risk of injection-related infections.

• A preconceived plan and clear procedural flow help to minimize complications.

Makeup should be removed, and the skin cleansed carefully with, for example, 2% chlorhexidine gluconate in 70% alcohol. Avoid ocular exposure to the disinfectant as chlorhexidine is toxic to the cornea. Beware of soaked or dripping gauze when used near the eyes or perforated tympanic membranes (due to ototoxicity).

• Stringent aseptic technique is mandatory: cleanse, degrease, and disinfect. There are no universally recommended topical antiseptics, but chlorhexidine, chloroxylenol, iodophors, alcohol, iodine, and hypochlorous acid may be appropriate.

• Rinsing the mouth with an antiseptic mouthwash containing chlorhexidine (0.2%) or povidoneiodine adequately disinfects the oral cavity for up to 8 hours and has been suggested as a preventative practice for perioral treatments or patients with a lip-licking habit.

• The treating physician should remove all jewelry, wash their hands with antiseptic cleanser, and use gloves for all cases of injection therapy. The procedure is not deemed sterile as the syringe itself is not completely sterile. Thus sterility is lost once the syringe is handled, making aseptic technique of paramount importance.

• Surgical principles of sterile technique, i.e., not touching any component of the needle or cannula that penetrates the skin, may further reduce infective complications. Constant vigilance against possible contamination is of the utmost importance.

• Cleansing over a sufficiently broad area is imperative as there is a higher infective risk upon inadvertent touching of cannulae on the adjacent skin.

• Frequent needle (and cannula) changes are advised for multiple entry points.

• The use of disposable sterile dressing trays with containers for prep solution, gauze, and disposable sterile drapes enhances a safe, clean work area in an office environment.

9. Insightful knowledge of injection anatomy is of paramount importance in avoiding danger areas and serves as the foundation for avoiding disastrous complications. Although the exact position of vessels is highly variable, the plane in which they run is far more predictable. Therefore, knowledge of “safety by depth” is a vital safety tool, as is constant awareness of the early signs of vascular compromise.

Seckel has divided the face into various danger zones, knowledge of which is important when treating specific regions.

Danger Zone 1

This zone is located by turning the patient’s head to the opposite side, palpating the sternocleidomastoid muscle, and drawing a straight line from the caudal edge of the external auditory canal to a point 6.5 cm below on the midpoint of the muscle belly. The area is encircled with an approximate radius of 3 cm.

The zone includes the region in which the **great auricular nerve** emerges from beneath the **sternocleidomastoid muscle**, making it susceptible to injury when dissecting over the muscle. The great auricular nerve originates from the cervical plexus branching off spinal nerves C2 and C3 and provides sensation to the skin on the mastoid area, parotid area, and the outer ear surface. Permanent injury to this nerve results in numbness of or painful dysesthesia (in case of neuroma) of lower two-thirds of the ear and adjacent neck and cheek skin.

Danger Zone 2

This zone is anatomically located by drawing a line from 0.5 cm below the tragus to 2 cm above the lateral eyebrow. Another line is drawn along the zygoma to the lateral orbital rim. The last line is then dropped from the point above the eyebrow through the lateral end of the brow to the zygoma. These lines form a triangle in which the temporal branch of the facial nerve lies on the undersurface of the temporoparietal fascia-superficial muscolar aponeurotic system (SMAS) layer and is more likely to be injured with deep-plane dissections.

This zone includes the region in which the **temporal branch** of the **facial nerve** runs under the **temporoparietal fascia-SMAS** layer. The branch emerges from beneath the parotid gland at the level of the zygoma and innervates the frontalis muscle in the forehead. Temporal branch injury may lead to paralysis of the frontalis muscle. The involved side of the forehead becomes paralyzed with ptosis of the brow and asymmetry of the brows.

Danger Zone 3

This zone includes the **marginal mandibular branch** of the **facial nerve** at its most vulnerable point and also the **facial artery** and **vein**. This zone is located by identifying a point 2 cm posterior to the angle of the mouth and drawing a 2 cm radius circle based on it. At this zone, the platysma-SMAS layer thins, thereby exposing the nerve and nearby facial vessels that are susceptible to injury.

Damage to this nerve leads to significant aesthetic and functional repercussions. At rest, the tone in the normally innervated zygomaticus major muscle will be unopposed by the now denervated DAO muscle, resulting in elevation of that corner of the mouth and the lower lip pulled up over the teeth. During grimacing or frowning, the now denervated DAO muscle cannot depress the corner of the mouth and lower lip, meaning the lower teeth will not show on the affected side.

Danger Zone 4

This zone includes the **zygomatic** and **buccal branches** of the **facial nerve**, which are superficial to the masseter muscle and Bichat's fat pad but deep to the platysma-SMAS and parotid fascia layers. These branches are no longer protected by the parotid gland and therefore are more vulnerable. The danger zone is located in a triangular region bound by the body of the mandible inferiorly, the parotid gland posteriorly, and the zygomaticus major muscle anteriorly. Using surface anatomy, this can be delineated by having a point at the oral commissure, at the highest point of the malar eminence, and the posterior border of the angle of the mandible. Injury to these nerves can result in paralysis of the zygomaticus major, zygomaticus minor, and levator labii superioris muscles.

This results in sagging of the upper lip during rest and a more apparent disfigurement during smiling when the contralateral unopposed zygomaticus major and minor muscles pull the mouth toward the innervated side.

If nerve damage occurs, the muscle paralysis is often not long-term, given the multiple interconnections between branches of the buccal and zygomatic nerves.

Danger Zone 5

This zone lies at the superior orbital rim above the mid-pupil where the **supraorbital (CN V)** and the more **medial supratrochlear (CN V) neurovascular bundles** are found. The supraorbital nerve lies deep to the corrugator supercilii muscle (CSM), and the supratrochlear nerve passes through the CSM. Nerve injury may cause numbness of scalp, forehead, upper eyelid, and nasal dorsum. This danger zone can be identified with a 1.5 cm circle with the supraorbital foramen at its center, which is easily palated at the supraorbital rim at mid-pupil l eve l.

Danger Zone 6

This zone lies in the infraorbital region where the infraorbital (V2) neurovascular bundle exits the infraorbital foramen. Nerve injury may cause numbness of the lateral upper nose, cheek, upper lip, and lower eyelid. Zygomatic branches of the facial nerve also run in this zone to innervate the levator labii superioris muscle. This danger zone can be identified by drawing a 1.5 cm circle that centers around the infraorbital foramen located 0.8–1 cm below the infraorbital rim at mid-limbus level.

Danger Zone 7

This zone contains the mental nerve which carries sensory innervation to the ipsilateral chin and lower lip and is a branch of the mandibular branch of the trigeminal nerve. The mental nerve exits the mental foramen, which is located at the midpoint of the body of the mandible, in line with the second lower premolar.

The mental foramen lies on a sagittal line drawn through the mid-limbus of the pupil, the supraorbital, and the infraorbital foramen.

Implications of nerve damage can be significant, with patients often not noticing when food is dribbling from that side of their mouth. Inadvertent lip biting may ensue while chewing.

10. Technical knowledge of placement and injection depth is cardinal to the success of dermal fillers.

Strategies for optimizing technique include:

• Knowledge of injection anatomy.
• Awareness of danger areas.
• Aspiration before injecting where applicable.
• Slow injection speed with the least amount of pressure possible.
• Moving tip with delivery of product where applicable.
• Incrementally injecting small 0.1–0.2 mL aliquots of product.
• Use of small syringe to deliver precise aliquots.
• Use of small needle to slow down injection speed. • Use of blunt cannulae where indicated.
• Careful consideration of the patient's medical history.
• Stopping injection if resistance is encountered or the patient experiences pain/discomfort.
• It is vital to routinely check perfusion in the treated areas as well as areas with watershed perfusion (glabella, nasal tip, upper lip), ensuring that makeup is not obscuring skin tone.
• Initial signs of vascular compromise may be subtle and fleeting.

CLASSIFICATION OF COMPLICATIONS

Filler complications are traditionally divided into four categories: allergic, intravascular, infective, and late onset nodules/inflammation. They may also be classified by onset, although multiple publications and timeframes currently lead to a lack of consensus. A 2014 consensus led by Signorini proposed a more generalized scheme of early and late-onset events.

The South American consensus panel defined three main intervals:

- Immediate onset (up to 24 hours)
- Early onset (24 hours to 30 days)
- Late-onset (after 30 days)

The panel also proposed the term persistent intermittent or cyclic delayed swelling (PIDS). This is defined as edema/swelling occurring in the filler area or vicinity and is often associated with events such as vaccinations, infection, or local trauma.

Classification of Soft Tissue Filler Complications by Onset of Adverse Event

Early reactions
Vascular infarction/soft tissue necrosis
Inflammatory reactions (acute/chronic)
Infection
Allergic reactions/hypersensitivity Injection-related events
Pain
Ecchymosis
Erythema
Bruising
Bleeding
Inappropriate positioning
Distant spread
Late reactions
Inflammatory reactions (acute/chronic)
Infection
Granuloma (typically chronic)
Differential diagnosis
Nodules
Dyspigmentation
Displacement of hyaluronic acid filler material

Consensus Recommendations for the Classification of Adverse Events Related to HA with Regard to Onset: Possible Diagnoses

Immediate onset (<24 hours)	Early onset (24–30 days)	Late onset (>30 days)
Vascular damage: embolization, arterial occlusion, etc. Allergic reaction Hematoma Overcorrection Ecchymosis Paresthesia	Vascular damage: ischemia, necrosis, telangiectasia Color changes: persistent, erythema, ecchymosis, Tyndall effect, post-inflammatory hyperpigmentation Systemic changes: infection, inflammation, paresthesia Scars: hypertrophic, atrophic Irregularities: overcorrection, infiltration (cellulite), nodules	Vascular damage: telangiectasia Color changes: post-inflammatory hyperpigmentation, persistent erythema Scars: atrophic, keloid Irregularities: Pile, nodules, late edema

THE CLINICAL SPECTRUM OF MANIFESTATIONS

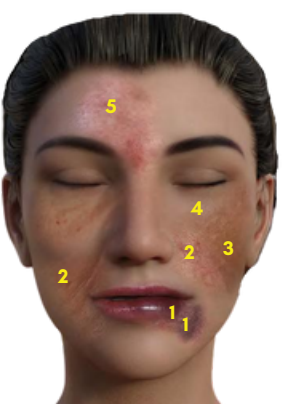
Certain complications are universal to HA filler treatment in all facial areas (skin discoloration, hematomas, swelling, edema, allergic manifestations, infections, vascular compromise, etc.), whereas others are more specific to individual facial zones and will be discussed in the individual modules.

It is important to evaluate patients holistically, exclude underlying systemic conditions (autoimmune disease, sarcoidosis, thyroid disease, etc.) and to approach complications systematically.

In addition to the presence of previous fillers, the differential diagnosis of periorbital edema

includes:

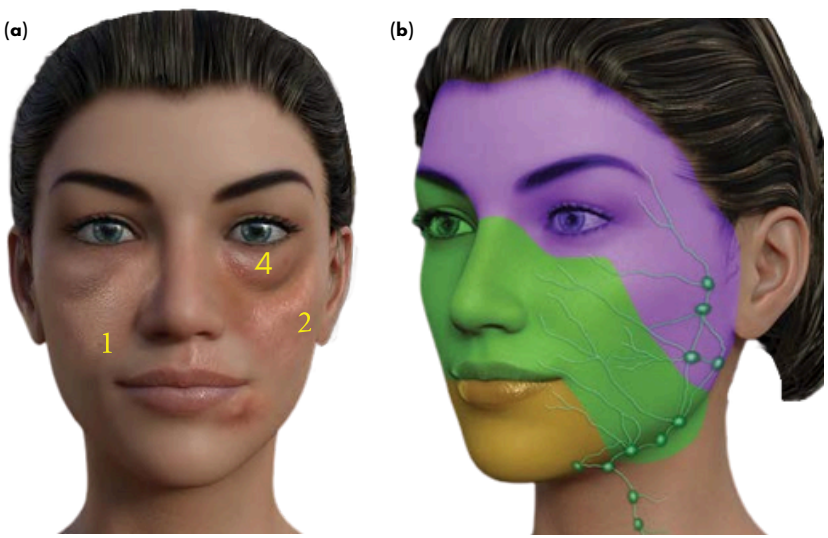
- Inflammatory/allergic conditions
- Renal disease
- Cardiac failure
- Thyroid disease
- Helminthic infections
- Autoimmune disease
  - LE
  - Dermatomyositis
- Previous filler



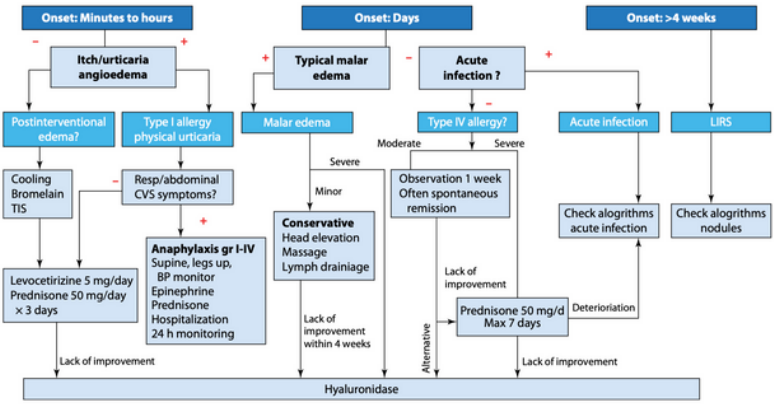
Causes of skin discoloration include: (1) hematoma/echymoses, (2) neovascularization, (3) hyperpigmentation, the (4) Tyndall effect and (5) ischaemia.

HYPERSENSITIVITY REACTIONS

Hypersensitivity reactions occur when the filler elicits an immune response. The response may be a type I hypersensitivity reaction, which typically has an early onset (within minutes to hours of injection), or a type IV reaction, which has a delayed onset (1-3 days up to several weeks after injection). The primary diagnostic symptoms of hypersensitivity reactions may include edema (localized or generalized), erythema, pruritus, pain or tenderness (pressure-related), rash, and induration. Delayed reactions may also present with various types of skin lesions, including painful erythematous nodules, abscesses, or cyclic urticarial swelling. In rare cases, acute hypersensitivity reactions may be severe, with cases of anaphylactic shock cited in the literature.



(a) Other than procedural swelling, causes of post-filler edema include: (1) malar edema, (2) late inflammatory response syndrome (LIRS), (3) late onset nodule, (4) PIDS and (b) schematic representation of facial lymphatic drainage.



Treatment of edema algorithms.

INFECTIONS

infections, both acute onset manifestations and delayed onset biofilms, are a risk for filler treatment in all facial areas, thus necessitating stringent aseptic technique. It is vital to remember that infections in adjacent areas (e.g., sinusitis, dental abscess) or even distant infections (e.g., gastrointestinal, urinary tract infections) may import microorganisms into the filler mass. When infections do occur, due diligence is required in diagnosing the causative agent. It is important to have a rationale for the source of infection, to adhere to local macrobiotic guidelines, and to exercise responsible antibiotic stewardship

Skin	Sinus	Dental	GI
Doxycycline Clindamycin Clarithromycin	Doxycycline Cephalexin Amoxicillin +] Clavulanic acid	Amoxicillin Clindamycin Cephalosporin	Metronidazole Clindamycin Ciprofloxacin
Azithromycin		Amoxicillin + Clavulanic acid	
Low-grade infections Doxycycline			

Antibiotic Choice by Area

Hypersensitivity reactions	
Early	**Delayed**
Check vital signs: Resuscitation measures: • Adrenalin • Intravenous access Fluids	Cold compresses H1-receptor antagonists H2-receptor antagonists Leukotriene synthesis inhibitors Oral corticosteroids Propranolol Ibuprofen

Algorithm for Early and Delayed Hypersensitivity Reactions

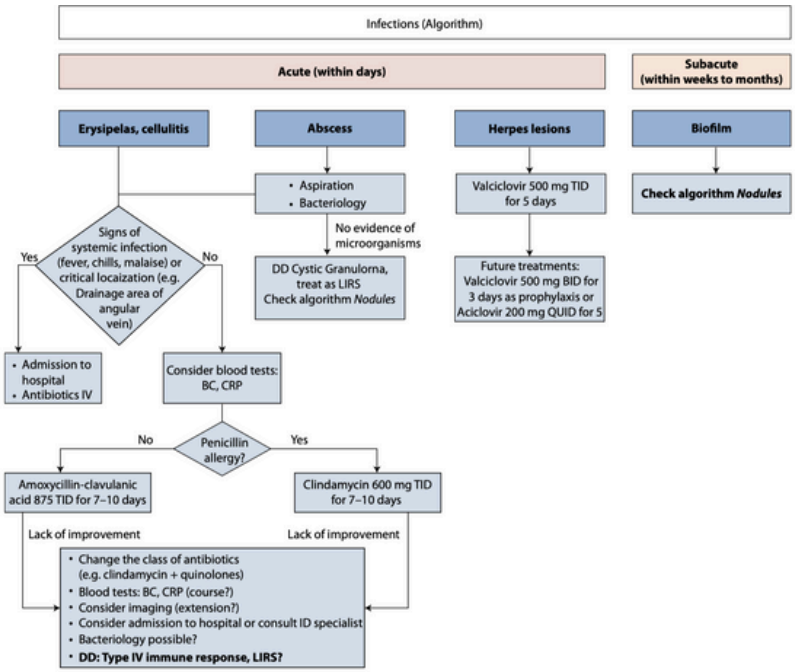
VASCULAR EVENTS

The initial symptoms of intravascular placement are often (but not always) pain and skin color change. It is important to realize that initial color change may be fleeting and remote from the injection point, manifesting in areas of watershed perfusion. This necessitates constant awareness of watershed perfusion areas such as the glabella, nasal tip, and upper lip. Initial pain may be masked by the addition of local anesthetics to filler formulations. Although usually immediate, onset has been reported to be delayed up to 24 hours. Red/bluish coloration is generally indicative of venous occlusion. Livedoid discoloration ("fish-net stocking pattern") should always raise the suspicion of vascular compromise.

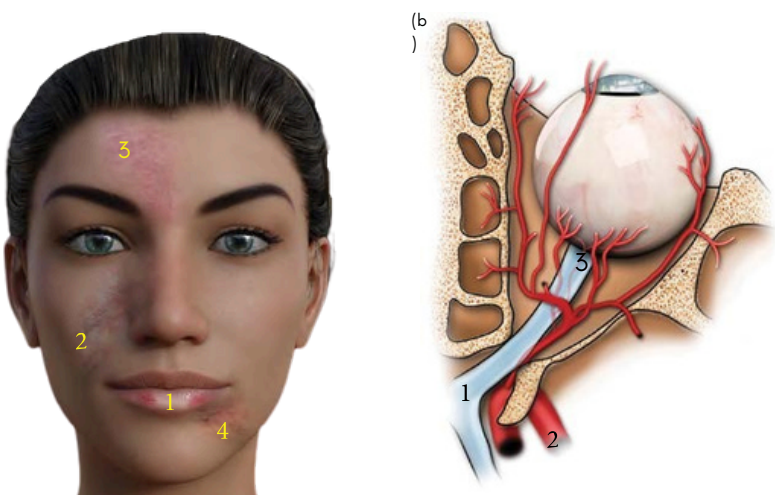
Later secondary diagnostic symptoms include blisters, pustules, tissue necrosis, and end-stage scarring.It is important to exclude vascular occlusion in all cases of pustulation occurring in the week after filler treatment, even if there is a history of herpes simplex, especially when occurring on a livedoid background.

TREATMENT

• Stop injecting immediately if there is any suspicion of inappropriate pain, skin blanching, or mottled discoloration.
• Apply warm compresses.
• Injection of hyaluronidase: High-dose pulsed hyaluronidase/HDPH (500-1000 IU) in repeated hourly doses for 3 hours forms the basis of the emergent therapy. The use of a large bore cannula is advisable for subdermal instillation of the hyaluronidase in order to avoid bruising, which would confound the visual feedback of improving skin color.
• Massage the affected area in the direction of smaller arterioles to increase the exposure of product to hyaluronidase.
• Give patient two tablets (650 mg) of aspirin to chew and swallow.
• Ultrasound, MRI, Doppler, arteriography, or phlebography may help in assessing filler placement and vascular damage, as well as aid in treatment planning.



Infections algorithm.



(a) Clinical examples of vascular occlusion: (1) Early blanching, (2+3) livedoid discoloration; (4) pustulation. (b) Vascular arterial supply of the orbit: (1) Ophthalmic nerve, (2) internal carotid artery, (3) central retinal artery.

	Vascular compromise (Di erential diagnosis)	
Diagnosis	**Peripheral ischemia with impending necrosis**	**Retinal ischemia**
Clinical presentation and time of occurrence	1. **Blanching phase** • Immediate onset, duration <1 min, sometimes painful 2. **Livedo reticularis phase (marbling)** • After a few minutes (seldom within a few hours), due to lack of oxygen resulting in venous dilation, virtually pathognomonic 3. **Blue-gray phase** • Tans of minutes to hours, due to sustained lack of oxygen. May appear more rapidly in the vermillion. 4. **Blister phase** • After 1-2 days, as a rst sign of skin necrosis 5. **Demarcation and ulceration phase** • After days to weeks, healing by secondary intention	• During Injection or immediately after injection: loss of vision, sometimes concurrent eye pain • Over the course sometimes ophthalmoparesis
Di erential diagnosis	• Vasoconstriction due to epinephrine containing local anesth. (blanching phase) • Hematoma (blue-grey phase) • Herpes simplex/herpes zoster lesions (blister phase)	• None
Therapy	• High dose hyaluronidase within a few minutes to hours • Warm compresses • Aspirin • Further therapeutic options (low evidence level): Low-molecular-weight heparin (LMWH), pentoxy llin, hyperbaric oxygen therapy (HBOT)	• Retrobulbar injection of hyaluronidase within 30 minutes • If retrobulbar injection cannot be performed by the injector: immediate referral to e (well-briefed) ophthalmologist • Afterwards hospitalization: possibly PGE1 therapy, consider other medical therapies (HBOT, LMWH, pentoxyfillin) • MRI of the brain to rule out any cerebral ischemia
Prophylaxis	• Use blunt cannulas (25G) in high-risk areas • Small boluses (max. 0.05 mL/bolus) in high-risk areas • Slow injection • Aspiration (needle) • Consider manual compression of the angular artery during injection • Risk factors: previous surgery, scars (including acne scars!), small-gauge needles/cannulas	

Vascular compromise differential diagnosis.

HYALURONIDASE

Hyaluronidase (Hase) may be used to reverse the effect of HA fillers (and also in some instances aid in reversing unwanted occlusion by non-HA fillers by partially degrading the dermal matrix).

Hyaluronidases are naturally occurring enzymes (endoglycosidases) that can depolymerize HA, leading to its degradation by hydrolyzing the disaccharides at hexosaminidic β-1 through β-4 linkages. This enzyme is often used off-label in aesthetic dermatology for treating undesirable effects of HA and indications include misplaced injections, overcorrection, the Tyndall effect, granulomas, inflammatory reactions, and vascular occlusion.

Hyaluronidase has

• Immediate effect
• A two-minute half-life
• Duration of action of 24-48 hours
• Efficacy lasting longer than it is short half-life due to continuing action despite partial degradation

Hyaluronidase initiates degradation of the crosslinks in the HA dermal filler, causing it to behave like endogenous HA, which has a half-life of 24–48 hours.

Because of individual rheological and crosslinking properties, different HA fillers demonstrate an individual sensitivity to degradation by Hase. Importantly, individual Hase products also vary in activity depending on their source and concentration. Insightful understanding of the variations in locally available Hase products, as well as their differential effect on the multiple HA fillers available, will maximize treatment efficiency should the need for reversal arise. This variation in response was clearly demonstrated in the recent publication by Casabona, who stated that there may be a three-fold difference in the amount of Hase required for degradation of a given volume of HA. Certain HA products denature more slowly than others, necessitating effective massage in order to break the HA bolus and expose it more efficiently to the Hase.

Dosages for All Indications Except Vascular Occlusion

The amount injected should be titrated to clinical effect, but actual dosages vary. A consensus opinion in the literature states that five to 10 units of hyaluronidase are required to break down 0.1 mL of 20 mg/mL HA, although there may be quite a range.

Treatment results may be assessed from 48 hours and repeated at intervals of 48 hours or longer, with the degree of further treatment depending on indication, risk: benefit ratio, treatment side effects, and patient or practitioner expectation.

Vascular compromise: High-dose pulsed hyaluronidase/HDPH (500–1000 IU) in repeated hourly doses for 3 hours forms the backbone of the emergent therapy. It is advisable to use a large bore cannula for subdermal placement of the hyaluronidase to avoid bruising, which would confound the visual feedback of improving skin color.

Although the timeframe for initiation of treatment for vascular compromise (excluding intraocular complications) is given to be as within 72 hours, there is potential benefit to initiating and/or persevering with treatment beyond this time limit should there have been treatment delay or suboptimal response.

It is important to realize that, although rare, allergic and anaphylactic reactions to hyaluronidase are possible. Human recombinant hyaluronidase carries the lowest risk of allergenicity but is not available in all countries.

PRACTICAL POINTS

• The use of high-frequency ultrasound may be useful in elucidating the presence, identity, and location of underlying filler for treatment with Hase.
• Massaging the treated area during and after injecting with Hase may help to optimize effect and to aid mechanical breakdown.
• When feasible, avoid treating with Hase when
○ Botulinum toxin treatments have been performed within the previous 48 hours.
○ There is an area of infected skin unless there is vascular occlusion, and the risks outweigh the benefits.

High-dose pulse hyaluronidase

Dosage	Standard dosage	500 IU per area
	Lip,nose,forehead	Act as multipliers
	Two areas	1,000 IU per hour
	Three areas	1,500 IU per area

Protocol
• Inject at least every 60-90 minutes until skin color has normalized and capillary refill time has normalized.
• Massage to increase embolus contact with the hyaluronidase by propelling the HA distally into thinner walled arterioles
• Aim to complete treatment within 72 hours of onset for complete resolution.
• Keep patient in clinic for observation and treatment until the capillary refill has improved (usually three sessions over 3 hours).

Do not apply nitroglycerin paste until the offending HA has been dissolved (day 2 or 3 of treatment) as dilation of adjacent, non-obstructed vascular pathways may lead to the propagation of the embolus toward the orbit by opening so-called "choke-anastomoses" acting as containment of further damage after a noxious event to tissue.

High-Dose Pulse Hyaluronidase (HDPH) Dosage and Protocol for Intravascular Events

INTRADERMAL TESTING

An intradermal test may be performed except when the indication is for vascular compromise, and a delay could result in further harm to the patient. Intradermal testing entails injecting 20 U of hyaluronidase in the forearm and observing the results after 30 minutes (a positive reaction at lower doses might not be recognized). Positive reactions are identified by a local wheal and itching, minor inflammation, and erythema.

A history of allergic reactions to wasp or bee stings may represent an increased risk of allergic reaction to Hase and constitute a relative contraindication, as the venom of stinging insects might contain hyaluronidase, and this mechanism might be the source of sensitization in affected individuals. Unless there is a past medical history of allergic reaction or anaphylaxis to hyaluronidase or insect bites, previous history of allergy seems unrelated to the safe administration of hyaluronidase and the risk:benefit ratio of treatment with Hase should always be considered.

Doses higher than 300 U may induce inflammation and eosinophil recruitment, thereby increasing the risk of type-1 hypersensitivity reactions such as urticaria and angioedema.

Observe at-risk patients for at least 2 hours after Hase treatment.

It is of vital importance that emergency resuscitation drugs are available and that expiry dates are current. It is highly advisable that dependable reminder systems (e.g., computer prompts) are in place for replacement of emergency drugs.

The use of high-frequency ultrasound may be useful in elucidating the presence, identity, and location of underlying fillers as well as in guiding aspiration and biopsies.

In this regard, we wish to encourage injectors to experiment with the effect of locally available Hase on the HA fillers regularly used in their markets.

LATE-ONSET ADVERSE EVENTS

Late-onset events may be either inflammatory or noninflammatory, presenting variously as edema, induration, nodules, cystic abscesses, or cyclic urticarial swelling. The degree of inflammatory change, manifesting as mild, moderate, severe, or frank fluctuation and infection, will dictate the preferred method of treatment.

ULTRASOUND DIAGNOSIS

Doppler ultrasound (duplex Doppler) is becoming an important tool in improving the safety of filler injections by:

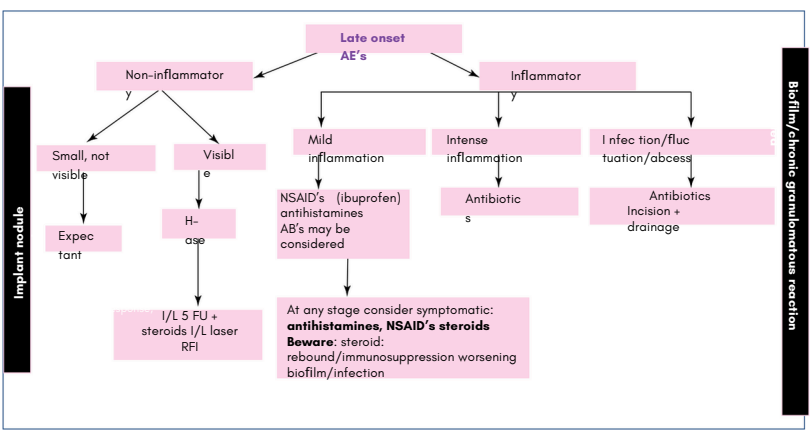
- Identifying the filler location and type in the event of a complication
- Locating important vascular structures before injection
- Identifying previous/unknown fillers in intendant treatment areas
- Guiding aspiration and biopsies.

The use of duplex Doppler devices allows gross visualization of blood flow as red and blue colors, thus enabling identification of larger vessels and blood flow in conjunction with underlying dermal structures. Duplex is proposed by some injectors to be utilized for identification of vascular structures in the proposed treatment areas, with the aim of minimizing intravascular placement of fillers.

However, it must be noted that no imaging modality can substitute for clinical experience and anatomical knowledge in terms of safety and efficacy.

In cases of diagnostic uncertainty, the following special investigations may be of value:

• CRP, an acute inflammation protein, usually associated with bacterial infections, immunohematological alterations, rheumatologic diseases.
• Full blood count and differential white blood cell count.
• Erythrocyte sedimentation rate (ESR).
• Acute-phase reactants, e.g., CRP, ESR, Procalcitonin. These appear to be the most sensitive markers for the presence of autoimmune/ inflammatory syndrome induced by adjuvants (ASIA) related to dermal filler use.



Differential diagnosis of late-onset adverse events.

• For suspected autoimmune disease,
○ ANA (antinuclear antibodies).
○ ENA: Antibodies to extractable nuclear antigens such as anti-SSA-Ro, anti-SSB-La (SLE, Sjogren Syndrome), anti-Jo1 (Polymyositis, Dermatomyositis), anti-Scl 70 (Progressive Systemic Sclerosis).
○ RF (rheumatoid factor).
○ ANCAs (anti-neutrophil cytoplasmic antibodies) detected in several autoimmune disorders, particularly with systemic vasculitis.
• Biopsy may identify both underlying foreign body granulomas and filler types.

• Angiotensin converting enzyme (s-ACE) and chest x-ray for suspected sarcoidosis.
• High frequency ultrasound (HFUS)
• MRI
• Biopsy with differential immunostaining.

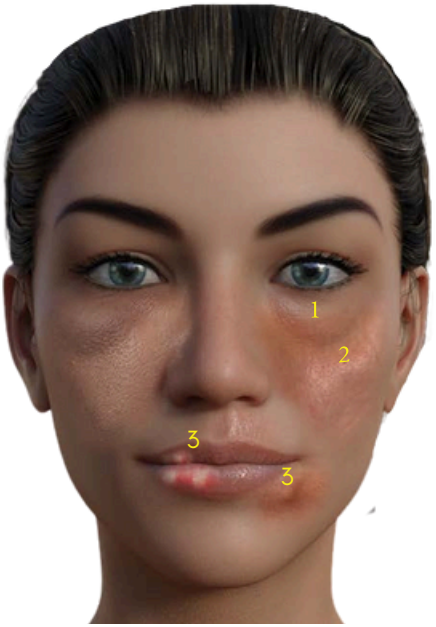
CONCLUSION

The field of aesthetic fillers is currently enduring an onslaught on safety. All injectors should work with an operative strategy aimed at reducing the risk of dermal filler complications through conscious awareness and careful pre-consideration of the possible confounding factors specific to each procedure. Algorithms for diagnosis and treatment are constantly evolving, and injectors should ensure that they are up to date with current best practice. It is vitally important that all injectors are aware of the increasing incidence of ophthalmic complications and are aligned with an ophthalmologic specialist adept at treating vascular occlusion as a complication of injectable fillers.

Recently, a combined technique of infrared (IR) facial heating and MRA (3D-TOF MOTSA) was proposed. Images may be acquired on a 1.5 or 3 Tesla (T) full-body MR system, using a dedicated head coil. Additionally, a flexible wrap-around surface coil may be mounted on top of the head coil in order to increase the signal reception from the facial vessels. Before the 3D TOF MRA examination, which is known to be flow dependent, the patient is positioned with closed eyes in front of an IR light source (300 W with an UV filter) for 10 minutes. This should induce vasodilatation and enhance vascular flow. At the same time, the patient is asked to stimulate their facial muscles by slowly moving the lips and forehead and switching between several facial expressions during the exposure time in order to further enhance the arteries (by vascular dilatation and increased flow speed due to muscle activation). An oblique coronal 3D-TOF MOTSA MRA sequence is acquired in an oblique coronal plane. During the acquisition, the patient is asked to remain completely still. A multislab technique is used to reduce the saturation effect of the inflowing blood signal. MIP images are made in a sagittal plane.

The MRA allows visualisation of the important facial arteries (facial [F]; angular [A]; superior [SL] and inferior labial [IL]; lateral nasal [LN]; dorsal nasal [DN]; supratrochlear [STr]; supraorbital [SO]; and superficial temporal [ST] artery)

Peri-orbital artefacts may hinder the visualization of the periorbital vessels and in patients with dental wires (after having braces), the perioral vessels may be hard to see 1.5T MRA also are more susceptible to motion artefacts due to the longer examination time.



Examples of late onset adverse events: (1) edema, including PIDS (2) induration and (3) nodules..

Although the reaction of the skin (red color) to the IR exposure will be visible, no adverse reactions due to the "IR enhancement" are noted, nor mentioned. The only (sometimes confusing) venous structure—running more posteriorly and laterally outside of the field of interest—is the large angular vein.

THE NOSE

INTRODUCTION TO THE NOSE IN AESTHETICS

Whilst the nose has primarily a breathing function, it also represents an aesthetically defining facial feature. The term non-surgical rhinoplasty is preferentially used for non-surgical aesthetic improvement with injectable fillers and toxins, whereas the term rhinoplasty is reserved for surgical nasal reshaping. Due to their reversibility, high G′ HA fillers are considered the safest material for non-surgical rhinoplasty. It is advisable that non-surgical rhinoplasty is performed only by highly experienced injectors with an intimate knowledge of injection anatomy and appropriate technique. Patient and product selection, accurate clinical assessment and correct injection techniques are of critical importance as the rich vascular network of the nose renders it a high risk area for severe complications.

The nose is centrofacially situated, surrounded by important anatomical structures and has a profound impact on facial appearance. Even minimal shadowing or asymmetry may lead to obvious aesthetic dysharmony.

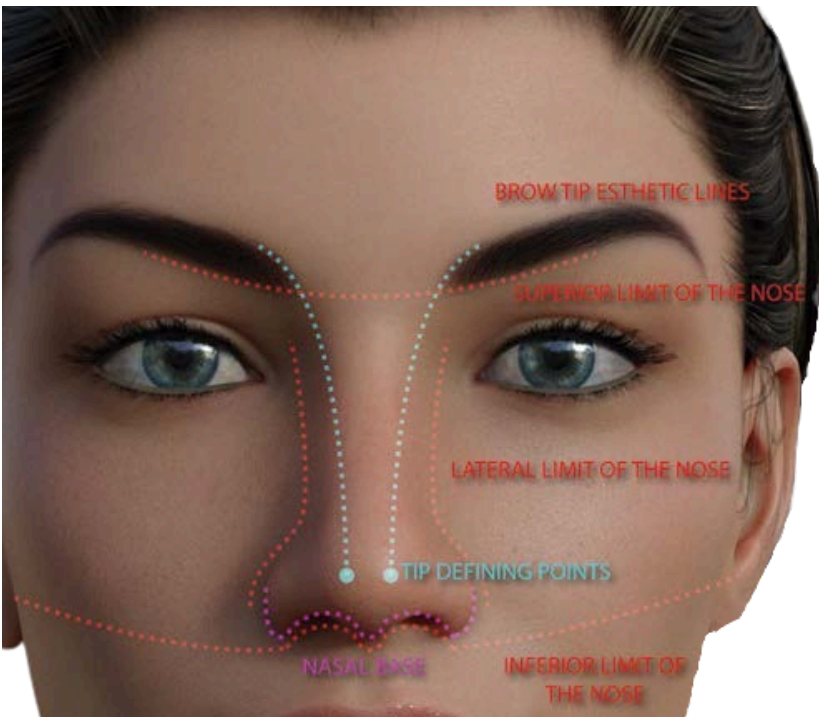
The glabella forms the upper nasal boundary, whilst the inferior border is formed by the nasal base and inferior nostril border. The lateral border is defined by an imaginary line between the ascending process of the upper maxilla and the nasal alar attachment. The lateral end of the aesthetic brow forms the tip of a line running from the nasal ala and past the lateral canthus.

Nasal boundaries: The nose lies between the two medial canthi. The glabella forms the upper limit and a horizontal line through the inferior nasal spine the lower limit.

AGING

Craniofacial growth continues after the age of 16-18 years, with continual nasal reshaping occurring thereafter. Changes in soft-tissue, muscle, skin and cartilage result in continual evolution as the underlying nasal structure (unlike bone and cartilage) continues to evolve over time. Insightful and detailed observation of specific, age-related changes provides the necessary clinical information for optimal non-surgical correction techniques.

Nasal measurements have proven to be significantly affected by age, with an increase documented in volume, area, and linear distance. In the majority of study groups, elderly subjects tend to have larger noses than younger members of the same gender and ethnicity. The nasolabial angle—the angle between the lower border of the nose and the subnasal line that connects to the upper lip border—has consistently been found to decrease, implying that the nose tends to droop with age.



Brow tip defining lines and tip defining points.

The nasal pyramid is the most influential aesthetic aspect of the middle third of the face. Although the aesthetic impact of the nose in profile view has been widely documented, the analysis of nasal proportions during the growth phase is less well documented than in mature populations, making assessment of the younger patient more complex.

Nasal soft tissue growth is greater, and occurs earlier, in adolescent girls than in age-matched boys.

Nasal height increases the most, doubling from birth to the age of 20. Overall, males have larger noses than females, but noses seem to grow more quickly in girls when measurements are compared over a lifetime. In 3-4 year old females, the volume of the average nose is approximately 42% of early adult size (at 18-30 years of age); for males, the average volume has been found to be approximately 36%. By the age of 30, nasal growth slows down considerably. Between 50 and 60 years of age, nasal volume will typically increase by a further 29% in men and 18% in women.

With aging, there is soft tissue deflation and less upper maxillary support, with resultant loss of nasal tip projection.

SKIN

Bone and cartilage form the supporting framework of the nose. From the glabella to the bridge, to the tip, the nasal skin is anatomically considered in vertical thirds

• In the upper third, the skin is thick and relatively distensible (flexible and mobile). It then tapers, becoming tightly adherent to the osseocartilaginous framework, thinning towards the dorsal nasal bridge.

• The middle third overlying the nasal bridge (middorsal section) has the thinnest, most adherent and least distensible, skin.

• The skin of the lower third is of equal thickness to the upper nose, due to more sebaceous glands, especially at the nasal tip.

FAT

The nose consists of a framework of skin, cartilage, and bone, with six distinguishable layers: skin, superficial fatty layer, fibromuscular layer (the superficial muscolar aponeurotic system, SMAS), deep fatty layer, periosteum-perichondrium, and bone-cartilage. Nasal subcutaneous tissue exists in discrete compartments that are determined by the underlying perforator blood supply.

• The skin is thicker in the radix area, becomes extremely thin in the mid-vault region, and thickens in the supra-tip area

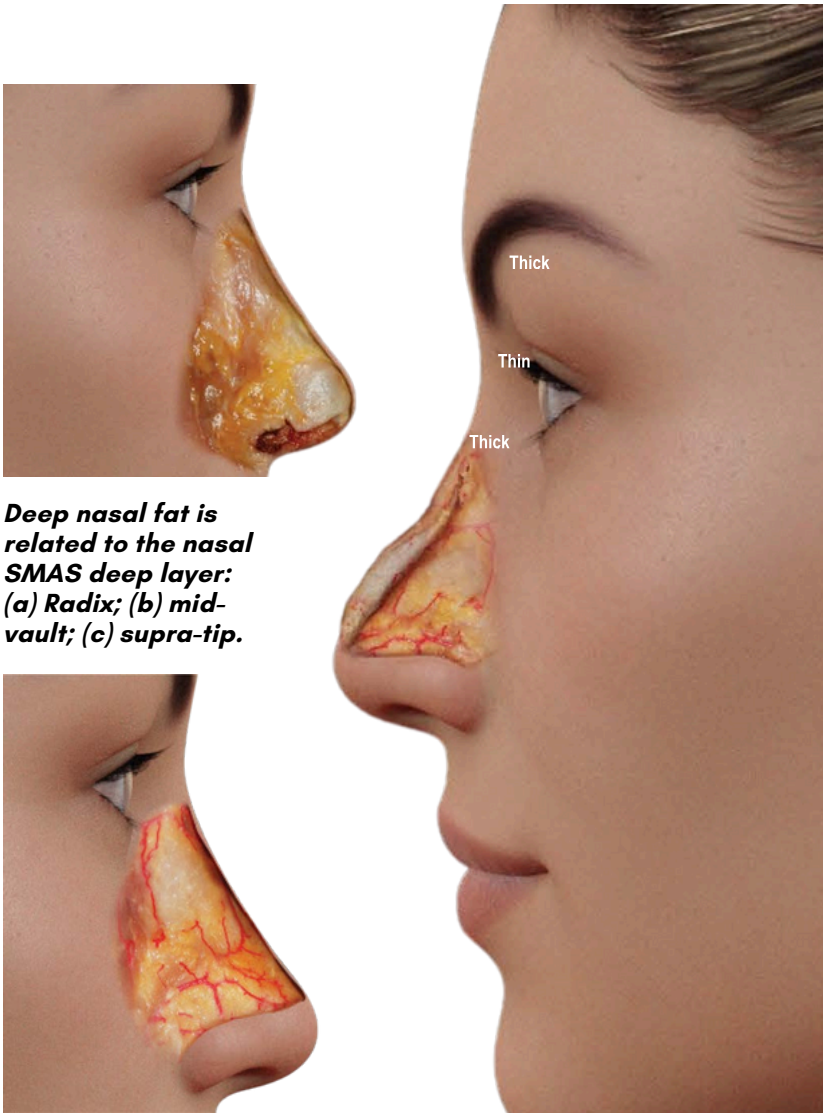
• Immediately beneath the skin, there is a superficial fatty layer comprising predominantly adipose tissue containing vertical fibers and septae extending from the skin to the underlying SMAS.

• The distinct nasal SMAS is in continuation with the facial SMAS.

• Subcutaneous fat is concentrated in the glabella, lateral nasal wall, tip, and supra-tip areas

• Distribution of the sub-SMAS fat is similar to that of the superficial facial fat, with an additional layer of fat beneath the transverse nasalis muscle and an interdomal fat pad confirmed in cadaver studies .

Skin on the nose is shown in elevation. The dorsal nasal skin is thinner, making every mistake visible.



Deep nasal fat is related to the nasal SMAS deep layer: (a) Radix; (b) mid-vault; (c) supra-tip.

Nasal fat is very thin. Note the superficial vessels in the lateral aspect.

MUSCLES

The nasal SMAS is a continuation of the facial SMAS; this layer ensheathes the muscles and is highly vascular. All the nasal muscles are innervated by cranial nerve VII.

Procerus

The procerus is the most cephalic muscle of the nose. It arises from the glabellar area, extends caudally in a vertical fashion, and joins with the wing-shaped transverse nasalis muscle covering the caudal portion of the nasal bones. The main function of the procerus is depression of the eyebrows, which can create horizontal wrinkles over the cephalic portion of the nose in aging patients.

Nasalis

The nasalis muscle has two components: The transverse nasalis or compressor nasi and the pars alaris.

The transverse part of the muscle spans the dorsum of the nose, covering the upper lateral cartilages. This muscle, also called pars transversa, arises from the lateral cephalic portion of the subpyriform crescent. The pars transversa joins with the procerus muscle and the opposite muscle in the midline to form the nasalis-procerus aponeurosis which compresses and elongates the nose, contracts the nostrils, and narrows the vestibules.

The second component of the nasalis muscle, the pars alaris (alar nasalis), arises from the crescent origin of the maxilla and is more lateral and slightly caudal to the bony origin of the depressor septi nasi muscle. The alar portion partially covers the lateral crus of the lower lateral cartilages and assists in dilatation of the nares. Damage to this muscle may result in collapse of the external nasal valve. In ethnic noses, the pars alaris is stronger and far more developed.

Depressor Alae or Myrtiforme

The depressor alae muscle originates from the border of the pyriform crest and then rises vertically, in a fan-like pattern, to the ala, acting as a depressor and constrictor of the nostrils.

Levator Labii Superior Alaeque Nasi (LLSAN)

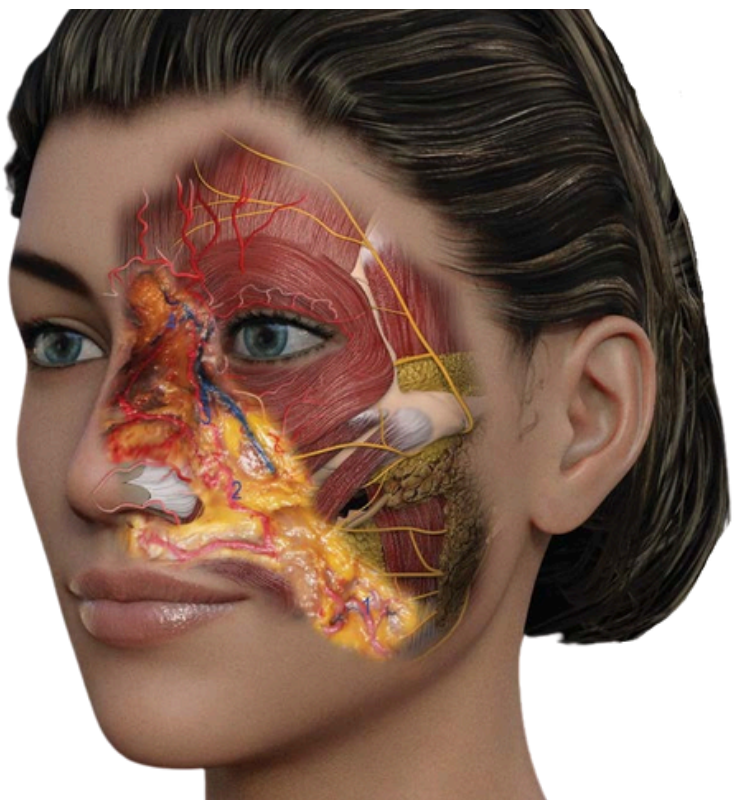
LLSAN plays an important functional role. It extends lateral to the nose in a cephalocaudal direction and has fibers that are attached to the nostril, thus contributing to dilatation of the nares. Paralysis of these muscles will contribute to collapse of the external valve.

Depressor Septi Nasi

Depressor septi nasi arises from the maxilla just below the nasal spine, sometimes fuses with fibers of the orbicularis oris muscle, extends along the columellar base, and attaches to the foot-plate. Occasionally, fibers of this muscle extend to the middle genu. Some authors believe that these muscle fibers extend to the membranous septum. The depressor septi nasi depresses the nasal tip on animation and alters air turbulence. Additionally, it is of aesthetic importance as its contraction narrows the labio-columellar angle. Release of this muscle not only eliminates the depressor effect on the tip but may also cause slight ptosis of the upper lip, which may or may not be beneficial, depending on the visibility of the patient's incisor teeth.

VASCULARIZATION

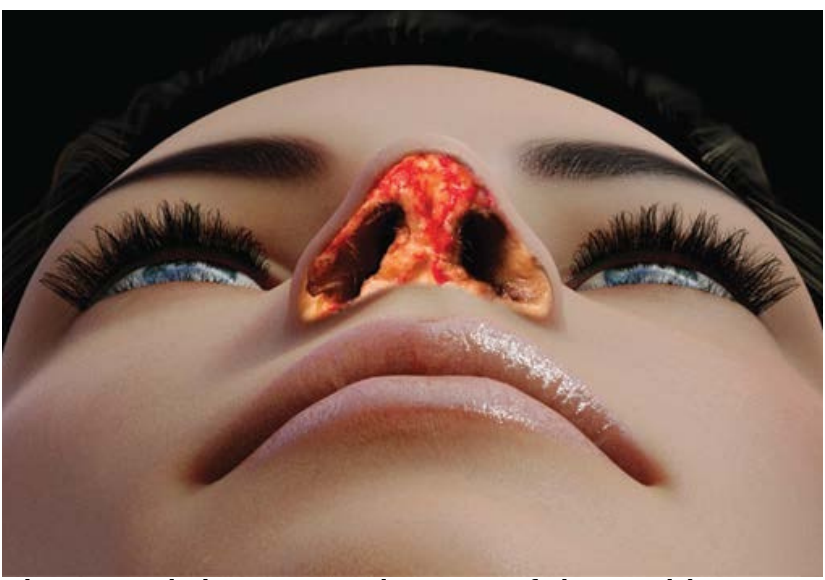
The arterial blood-vessel supply to the nose is two-fold: There is internal vascularization through branches of the internal carotid artery a branch of the anterior ethmoidal artery, and a branch of the posterior ethmoid artery, which derive from the ophthalmic artery; and branches of the external carotid artery the sphenopalatine artery, and the greater palatine artery coming from the internal maxillary artery, the superior labial artery, and the angular artery coming from the facial artery. The latter becomes the angular artery in the proximity of the nasal ala and then courses over the superomedial aspect of the nose go become the lateral nasal artery. The dorsal nose is supplied by branches of the internal maxillary artery (infraorbital) and the ophthalmic arteries, deriving from the internal carotid artery. This anastomotic area between the internal and external carotid circulations is very important as it may lead to intravascular embolization of injected fillers



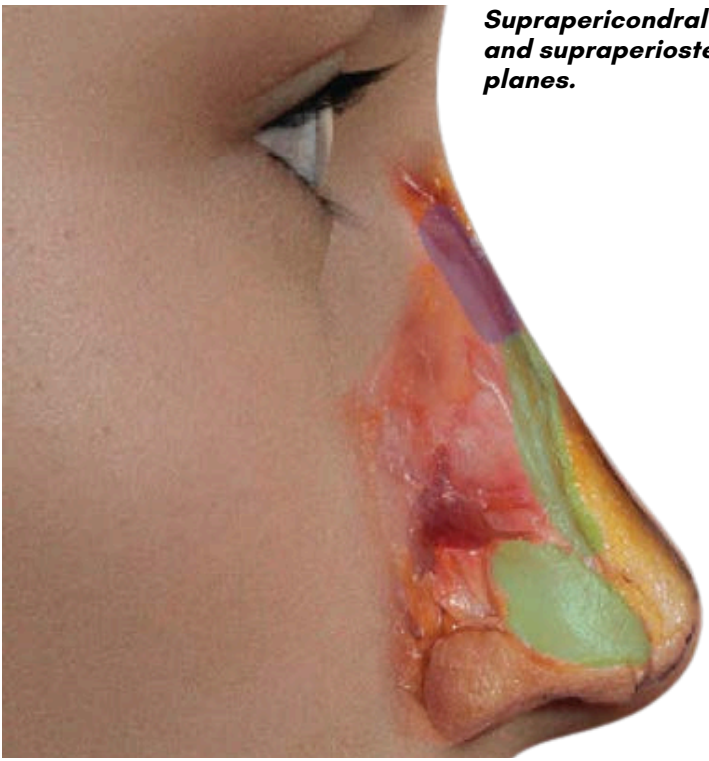
(1) Facial artery; (2) angular artery; (3) lateral nasal artery; (4) anastomosis with the ophthalmic artery (internal carotid system).

Blue arrow: Danger area where the lateral nasal artery (external carotid system) anastomoses with the ophthalmic artery (internal carotid system).

The superior labial and the angular artery are the main branches that respectively form the columellar branches and the lateral nasal branches. The lateral nasal vessels are 2-3 mm above the alar groove and, together with the columellar artery, arise deep at the nasal base to end at the tip in the subdermal plexus; both supply the tip of the nose. Internally, the lateral nasal wall is supplied by the sphenopalatine artery (from behind and below) and by the anterior and posterior ethmoid arteries (from above and behind). The nasal septum also is supplied by the sphenopalatine, anterior and posterior ethmoid arteries, with additional contribution by the superior labial and greater palatine arteries. These three vascular supplies to the internal nose converge in the Kiesselbach plexus (Little's area), a region in the antero-inferior third of the nasal septum. Furthermore, the venous supply of the nose generally follows the arterial pattern of nasal vascularization. The nasal veins are biologically significant because they have no valves, and communicate directly with the cavernous sinus. This may potentially cause intracranial spread of bacterial infections originating from the nasal region. Intra-arterial injections in this area may lead to skin necrosis and even blindness. Danger zones exist particularly in the areas where the internal and external carotid systems communicate (angular artery and columellar artery). Due to great anatomic variability, the midline position as a safe reference may sometimes be unsafe for injection. Therefore, we emphasize the principle of "staying deep," just above periosteum and perichondrium.



Skin removal showing vascularization of the nasal base; note the columellar arteries.



Suprapericondral (green) and supraperiosteal (blue) planes.

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INNERVATION

Nasal sensory innervation is derived from the supraorbital and infraorbital nerves; motor innervation is via the buccal branch of the facial nerve.

BONE

The skeletal component of the nose consists of bone and cartilage. The paired nasal bones and the frontal process of the maxilla form the lateral aspect whilst the lateral surfaces of the upper two-thirds join in the midline at the nasal dorsum. Supero-laterally the paired nasal bones connect to the lacrimal bones, and infero-laterally they attach to the ascending processes of the maxilla. Postero-superiorly, the bony nasal septum is composed of the perpendicular plate of the ethmoidal bone. The vomer lies posteroinferiorly and partially forms the choanal opening into the nasopharynx.

The nasal floor is formed by the pre-maxillary and the palatine bones, which also form the roof of the mouth.

The bony nasal vault comprises the paired nasal bones and the ascending frontal process of the maxilla. This part of the nose is pyramidal in shape, the narrowest portion being at the intercanthal line. Theaverage length of the nasal bone is 25 mm; although there may be both individual and significant ethnic variation (African American noses often have short nasal bones). Laterally the nasal bones join with the frontal process of the maxilla.

The circle created with the nasal spine, the thin portion of the frontal process of the maxilla, and the thin caudal border of the nasal bones is called the pyriform aperture. The nasal bones fuse with the superior edge of the perpendicular plate of the ethmoid bone cephalad to the intercanthal line. The confluence of cartilaginous nasal septum, ethmoid bone, and nasal bone is called the keystone area.

CARTLIDGE

The cartilaginous nasal frame consists of a pair of upper and lower lateral cartilages and the nasal septum.

The upper lateral cartilages are paired rectangular cartilages that support the lateral nasal walls.

These cartilages join the septum in the midline, although the fusion between the upper lateral cartilages and the septum occurs in a manner which almost creates a single unit cephalically. The lateral border of the upper lateral cartilages frequently terminates at the level of the lateral nasal bone suture line. This leaves a space between the bone and upper lateral cartilage, which is termed the external lateral triangle. It is surrounded by the caudal border of the upper lateral cartilages cephalically, the frontal process of the maxilla laterally, and the cephalic border of the lower lateral cartilage caudally. The cephalic portion of the upper lateral cartilage is overlapped by the nasal bone. The amount of overlap is highly variable and can range from 2 to 11 mm.

The lower lateral cartilages have four components: the medial crus, middle crus, dome, and lateral crus.

Medial Crus: The medial crus has two distinct segments: the footplate and the columella. The footplate varies in size and in the degree of lateral angulation. This angulation of the footplate governs the width of the base of the columella. The columellar segment of the medial crus varies in length and width; the longer the columellar portion, the longer the nostril and thus the potential for a more projected nasal tip.

The circle created with the nasal spine, the thin portion of the fron Cephalad to this portion of the medial crura is the membranous septum, which is composed of two layers of soft tissues encasing some ibrous bands named septocolumellar ligaments.

Middle Crus: This part of the lower lateral cartilage extends between the medial crus and the domes and its length and width largely control the configuration of the infratip lobule.

Dome: The domal segment is the narrowest and thinnest portion of the lower lateral cartilage; yet, it is the most important in relation to the tip shape. There is tremendous variation in its shape; on rare occasions, it has a convolution that, when present, invariably results in bulbosity of the tip.

The medial and middle crura are tightly bound together by fibrous bands. The most anterior one is called the interdomal ligament.

Additionally, there are fibrous bands more anteriorly binding the domes to each other and the overlying dermis; these are called the Pitanguy ligament. There are additional fibrous bands at the level of the footplates and between the upper and lower lateral cartilages.

Lateral Crus: This portion of the nasal lobule is the largestcomponent. It is narrow anteriorly but becomes wider in the mid-portion and narrows again laterally. The lateral crus of the lower lateral cartilage (LLC) is usually in contact with the first chain of the accessory cartilages that abut the pyriform aperture. Medially, the lateral crus is continuous with the domal segment. The anterior portion of this cartilage can curve in a variety of directions and controls the convexity of the ala. It also provides support to the anterior half of the alar rim. However, posteriorly, it diverges and does not have much contribution to the ala, yet does contribute to the function of the external valve. Generally, this cartilage is oriented at a 45° angle to the vertical facial plane. The curled junction of the cephalic edge of the lateral crus and the caudal edge of the upper lateral cartilage is referred to as the scroll area.

The magnitude of curling can vary from patient to patient and is sometimes significant enough to cause external visibility and fullness in this area. The lower lateral cartilage is commonly short and weak in non-Caucasian noses.

The accessory cartilages are a series of small cartilages situated on the nasal ala close to the tail of the alar cartilages.

The nasal septum is situated on the central part of the nose and its connected with its inferior surface.

Nasal bone (1) and cartilages (2) are visible. Green dot: the keystone area.

ELEVATION OF THE NASAL TIP

The target muscle here is the depressor septi nasi. The bony origin is from the maxilla at the level of the canine tooth, and the skin insertions are along the columella up to the nasal tip. Patients with a low nasal tip will not benefit from this treatment if the defect is caused by a long inferior projection of the nasal septum. Local examination will quickly clarify this. A single 2-3 U subdermal injection at the base of the columella will gently elevate the nasal tip when the indication is correct. Further improvement may be achieved by injecting hyaluronic acid in a deeper plane to open the nasolabial angle to some extent, and in selected cases, this combination is quite successful.

BUNNY LINES

Bunny lines are generated by the nasal part of the transverse nasalis muscle. Although they may be visible at rest, dynamic assessment is essential to appreciate their precise extent. Treatment is quite straightforward and a single 2-3 U aliquot in the middle of the bunny-line area usually solves of the problem. Avoid injecting laterally to prevent involvement of the levator labii superioris alaeque nasi, as this would lead to alteration of the smile and lengthening of the upper lip.

BOTULINUM TOXINS: BUNNY LINES

BUNNY LINES

Target Muscle and Anatomy

A bunny line is a horizontal wrinkle that appears on both lateral and dorsal aspects of the nose when smiling and frowning. The nasalis m. produces horizontal lines on the middle of nasal dorsum; however, the levator labii superioris alaeque nasi m. (LLSAN) and the additional medial muscular band of the orbicularis oculi muscle produce bunny lines, which form on the sides of the nose. In Asians, this muscular band was observed in 66 %.

Injection Points and Methods

2U is injected into three points: the nasal dorsum and both the lateral sides. In the strict sense, bunny lines cannot be completely removed because the muscle fi bers of the LLSAN cannot be entirely paralyzed.

Injection points of botulinum toxin for bunny lines

BOTULINUM TOXINS PLUNGED TIP NOSE

PLUNGED TIP OF THE NOSE

Target Muscle and Anatomy

People with well-developed nasalis m. or depressor septi nasi, which draws the tip of the nose downward, usually present a drooping nasal tip. A toxin injection could be useful in weakening these muscles by allowing them to lift the nasal tip a little. If the patients present a drooping nasal tip when smiling, it is effective to inject into the LLSAN as well as the depressor septi.

The depressor septi located deeply on the upper lip arises from the incisive fossa of the maxilla and inserts into the movable part of nasal septum. The depressor septi drops the tip of the nose when contracted; therefore, the tip of the nose may be flattened, and the nostril may appear larger when smiling.

Injection Points and Methods

4U is injected into the subnasale inferior to the columella and the ala of the nose at both sides with a total of 12 U doses. Additionally, 2 U is injected into the LLSAN at each side only if the patients present a drooping nasal tip when smiling.

Injection points of botulinum toxin for plunged tip of the nose (a, b)

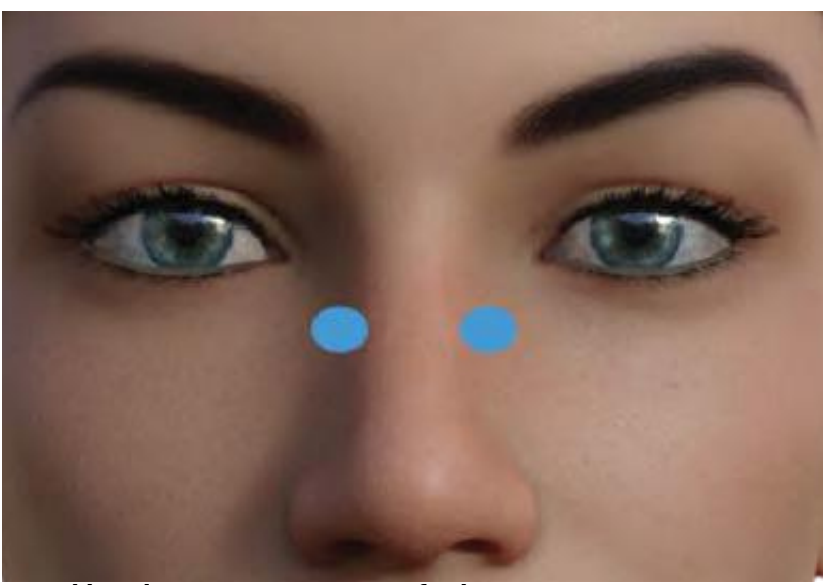
HOW I DO IT: BOTULINUM TOXIN

HOW I DO IT: BOTULINUM TOXIN

Bunny Lines

Bunny lines are generated by the transverse portion of the nasalis muscle in synergy with the levator labii superioris alaeque nasi muscle. The dose per injection site is 2 U per side in both men and women.

The injection is performed at a 45° angle to the skin with a low-to-high direction. A single injection is performed on each side of the nose, in the center of the affected area. Needle penetration is limited to the tip only, as the muscle lies immediately under the skin. Care should be taken not to inject too laterally in order to avoid functional interference with the levator labii superioris alaeque nasi muscle, which would produce an undesirable elongation of the upper lip.



Nasal botulinum toxin injections for bunny

Often, when treating the bunny lines, procerus treatment is performed concomitantly to manage the horizontal lines at the nasal root.

Sagging Nasal Tip

The target muscle for treatment of a sagging nasal tip is the depressor septi nasi. When hypertonic, the depressor septi nasi lowers the tip of the nose, which is usually more visible upon smiling. To treat a sagging nasal tip, a single injection with a 2–4 U dose is recommended in both men and women.

Treatment is performed by inserting one-quarter of a 30G × 1/2″ needle, at the base of the columella, pointing toward the nasal spine. The treatment produces upward rotation of the nasal tip. However, clinical assessment is crucial for correct patient selection as treatment of the sagging nasal tip should be avoided in patients with a long upper lip. A long caudal septum is also a contraindication to toxin treatment as it prevents the desired tip rotation.

NOSE FILLERS

NOSE FILLERS

The nose, being located at the center of the face, plays an important aesthetic role in creating balance between the eye and the mouth. For Asians who have a relatively lower nose bridge, filler injections rather than the rhinoplasty may be a simple and effective method for nose augmentation. Although fillers have the advantages of being less invasive than surgery, fillers lose their volume over time by naturally breaking down, and periodic injections are needed to maintain its clinical outcome. Regarding safety, side effects such as contracture of the nose that may be entailed after the surgery do not pertain in filler injection procedure; however, cosmetic effects of filler treatments are limited in that filler injections cannot make the blunt nose tip sharper or reduce big nose, remove hump, and narrow a wide nose. Nonetheless, among Asian populations with high demand for nose augmentations, filler injections are often a suitable replacement for invasive surgical procedures.

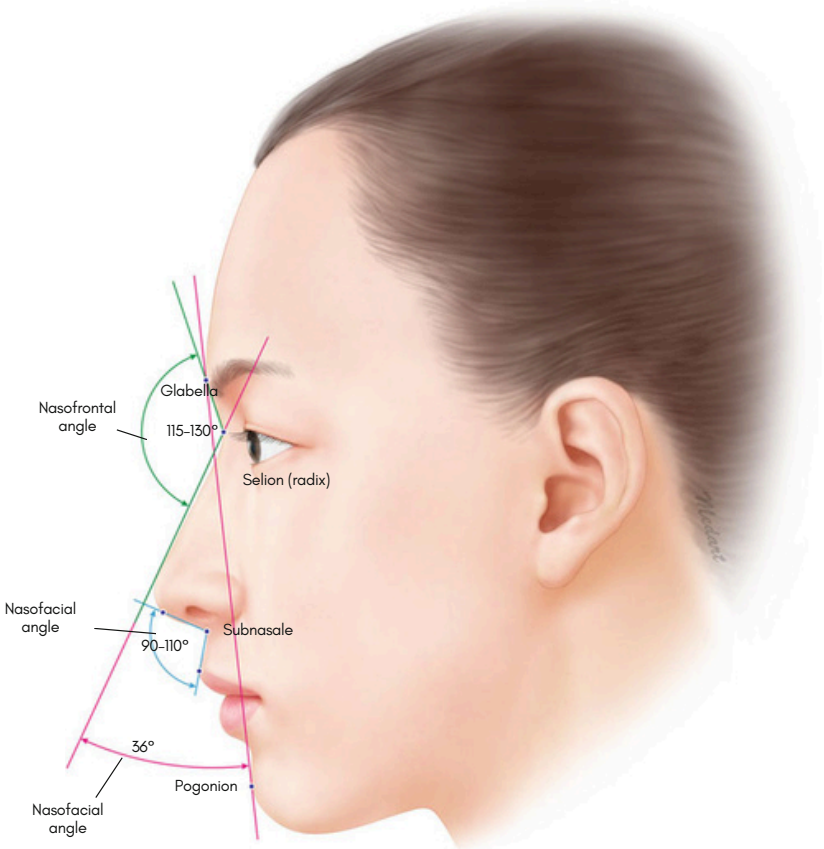
Since the criteria of aesthetic appeal vary for different populations, it is difficult to state an ideal angle for the nose that is the most appealing. However, it helps to keep in mind a range of angles for the nose that gives a person an attractive look: nasofrontal angle, 115–130°; nasofacial angle, 35–40°; and nasolabial angle, 90–110°.

Asians tend to have thicker skin and an abundant subcutaneous tissue than Caucasians. From a cosmetic perspective, the presence of an abundant soft tissue of the nose helps to minimize the undulating appearance that is more prone to occur in Caucasians. When treating special cases such as a hooked nose, the physician should be aware that the rhinion is the thinnest area of the dorsum of the nose.

From a clinical perspective, a thick subcutaneous tissue in Asian populations is a point of greater caution because an improper injection may lead to improper intravascular injections of filler products within the subcutaneous layer.

The soft tissue of the nose consists of the skin, the superficial fatty layer, the fibromuscular layer, the deep fatty layer, and the periosteum or the perichondrium.

The fibromuscular layer is located between the superfi cial and deep fatty layers. Muscles near the nose are interconnected by SMAS (superficial musculoaponeurotic system) enclosing the face.

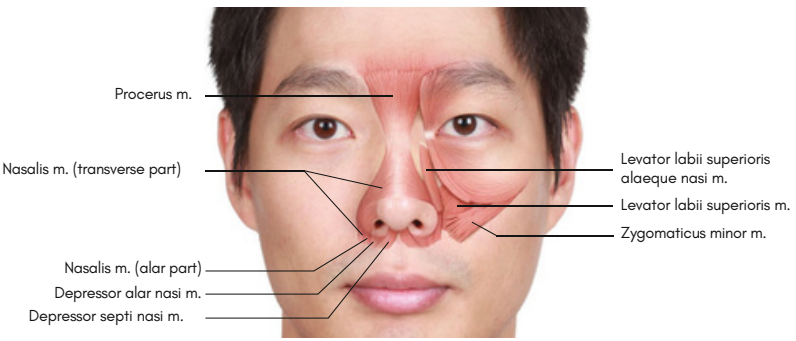


Ideal facial angles

It is known that the main arteries of the dorsum of the nose are located at the level of the superficial or deep fatty layers. However, at the lower portion of the dorsum of the nose, the dorsal nasal arterial branches are located adjacent the fibromuscular layer and the deep fatty layer.

On the other hand, at the upper portion of the dorsum of the nose (bony nose), the dorsal nasal a. run at the level of the superficial fatty layer just above the fibromuscular layer.

Paranasal muscles include (1) the procerus m., (2) the nasalis m., (3) the depressor septi nasi muscle, and (4) dilator naris vestibularis and anterioris muscles of the nasal ala. In addition to the muscles stated previously, there are other muscles attached to the nasal ala.



The attachment of the levator labii superioris muscle to the nasal ala in the posterior aspect. Some deeper muscle fi bers of the levator labii superioris

1. Procerus muscle: The procerus m. is a small muscle that arises from the nasal bone and the lateral cartilage of the nose and attaches to the skin at the radix and glabella. At this point, the muscle fibers of the procerus m. intermingle with muscle fibers of the frontalis. The procerus m. is responsible for pulling on the medial portion of the eyebrow and forming a transverse wrinkle of the glabella.

2. Nasalis muscle: The nasalis m. is formed by the transverse part and the alar part. The transverse part of the nasalis m. in a triangular shape originates from the canine fossa of the maxilla and inserts into the lateral cartilage of the nose. The alar part is a square-shaped muscle that arises from the maxilla above the maxillary lateral incisor and inserts into the lower portion of the alar cartilage. The transverse and alar parts are responsible for the narrowing of the nostrils by the contraction of the nasal aperture and widening of the nostrils, respectively.

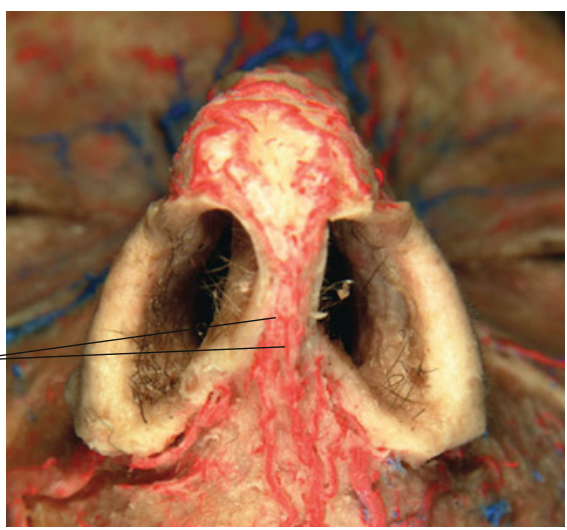
3. Depressor septi nasi muscle: The depressor septi nasi m. is located deep within the lip. It originated from the incisive fossa of the max- illa and inserts into the mobile portion of nasal septum and intermingles with the deep muscle fibers of the orbicularis oris muscle. This muscle acts for pulling down the tip of the nose as to enlarge the nostrils.

4. The dilator naris vestibularis muscle is located between the external and vestibular skin of the alar lobule. Its muscle fibers radiate along the dome-shaped nasal vestibule.

The dilator naris anterior muscle originates from the frontal surfaces of the lateral half of the lateral crus and the accessory alar cartilage adjacent to the lateral crus

The dilator naris anterior muscle originates from the frontal surfaces of the lateral half of the lateral crus and the accessory alar cartilage adjacent to the lateral crus

In addition to the muscles stated above, 90% of the levator labii superioris alaeque nasi m. and 28 % of the zygomaticus minor m. are attached to the nasal ala.



Columellar branches from the superior labial artery (a)and coronal section of the columella (middle of the columella (×10)

The filler treatment must proceed with a full awareness of the arteries and veins distributing the nose. Although rare, there are cases when intravascular injection leads to problems caused by filler products in surrounding tissues.

Symptoms that follow can be divided into the intravascular embolism and the extravascular compression.

The use of a cannula is advised to reduce chances of intravascular injection.

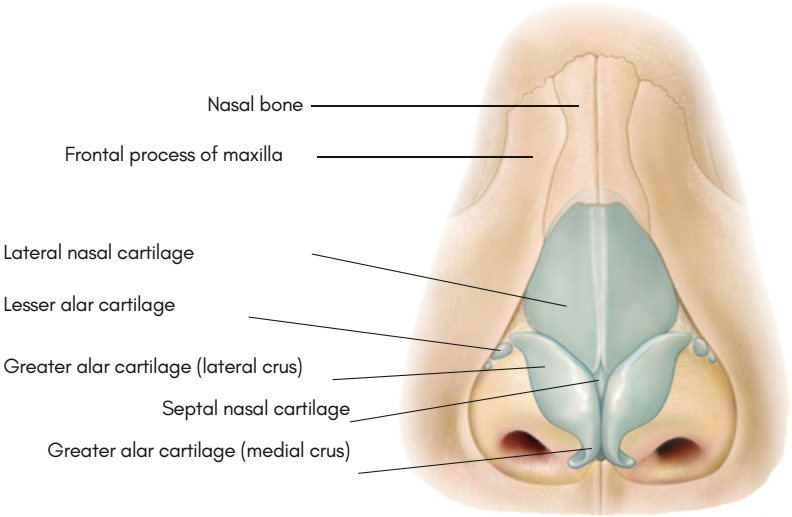
The major arteries of the nose consist of the lateral nasal a. arising from the facial a. and the dorsal nasal a. arising from the ophthalmic artery. The arteriovenous anastomosis that results from the branches of the lateral nasal a. and the dorsal nasal a. is a characteristic anatomical feature of the external nose. Furthermore, the columella branches that arise from the superior labial a. distribute at the tip of the nose. In addition to the lateral nasal a., the inferior alar a. traverses below the nasal ala. Overall, the lateral nasal and dorsal nasal arteries are largely responsible for arterial blood supply to the tip of the nose. Branches of the dorsal nasal and the angular arteries distribute at the dorsum of the nose.

The dorsal nasal a. is slender and, as stated before, run at the level of the deep fatty layer at the lower dorsum of the nose. However, at the upper portion of the dorsum of the nose, the dorsal nasal a. tends to be located at the superficial fatty layer of the procerus muscle. The dorsal nasal a. originates from the ophthalmic a.; hence, it tends to run along the sides of the dorsum of the nose. Small communicating branches from bilateral dorsal nasal a. are observed at the dorsum of the nose (78% of the cases). Especially, it is memorable that the cases in which the dorsal nasal a. from one side crosses the midline of the nose and mainly supplies the opposite side can be found (21.6 % of Asian cases).

Furthermore, a case that a relatively thick tortuous arterial branch from the labial a. entered the columella via the columello- ophthalmic a. emerges from the upper medial canthal area and runs along the lateral aspect of the dorsum of the nose and then communicates with the lateral nasal a. can also be seen. In this case, injecting fillers using a needle into a point deviating lateral to the radix may be highly dangerous; therefore, aspi- ration should be performed prior to the injection.

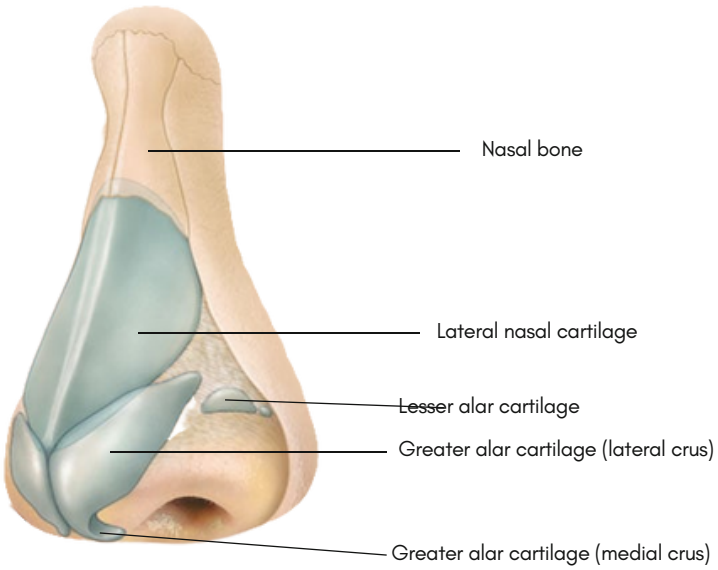
The columellar branches from the superior labial a. entered the columella via the columello-labial junction and distribute to the base of the nose. These branches proceeded to the medial crus of the lower lateral cartilage at the midline, at the basal and posterior portions of the septum.

a

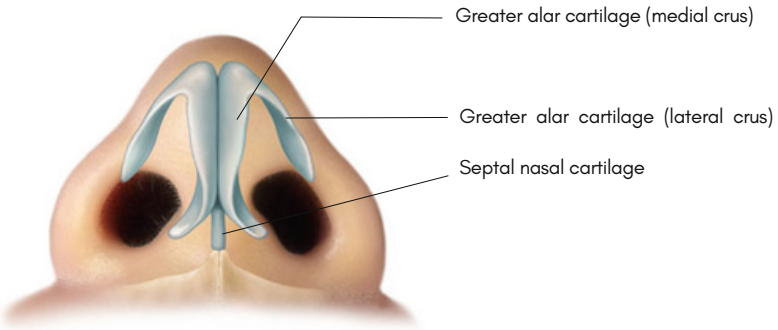


Cartilage of the nose (afrontal view, boblique view,

b



c



Cartilage of the nose cbasal view

These vessels travel closer to the medial crus than the epidermis and locate at the level of the deep fatty layer of the infratip of the nose.

However, as these vessels run close to the tip of the nose, they give off many fine arterial branches into both the superficial and deep fatty layers of the nose. When injecting fillers for columellar augmentation, the needle entry should be done deep to the skin at the tip of the nose. Deep columellar injections toward to the medial crus are highly advised.

Injection Points and Methods

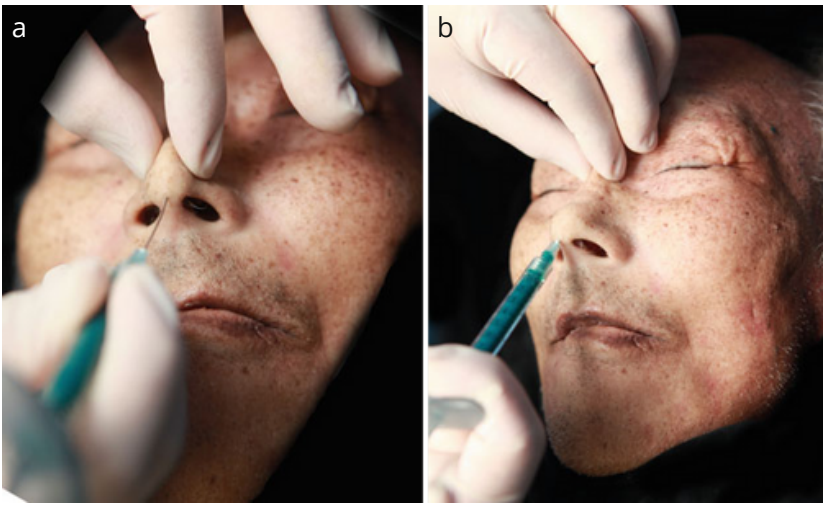
Dorsum of the Nose

The injection plane of the filler with a cannula should be at the level of the supraperichondrial and supraperiosteal layer. The entry point of the cannula should be made at the infratip lobule. Beginning from the sellion (deepest point of the radix), the augmentation for dorsum of the nose should follow with the retrograde thread injection technique

Columella

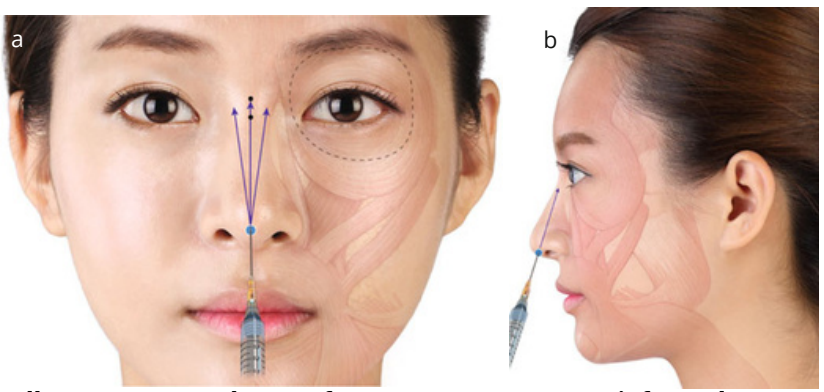
For the columellar augmentation, the cannula should be located between the medial crus of the lateral alar cartilage and the anterior nasal spine.

The insertion point of the cannula should be made at the infratip lobule. The HA filler is the most appropriate filler for nose augmentations since it can be an easily



Filler injection for nose augmentation (a, b)

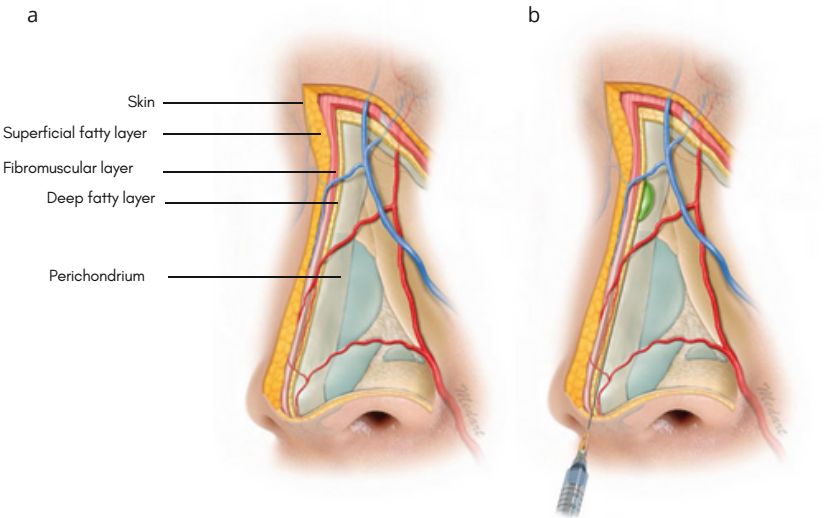
The insertion point of the cannula should be made at the infratip lobule.



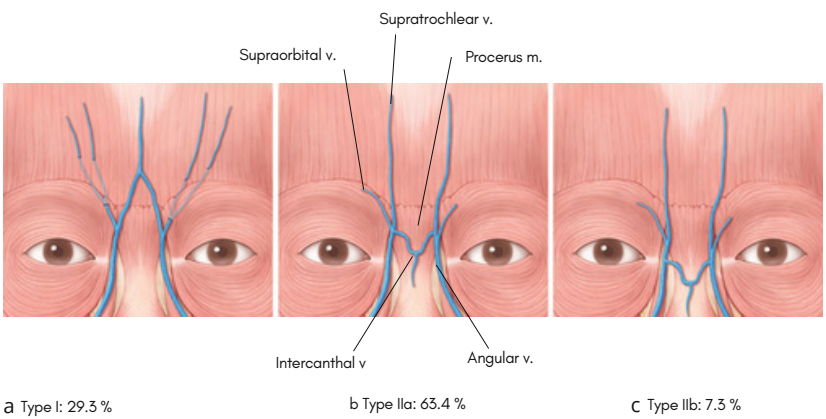
Filler injection technique for nose augmentation (afrontal view, blateral view)

The HA filler is the most appropriate filler for nose augmentations since it can be an easily correctable material when side effects occur. Furthermore, the use of a biphasic HA filler with a large particle size is recommended than a monophasic HA filler to minimize the risk of intravascular injection of filler mass. Fillers with a large particle size would be more suitable among various products of biphasic fillers.

Although gel-type monophasic fillers tend to last longer, their properties of water absorption may lead to the side effect of a widening of the dorsum of the nose. If calcium hydroxylapatite fillers are injected, it is hardly correctable and provides little buffer room when lumps will occur in relatively thin skin area or dorsum and columella line will be not at the proper line. So HA fillers are strongly recommended.



Anatomical layers of the external nose (a)and appropriate plane for filler injection on dorsum of the nose (b)



Distribution patterns and classification of the intercanthal vein (ICV). (a)Type I, near the midsagittal line, a single STV divides into two branches and each of the branches of the STV runs obliquely downward close to the medial canthus to meet the SOV, ultimately becoming the AV. (b)Type IIa, the ICV is located above the intercanthal line (ICL). (c)Type IIb, the ICV is located below the ICL in the nasoglabellar area.

HOW I DO IT: FILLERS
NOSE FILLERS

NON SURGICAL RHINOPLASTY

Difficulty: ●●●● Patient Satisfaction: ●●● Risk: ●●

Indications

Because the nose occupies the center of the face, mild asymmetries can be quite striking. Rhinoplasty surgery is not always a perfect procedure, and postsurgical defects can be difficult to correct. As a result, the use of fillers in small quantities to treat specific nasal deformities has become a way to fine-tune postsurgical noses. In addition, in some clients who refuse surgery or who are not surgical candidates, a nonsurgical approach to their nasal concerns may be possible using filling agents.

Anatomic Considerations

Knowledge of the ideal proportions of an attractive nose will be necessary as well as knowledge of the basic anatomy of the bony, cartilaginous, and soft tissue structures involved. It is also important from a safety standpoint to be aware of the key vascular channels to avoid intravascular injections.

Injection Technique

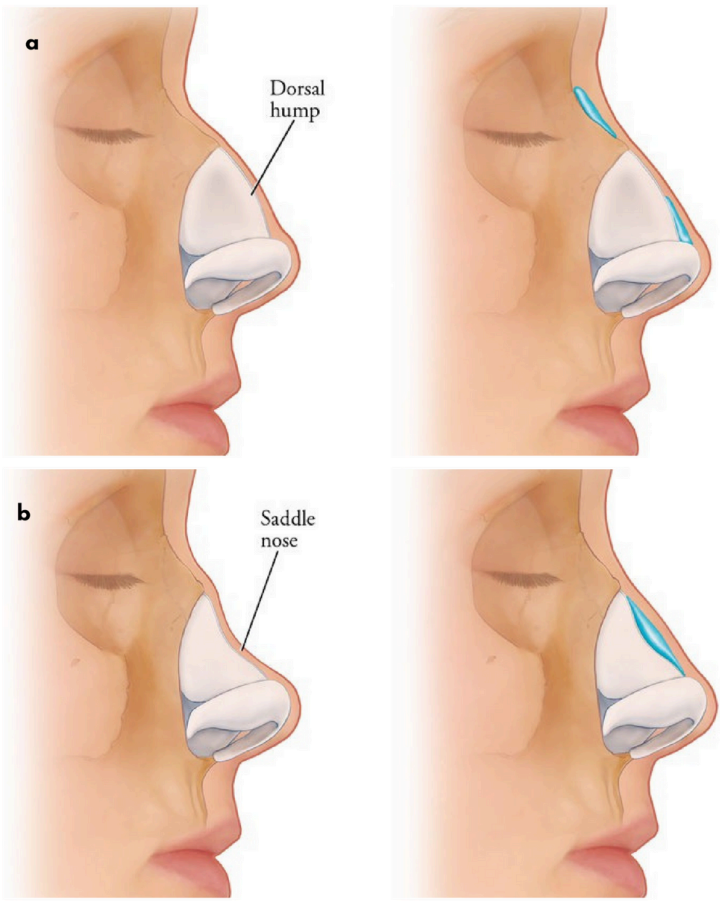
When injecting a hollow or void in the nose, it is best to start deep on the bone or cartilage and perform a retrograde injection with a threading movement to avoid a direct depot injection that could possibly flow into a blood vessel.

Dorsal Hump

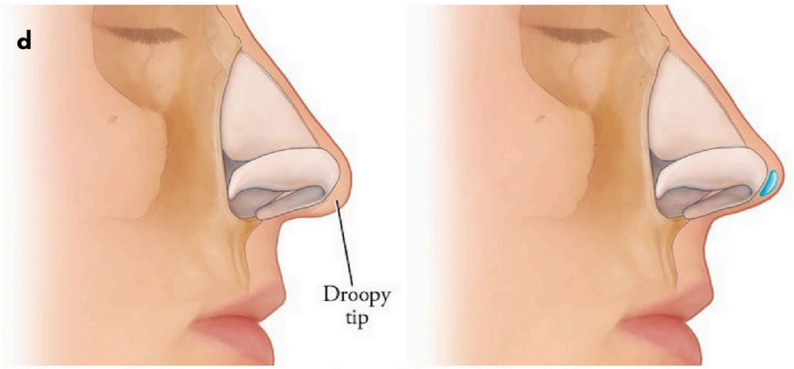
To straighten a dorsal hump, inject both above and below it as needed to straighten the dorsal profile. This technique can also be used on a wide nose to give the illusion of a higher and narrower nasal profile.

Post-Injection Instructions

Ice and pressure are helpful to prevent bruising. The product will swell some with an HA and feel firmer to palpation the first week and then blend in more naturally. The client should expect that the areas injected will look raised and welted at first. Swelling should improve within about 2 to 4 days.



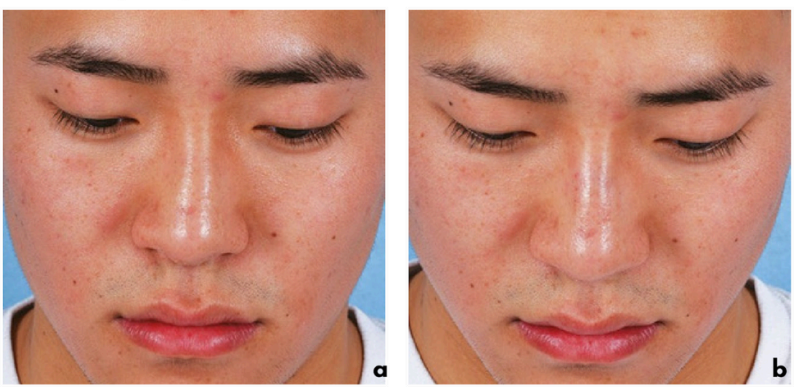
(a) Dorsal hump. Filler can be placed above and below a dorsal hump to straighten the dorsal profile. (b) Saddle nose. The concavity of the dorsum seen in the saddle nose deformity can be improved nonsurgical by using filler.



(d) Droopy tip. Filler can be used as a "tip graft" to define and elevate a ptotic tip.

Risks

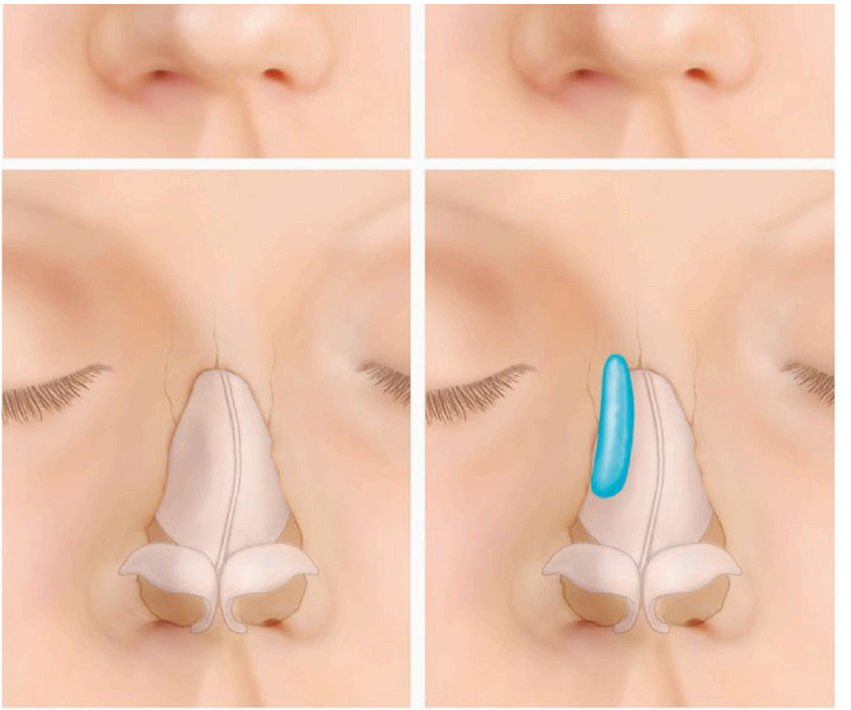
The most significant risks involve injection into a vessel that could lead to vascular necrosis. Retrograde injections and avoiding high pressure on, or blanching of, the skin during treatments can help prevent this devastating complication.



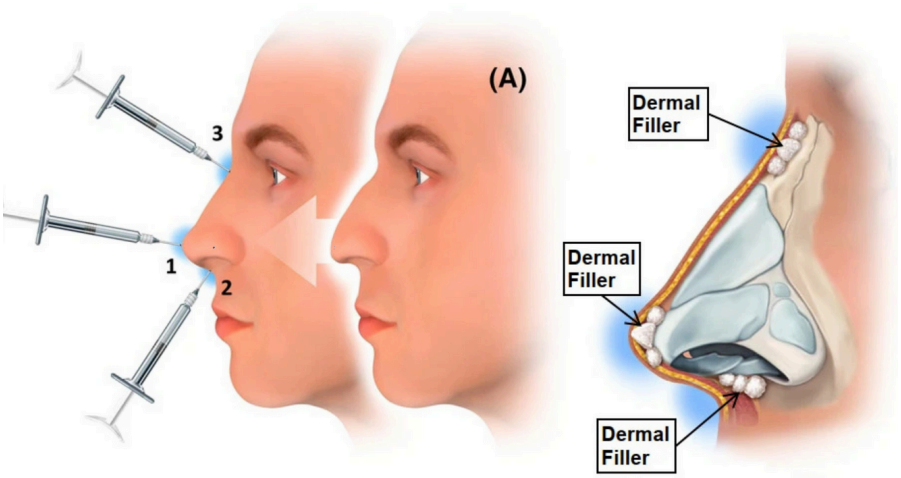
(a) Mild right lateral nasal sidewall depression post rhinoplasty. (observe the light reflexes) (b) Improvement of right sidewall depression by placing filler in the defect.

Notes of Injection

Under correction is recommended in this region. Also, keep the needle in motion, so as not to inject into a vessel and create an occlusion or embolic situation. Proceed with caution in post-rhinoplasty clients because prior surgery may compromise the blood supply to the nasal skin, which may increase the chance of skin necrosis.



(c) Crooked nose. Filler is placed along the periosteum or perichondrium in the concave aspect of the nasal sidewall to give the illusion of a straight dorsum. Pre- (left upper and lower panels) and post- (right upper and lower panels) injections along the right nasal sidewall improve a mildly crooked nose in a client with a persistent deformity after closed reduction of a nasal fracture.

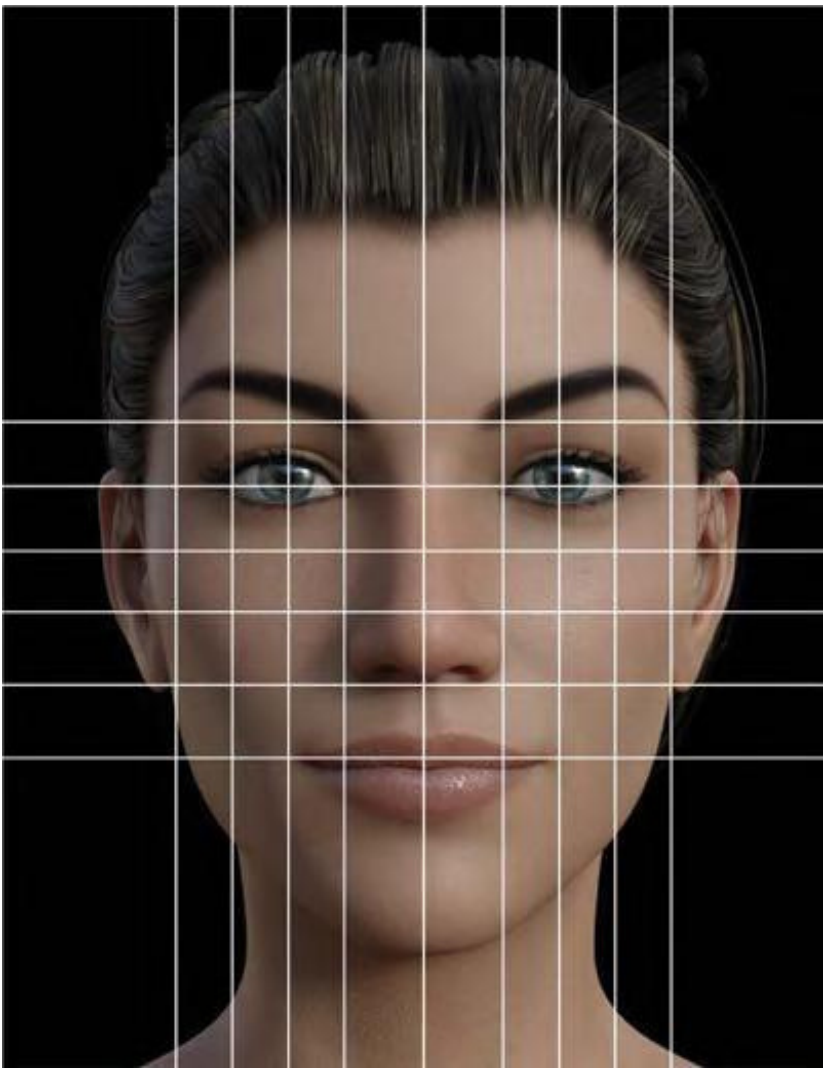

(A)
3
1
2
Dermal Filler
Dermal Filler
Dermal Filler

Patient selection is of utmost importance for nose reshaping with fillers. Both patients with thin and thick skin are suitable candidates for filler rhinomodulation.

Patients with thinner skin usually require less product. In these patients in particular, however, any mistake (too much material either absolutely or relatively) can be visible during and after treatment. Biodegradable substances, although temporary, are thus the best to start with. Thicker skins do not expand as easily as the thinner ones and usually require more product or larger molecules.

Nasal botulinum toxin injections for sagging nasal tip.

As a general rule, HA should be injected in small quantities for every treated area with constant checkup of the patient during injections and after 3 and 10 days. Injection volume and skin color should be assessed regularly to avoid any compromise of vascular perfusion. It is best to conduct a nasal grid analysis immediately before filler injection and during treatment (see Figure 5.18a). The nasal grid is traced with standard make-up pencils and analysis allows identification of primary defects: deep glabella, nasal hump, pseudohump, double dome or defective nasal projection, nasolabial angle and hidden columella, crooked nose, saddle nose, and nasal base asymmetry.



The nasal grid.

Injection Plan and Procedure

Skin is disinfected with a 75% alcohol solution. The nasal grid analysis is traced and the treatment performed by sole injector following the injecting protocol. Select a HA gel with a high G′, 20 or 25 mg/mL with 0.3% lidocaine, characterized by intrinsic viscosity and cohesivity. The main injection guidelines include the following:

1. To avoid vascular complications, it is prudent to stay in the midline , as the "major" vessels (angular arteries, columellar arteries, and dorsal arteries) normally lie laterally. In some patients, dorsal nasal vessels cross the midline, as apparent in some anatomic dissections, usually at the middle third of the nose. Variations in the anatomy could put some patients at higher risk for intravascular injection. Always aspirate for a 5–7 seconds before injection.

2. A 27G 13 mm needle should be used (or a 38 mm 25G cannula).

3. Two distinct planes of injection are strictly adhered to, in order to minimize vascular accidents:

• Supraperiosteal injection in the glabella/nasal dorsum/anterior nasal spine/columella.

• Deep dermal injection in the nasal ala/tip (and also permitted in the glabella in addition to the supraperiosteal injections).

4. Slow injection with small volumes is recommended, with constant patient monitoring mandatory for signs of vascular compromise.

5. Pinching soft tissue in midline: The treatment area is constantly pinched up and compressed during injection to avoid lateral HA displacement.

6. Lateral defects, where there is increased risk of intravascular injection, can be most safely corrected by midline injection followed by molding and purposeful lateral displacement of the body of HA. This technique is useful where there are lateral defects for correction and allows for avoidance of an increased risk of intravascular injection by leaving midline.

7. **Smoothing massage** at the end of every injection, with wet gloves, help to avoid “bumps.”

Treatment Plan Design

A sequence of injections is used in order to achieve the following aims:

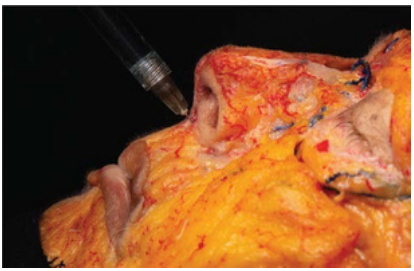
• Tip support and rotation (Sn), where there are two columellar arteries following the medial cartilaginous crus.

• Nasal base support (Rnb, Lnb), where the lateral nasal artery creates a subdermal plexus

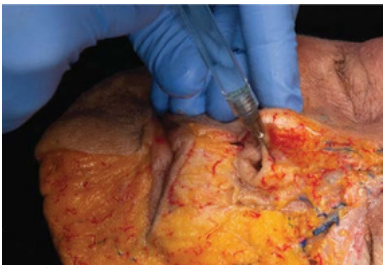
• Nasal tip projection (Nt), where columellar arteries and lateral nasal arteries create arcades.

• Glabella is the next step (Figures 5.25 and 5.26). • Then, inject in the midline on nasal dorsum (nasion = Na and nasal dorsum = Nd), where nasal dorsal arteries provide vascularization from the angular and the ophthalmic arteries.

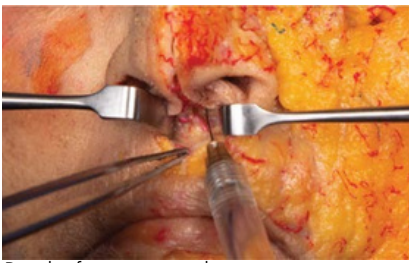
• The nasal dorsum can also be treated with the cannula



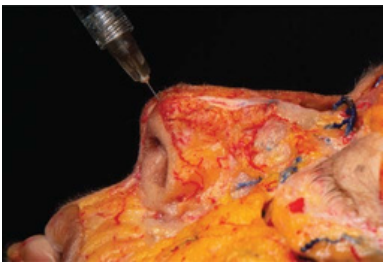
Anterior nasal spine injection.



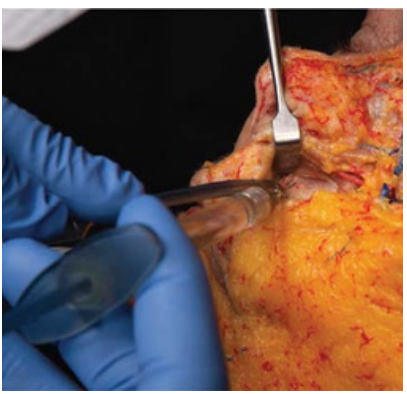
Depth of nasal ala superficial injection.



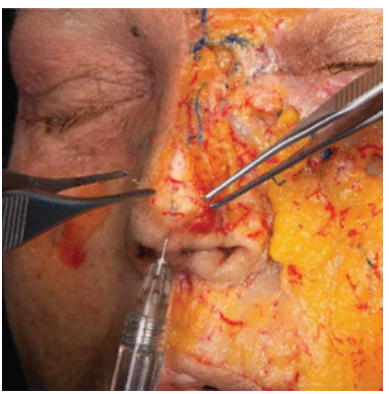
Depth of anterior nasal spine injection.



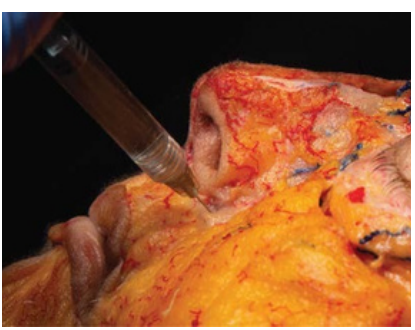
Depth of nasal intradomal deep injection.



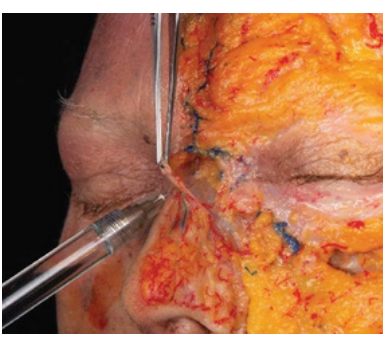
Depth of nasal base deep injection.



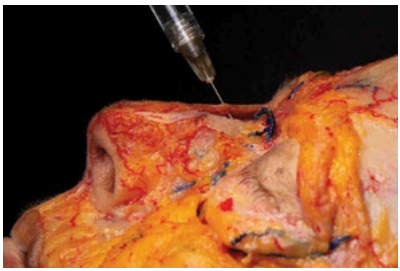
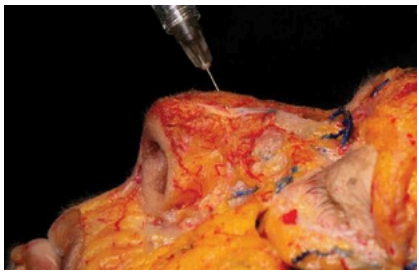
Depth of interdomal tip deep injection.



Depth of nasal base superficial injection.



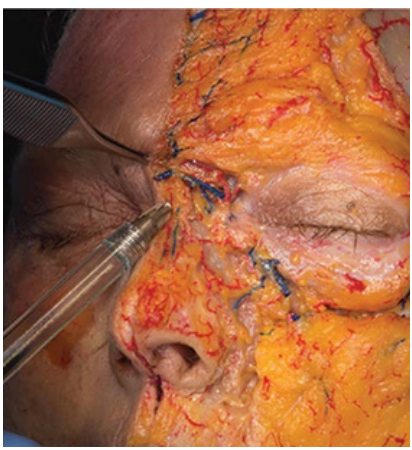
Depth of nasal glabella deep injection.

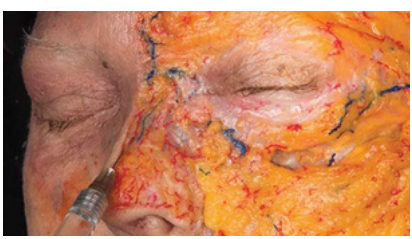
(a) Depth of nasal glabella deep injection. (b) Depth of nasal dorsum deep injection.

Following the nasal grid points, associated defects of the nasal structure should be treated with this sequence: Sn, Rnb and Lnb, Nt, Na and Nd. Remaining points through the grid are right and left nasal ala and right and left nasal base (Rna, Lna and Rnb, Lnb) for each side.

Columellar refinements are also possible in superficial fat or deep between the domes



Depth of nasal glabella deep injection with danger area

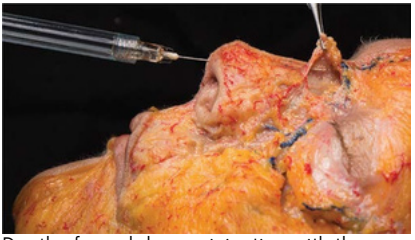


Depth of lower nasal dorsum deep injection

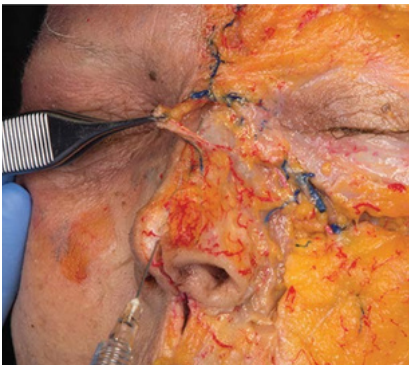
Isolated Defects

Deep glabella: Start with glabella and proceed caudally with few drops on the nasal dorsum and proceed with molding.

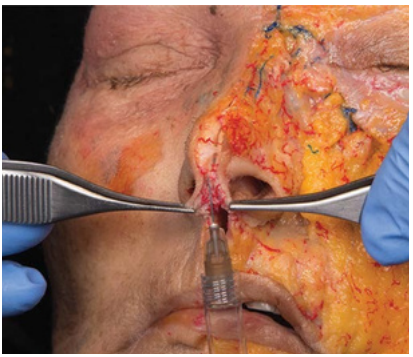
Nasal Hump: Treat first by injecting above the hump, extending and defining the nasofrontal angle. Overfilling of this area and the resulting straight-line deformity from the forehead to the hump should be avoided. Then augment the area of the dorsum below the hump. You can proceed with the tip if needed. Infradomal injection gives the illusion of cephalic tip rotation. A supra-tip break has to be preserved by filling this area as a last step, so the injection sequence is Na, Nd, Nt, St.



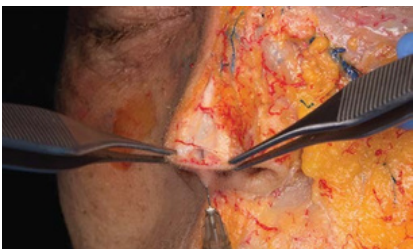
Depth of nasal dorsum injection with the cannula visible on the profile view.



Depth of nasal dorsum injection with cannula (deep) injection.



Depth of nasal columellar deep injection.



Depth of nasal tip deep injection.

Saddle nose: This is usual in Asian patients. If a CT scan shows a structural connection of the nasal dorsum anatomy, the shape can be corrected by filling the saddle area, which should be treated in multiple steps if the sagittal dorsal defect is more than 3 mm. First, define the tip projection, then filling runs from nasion to dorsum. So the injection sequence is Nt, Na, Nd, St.

Deprojected nose: The nasal spine, septum, and nasal base work as a scaffold that give support to the nasal tip. Subnasal injections that place HA anterior to the nasal spine produce an opening of the nasolabial angle and an illusion of tip cephalic rotation. Then, augment the nasal tip. Proceed with Na and Nd, setting a straight line from the height of the new nasal radix to the dorsum; set the sagittal projection of the tip and avoid an undesirable fullness of the supra-tip. So the injection sequence is Sn, Rnb, Lnb, Nt, Na, Nd, St.

Nasolabial angle and columella: A defined nasolabial angle is vital to achieve a harmonious and balanced relation between tip and lip.

Columellar retraction gives the illusion of tip caudal rotation. Perform a subnasal injection that places HA anterior to nasal spine, increasing columellar projection when needed. The second step is to support the lateral ala; then, perform a glabellar injection that should be carried out slowly and carefully to avoid ophthalmic artery embolization, deep to the bone. Next, perform tip injections and, only at the end, proceed to the nasal dorsum for refinement. So the injection sequence is nasal base, Nt, Ng, Nd.

Secondary Defects

Crooked nose deformity: Usually, the goal is to fill the concavities. The key point is the need to avoid the lateral nasal artery: the needle must enter on the midline; once the periosteum or perichondrium is reached, slide laterally deep to the defect or inject medially and mold laterally. The injection sequence is thus: Sn, Rnb or Lnb, Nt, Na, Nd, St.

Saddle nose deformity: This can be a consequence of aggressive primary surgery. If a CT scan shows a structural connection of the nasal dorsum anatomy the shape can be corrected by illing the saddle area.

First, define the tip projection; then filling runs from nasion to dorsum. The injection sequence is Nt, Na, Nd, St. If there are dorsal structural voids, such as exaggerated scarring or mucosal defects, filler should not be injected because of the deficiency of the structural integrity of the scaffold. The only procedure that can solve this complication is a secondary rhinoplasty.

Alar retraction or collapse: This can be reshaped with Rna and Lna injections. This should be undertaken by expert injectors—nasal surgeons or injectors that have more than two years of experience in full facial rejuvenation—because of the high risk of skin necrosis. Filling is done in the deep subdermal layer; as soon as skin blanching is detected, injecting has to be stopped.

Checkups

Nasal reshaping with fillers requires regular maintenance. Results usually last 4-12 months once the desired result has been achieved. Maintenance of these results would require one or two treatments the first year, followed by treatment once a year. This means that a non-surgical nasal correction should be proposed as a treatment protocol, not based on the amount of the material used.

Although injectable facial fillers can offer an efficacious alternative to surgery for the aging face, they also have their limitations. It is important to recognize specific circumstances that may be best managed with an alternative to fillers.

COMPLICATIONS NOSE FILLER

COMPLICATIONS

DANGER ZONES

To avoid vascular complications, it is better to **stay in the midline** because all the "major" vessels (columellar arteries, angular arteries, lateral nasal arteries and nasal dorsal arteries) normally lie away from the midline. The **plane of injection** is somewhat controversial, but **deep to periosteum and deep to perichondrium is our recommendation. Constantly holding the treatment area between two fingers during slow injection can avoid lateral filler displacement.** It has been possible to inject deep in the midline and to displace hyaluronic acid with molding. This technique has been useful to correct lateral nasal defects where there was an increased risk of intravascular injection. At the end of every injection, palpation and smoothing out of the skin with wet gloves helps to avoid undesired "bumps." The upper lateral cartilages are continuous with the nasal bones. The lower lateral cartilaginous vault comprises the medial, middle, and lateral crura. The anatomic dome is the junction of the medial and lateral crura. Depending upon the intrinsic relationship of these structures, the tip of the nose can be normal, bulbous, or boxy. Tip support is basically a combination of skin, ligaments, and cartilage. The depressor septi nasi is the most important muscle that acts on the tip and lip complex. It shortens the upper lip and drops the tip when smiling. Surgical resection or blocking with BoNT-A may be necessary to enhance the result of fillers, although this is not the rule. When reshaping the nose with fillers, the angles with the lips and the forehead are important. The former is the nasolabial angle and should be between 90° and 100° in men and between 100° and 110° in women. The latter is the nasofrontal angle, which is between the forehead and the nasal dorsum.

Clinical Considerations

It is important that all patients have realistic expectations for these procedures and understand that fillers can restore balance, enhance appearance, and minimize defects. Follow-up procedures a few weeks after the initial treatment may optimize results and lengthen the duration of the effects of these products which varies from about 3-12 months.

Non-surgical rhinoplasty is a safe procedure but complications may occur. Inexperienced injectors, improper patient selection, and defect/filler mismatch likely play an integrated role in the onset.

Although most complications of facial fillers are transient and minor in nature, it is important to discuss them with patients prior to injection. Common complications include bleeding, swelling, irregularity, erythema, bruising, and discoloration. Rare complications can include infections, lumps, the Tyndall effect, vascular compromise, necrosis and blindness.

Fillers should be resorbable to avoid undesired complications such as granuloma, nodules, dislocation, allergies, and long-term palpability. Currently high G′ HA is the "gold standard." Other materials can be palpable under the relatively thin nasal skin. Semipermanent filler such as calcium HA or permanent soft tissue filler such as silicone or acquamid can also dislocate and produce severe granulomatous reactions with resulting nasal cellulitis, nodules, or ulcers that are difficult to be treated. Moreover, clinicians cannot correct injection imperfections because of a lack of an equivalent to hyaluronidase.

Immediate Complications

Bleeding is associated with patient anticoagulation due to concurrent and/or recent use of aspirin, NSAIDs or blood-thinning medications. The use of large-bore needles and injection into highly vascular areas, such in the nose, can worsen the bleeding.

Irregularity may be common. In cases with a deviated nose, the best method is to inject small amounts of filler, slowly, in a multiple-step procedure.

Embolization can produce necrosis of down-stream structures. If this were to occur, injection of hyaluronidase (Hase) is mandatory. Inject as soon as possible and distribute Hase around the injected area at least 300 U/hour for at least 5 or 6 injections. Hot packs, soft massages, and application of nitroglycerin paste help promote vasodilatation but are less necessary if hyaluronidase has been injected. Low molecular weight heparin may be used to decrease thrombosis.

Delayed Complications

Bruising may be caused by needle injury. Avoid further injection unless essential; compress with gauze and apply ice packs to minimize bruising.

Infections are rare; however, patients with a susceptibility to or a history of herpes simplex may be candidates for prophylactic antiviral therapy.

Erythema accompanied by pruritis and fever may be related to hypersensitivity. In severe circumstances, corticosteroids are necessary.

Lumps are usually related to nodules or to a granulomatous reaction mediated by an inflammatory response. Treatment options include corticosteroid injection, hyaluronidase, and surgical removal.

The Tyndall effect denotes a bluish discoloration due to superficial product placement. Placement of the correct product in the proper plane may limit the likelihood of the Tyndall effect. Hyaluronidase may be needed for reversal.

CAUTION

Embolization can involve the ophthalmic artery which can lead to blindness. Embolization can be a consequence of direct injection of the internal external carotid network, particularly in the facial danger zones. Simptoms can be Immediate (the majority of cases) or rarely delayed between 10 minutes and 1 hour. The dose of hyaluronidase differs among reports; however, we suggest 300–1500 IU of hyaluronidase repeated at frequent intervals (every 2–4 hours). The worst potential event is embolization of the ophthalmic artery, which can lead to blindness [1,7]. Should this occur, the clinician has the possibility of retro-bulbar injection of hyaluronidase with a 25G cannula within a timeline of 90 minutes. Options can be injecting hyaluronidase through a cannula either 8 mm below the inferior tarsal strip, following the orbital floor with a downward curved direction at a depth of 3 cm from the bony orbital rim or with entry 1 cm above the medial canthus directed 30° medially following the medial orbital wall.

THANK YOU FOR READING