CHAPTER 1

Injection Anatomy: Avoiding the Disastrous Complication

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Over the past two decades, neuromodulators and 'dermal' fillers have provided cosmetic physicians with the tools necessary to enhance facial features non-surgically and usually with minimal discomfort. Having a profound impact on beauty is no longer limited to a surgeon's knife wielded by an experienced specialist proficient in facial anatomy and aesthetics. Originally intended for the safer location of intradermal deposition, synthetic filler therapy has been extended beyond the eradication of unwanted wrinkles and folds into the realm of facial contouring and volume restoration. The transition of more robust fillers into deeper treatment planes by practitioners unfamiliar with the attendant vital anatomy has resulted in the appearance of devastating intravascular complications.

Complications have arisen from the use of various dermal fillers since their inception. Historically, paraffin, Vaseline, and many other materials were used that could not only cause many of the same types of devastating vascular complications, but also result in serious adverse events that were long lasting due to tissue incompatibility and immune-mediated issues [1]. Fat may be considered the archetype of 'deep' fillers, and embolic phenomena have been reported from many sites over the decades since it was first developed as a technique for volume replacement (including blindness, stroke, and tissue necrosis) [2]. Since the development of the hypodermic needle, many different drugs that were either partially insoluble or that had serious inflammatory effects on the linings of blood vessels (particularly arterial wall linings), were implicated in many serious adverse events resulting from the ensuing ischemia when accidental intra-arterial injection

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caused inflammatory desquamation of the arterial lining tissue. From this historical perspective, then, the present state of filler complications does not present anything new, but rather mirrors experiences with other filler materials. The most successful modern fillers (the class of hyaluronic acid derivatives) now mainly show improved results for tissue integration and also lack inflammatory effects [3].

It is imperative that all injection specialists have an intimate understanding of facial anatomy and its relationship with injection therapy so that serious adverse events are minimized. A 100% foolproof method of facial injection therapy is impossible because of the variability in facial anatomy. Anatomy textbooks only give an average depiction of what exists *in vivo*, with numerous classifications and variations of vascular patterns reported (with their intendant percentages) for every facial region [4]. It is therefore crucial that treating physicians familiarizes themselves with the different techniques available to limit intravascular compromise (Table 1.1).

Table 1.1 Techniques to limit intravascular injection.

1. Know your injection anatomy – avoid danger areas and depths.
2. Aspiration before injection in higher risk areas [5]. This is not a guarantee of extravascular location as false negatives are high. Nonetheless, it is still the authors' recommendation, especially in higher risk areas.
3. Slow injections with the least amount of pressure [6] (definitely advantageous). Adverse events will commonly occur when the injector is rushing to complete a treatment.
4. Move the tip of the needle slightly with delivery of the product [7]. Although theoretically this will limit the amount of possible embolic material, it is controversial as the tip can move in or move out of a vessel.
5. Incremental injections of 0.1–0.2 cm^3 of product [8]. Severe adverse events have been associated with a significant deposition of product.
6. Small syringe to deliver precise aliquots [9]. The amount of product deposited over time is a significant factor in embolic events.
7. Small needles to slow the injection speed [10]. This is controversial in that the higher gauge can conversely access the smaller diameter vessels inaccessible to larger bore needles.
8. Blunt flexible microcannulae [11]. Intravascular transgression is still possible and has been reported. A cavalier approach is not warranted.
9. Addition of a small amount of vasoconstrictor in the product or as a preparatory step may effect some vasoconstriction without the long lasting block nor blanching of the skin [12].
10. Patient selection (e.g. previous surgery with scarred beds portends an increased risk of a vascular event).
11. The injector should always observe the skin at the area of injection and not the syringe in his/her hand, just as a driver watches the road and not the steering wheel. Drivers have rear view mirrors into which they glance to prevent accidents – so too does the injector whose rear view mirror is the glabellar region. Glabellar blanching can be the first indication of intravascular injection in the face regardless of the injection location. Therefore occasional glancing into the 'rear view mirror' of the glabellar region for signs of blanching, *regardless of the site of facial injection*, is advisable.
12. The occurrence of patient pain distant to the site of injection, in spite of lidocaine with the commercially available products [13]. (N.B. This is not noted in every case)
13. The possibility of delayed onset (several hours later) of symptoms and signs that require emergent care [14].

In principle, all the facial areas currently considered for treatment can be divided into higher risk or lower risk areas, but as we shall see, there are no 'zero risk' areas. This is an important detail that is often glossed over by manufacturers, who are keen to promote fillers as safe and effective. As the numbers of practitioners (who often learn the techniques in a weekend course) increase, certain trends in complications have been identified. One important issue is that many new practitioners have no recent experience with the vascular anatomy of the face, and worse, are completely unfamiliar with the previous reports of serious complications with the use of fillers. The combination of exuberance for a new technique, its seemingly easy implementation, and the lack of knowledge of the consequences of severe complications, has resulted in many unrecognized adverse events with high morbidity. Although serious adverse events can happen even in the hands of the most experienced injectors, when these are properly recognized and treated appropriately, the outcome can be good, but when they are not, the outcome can be seriously debilitating or mutilating. The purpose of this chapter is to familiarize the injector with the 'injection anatomy' of the face, so that practitioners can properly gauge the level of risk for the intended treatment.

1.1 Injection Anatomy Defined

Many different categories of human anatomy have been described, most of which relate to the instrument being used and the ensuing treatment or therapy (e.g. surgical anatomy, radiological anatomy, etc.).

Injection anatomy, not previously described, is centred on the use of a syringe and needle rather than a scalpel. Where the tip of the needle resides (the point from which the product will flow) once under the skin is crucial. Injecting under the skin involves encountering vital structures. Knowledge of injection anatomy therefore pertains to the *depth* of injection as it relates to the location of the tip of the needle.

Injection anatomy can be defined as the study of regional anatomy as it relates to surface landmarks and the underlying *depth* of targeted tissue and vital structures. Although a myriad of vascular patterns exist in two dimensions, there is relative consistency in the depth (third dimension) at which vessels pass through the tissues in specific geographical regions of the face. Appreciating the depth location of the tip of the needle, although not infallible, should guide treatment into 'safer' lower risk zones for specific facial regions. The clinician's ability to delineate these facial anatomical zones at the time of treatment is limited to visual and palpable topographical assessment. To this end, five bony and three soft tissue landmarks must be discerned, which divide the face into specific treatment regions according to depth (Figure 1.1).

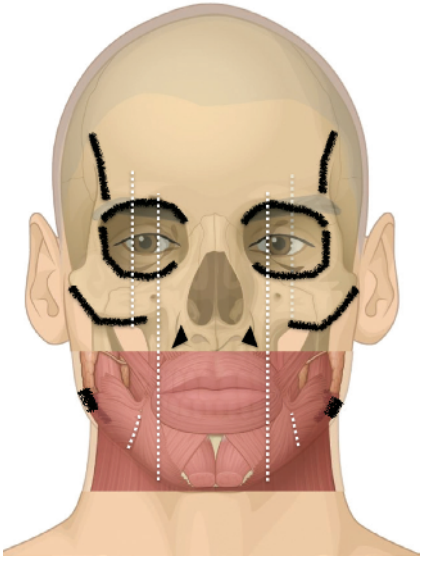


Figure 1.1 Eight topographical landmarks for defining injection zones. The five boney markers are the temporal crest, the orbital rim, the inferior maxillary border, the pyriform fossa, and the gonial angle. The three soft tissue landmarks are the medial iris, the lateral iris, and the anterior border of the masseter.

1.2 Pathogenesis of Vascular Obstruction

Intravascular deposition (either arterial or venous) of filler product, and subsequent embolization is a necessary feature of vascular compromise in the face. Unlike the distal limbs where compartment syndromes may occur from external compression, this is not the case for the facial partitions, especially considering the amount of product being deployed and the extensive collateral vascular arborization.

In terms of general organization, the arterial blood flow is from larger bore to smaller vessels, whereas the reverse is true in the venous system (smaller veins connect to larger diameter veins). Intravenous injection of sclerosing drugs works because the material is rapidly diluted and mixed with blood as the material flows through ever-larger vessels on the way to the right heart. Any foreign material in the venous system is ultimately filtered out in the pulmonary arterial system. When relatively large amounts of foreign material are injected intravenously, serious pulmonary complications have been reported [15]. However, with the small aliquots of hyaluronic acid (HA) filler typically injected, the resulting pulmonary lesions often go unnoticed or undiagnosed.

Tissue necrosis following a filler treatment is invariably due to inadvertent intra-arterial embolization of filler material as confirmed by the

finding of intraluminal filler material on pathological examination of necrotic skin [16]. The filler may enter the vessel directly, as a result of intra-arterial needle tip location, or indirectly, through tissue channels created by the passing of a needle or cannula through the vessel (discussed in detail below). The embolism may be anterograde, i.e. distal or downstream from the site of injection, or even retrograde, contrary to the normal direction of blood flow (vide infra). Although filler material has been identified in the lumen of arteries in every case examined histologically [17], there is a common misconception that external pressure on a small artery is sufficient to cause skin necrosis by limiting blood flow. This is incorrect – tissue necrosis is the sine qua non of accidental intra-arterial injection. Repeated separate laboratory studies in animals have not been able to cause tissue necrosis by external pressure alone, even when very large amounts of filler have been used to significantly increase interstitial pressure [18]. This is analogous to the situation when intravenous fluid has 'gone interstitial'. There may be tissue blanching from the pressure increase of the injectate, but unless the liquid is toxic (e.g. chemotherapeutic drugs, etc.), the pressure resolves long before any tissue necrosis occurs. Similarly, extensive experience with tissue expanders has demonstrated that skin is extraordinarily resistant to necrosis on the basis of pressure alone, unless extraordinarily severe, and/or the tissues are abnormally scarred. (Tissue manometry studies carried out decades ago on tissue expanders showed that tissues respond rapidly, first by elastic and then by plastic deformation in response to pressure, and indeed, this forms the basis for the technique of tissue expansion.) Although future information may reveal exceptions to this rule, in general, any type of tissue necrosis following a filler injection is due to inadvertent intra-arterial injection [19]. The filler may travel extraordinary distances depending on the unique vascular anatomy of the area, even crossing the midline in certain situations. Almost 100 years ago, Freudenthal [20] described necrosis of the digits from an injection in the deltoid due to arterial embolism. Understanding this phenomenon explains the dozens of cases of blindness reported due to filler injections in the face [21].

Every injection under the skin violates a vascular entity, regardless of the appearance, or not, of a drop of blood from the injection site. Skewering a vessel with the subsequent deposition of product outside its walls, in a previously non-violated, non-scarred bed, should limit any adverse event to possible bruising (once the tamponade effect of the needle is lost upon its withdrawal). Application of immediate pressure over the injection site should limit this untoward aesthetic consequence.

As mentioned earlier, intravascular instillation of product may occur as a result of the tip of the needle being located inside the vessel, or more uncommonly as a result of a side-cut in the vessel created by the needle

in zones of scarring where subsequent flow of product follows the tunnel created back into the vessel. With the tip of the needle inadvertently located within a vessel, once the plunger of the syringe is depressed, the pressure generated at the tip of the needle surpasses the systolic pressure within the vascular system. The flood of product that ensues is indifferent to the actual direction of blood flow, and obeys Poisseuile's law of resistance, which is inversely related to the radius (to the fourth power) of the vessel (Figure 1.2). Initial intravascular injection, if the pressure is sufficient, proceeds in a retrograde fashion down the larger proximal vessel, and subsequent distal laminar flow down tributaries ensues once the pressure from the plunger is released [22]. The local pressure gradient of the blood inside the artery in comparison to the pressure of the product at the tip of the needle will determine the direction of flow. A slowly injected filler may gradually fill the distal artery, then fill proximal to the site of injection. From there, it may travel along collateral vessels or tributaries to distal sites. This explains the occurrence of biopsy-proven arteriolar embolization in the purple livedo reticularis skin zones proximal to the actual needle insertion site. Therefore, any complaint of even a late occurring 'bruise' remote to the site of injection necessitates that the patient is seen to rule out a possible vascular event. A very large bolus can be thus expected to completely fill the distal vessel, then fill the proximal vessel past the branch points and into the larger supplying vessels, and from there be carried to distant sites by the normal

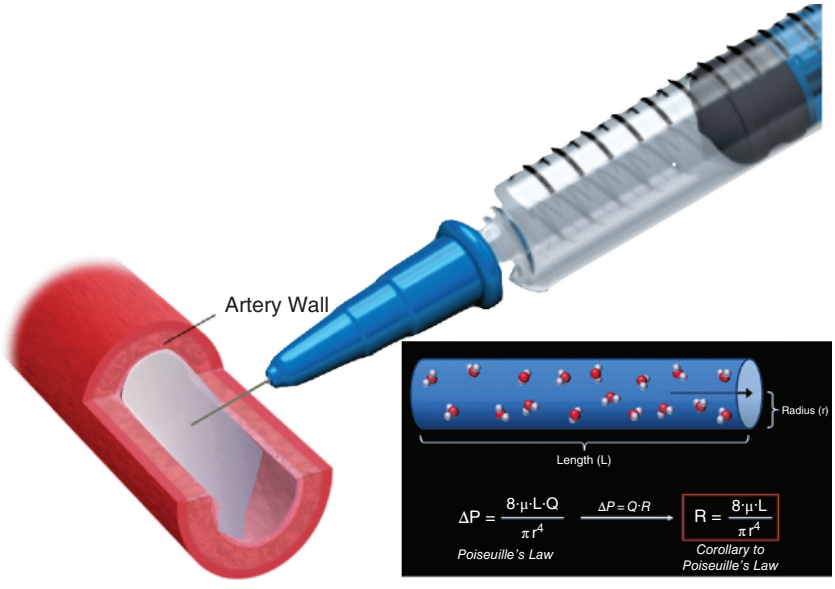


Figure 1.2 Intraluminal injection of filler results in flow of product following Poiseuille's Law preferentially down larger diameter vessels (usually proximal).

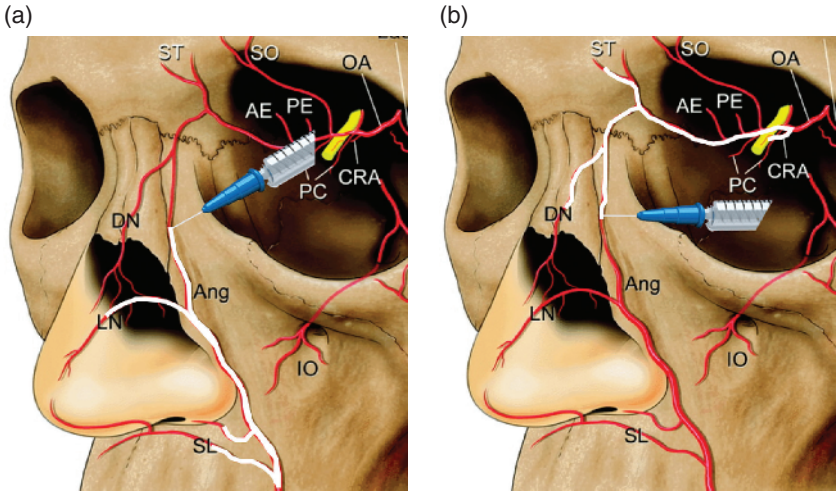


Figure 1.3 Depending on the direction of intravascular product flow, skin necrosis (a) and/or loss of vision (b) may occur.

flow of blood. The rare occasional delay in these occurrences can be explained by the initial clogging of a proximal vessel without obvious vascular compromise (due to distal collateral flow from adjacent vessels), followed hours later by dislodging of the product with resultant distal arteriolar branch obstruction (beyond the ability of collateral vessels to supply additional circulation). However, usually the effects of ischemia are immediate, with instantaneous skin blanching, followed by livedo, then dusky blue-black discoloration of the skin. Because most commercially successful fillers are now compounded with lidocaine, pain may no longer be an early sign of embolism. Fillers vary in their cohesivity, and very 'thin' fillers (reconstituted fillers in particular) may travel further into the smaller pre-capillary arterioles compared with the more viscous fillers, which become lodged in the larger arteries.

In summary, a typical vascular adverse event is due to filler material inside the arterial lumen. The filler may be propagated distal or proximal to the site of injection (Figure 1.3), and may cross the midline through collateral vessels.

1.3 The Forehead

The main arterial supply to the forehead is from three vessels: the paired supraorbital (SO) and supratrochlear (ST) vessels (internal carotid origin), and the frontal ramii of the bilateral superficial temporal arteries (external carotid origin). These latter vessels most often anastomose with the supraorbital



Figure 1.4 The supraorbital and supratrochlear arteries are extensions of the ophthalmic artery and exit the skull at the medial iris and medial corrugator crease overlying the supraorbital ridge, respectively.

or supratrochlear arteries, completing direct links for the entire forehead arterial supply with the ophthalmic artery within the orbit.

The SO and ST arteries (the extracranial continuations of the ophthalmic artery), exit the orbital fossa at the level of the supraorbital rim (SOR) of the frontal bone (Figure 1.4). Retrograde flow of product through these vessels allows for direct access to the globe and has been implicated in visual loss due to embolization of the central retinal artery.

The SO artery origin is typically found at the eyebrow level within 1 mm from a vertical line above the medial canthus, and is one of the most consistent anatomical findings of the upper third of the face [23]. In anywhere from 50 to 80% of anatomical dissections, it emerges through a notch that is palpable above the medial iris, 20% of the time through a foramen or ostium that is not palpable, and 20% of cases show a notch on one side and a foramen on the other [24]. The supratrochlear vessel location in the glabellar region is more variable, emerging from the orbit by coursing around the SOR 8–12 mm medial to the origin of its SO counterpart. Topographically, it can be located directly beneath or 2 mm lateral to the most medial crease of the contracted corrugator supercilii muscle.

Upon their emergence from the skull, and within 1 cm of their exit, the SO and ST arteries pierce the galea underlying the forehead muscles to ascend the forehead initially within the glabellar complex of muscles. These vessels course from deep to superficial for the next 1.5 cm as they continue within the substance of the frontalis to lie on the muscle's superficial aspect (Figure 1.5) under its anterior fascia [25]. With increased intravascular pressure and its associated vascular dilatation during exercise exertion, the pulsating arteries or accompanying engorged veins can be visualized along their superficial courses beginning approximately 1.5 cm above the

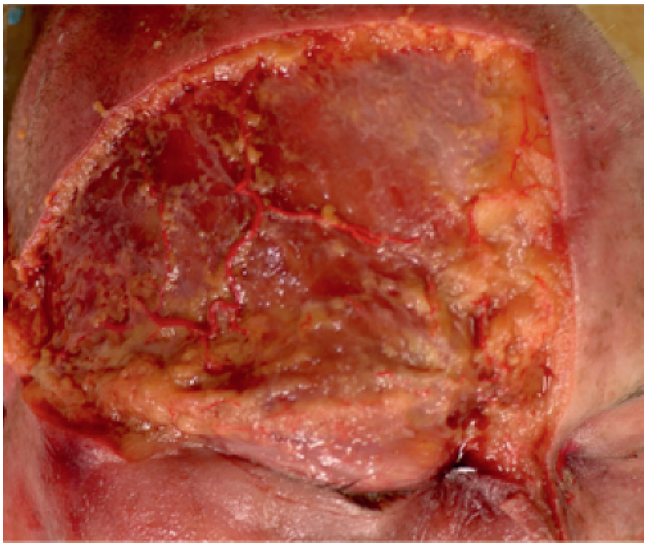


Figure 1.5 The emergence of the major vessels of the forehead on the surface of the frontalis muscle 1.5 cm above the orbital rim. The frontal branch of the superficial temporal artery can be seen anastomosing with these vessels in the forehead.

SOR. They are not apparent in the eyebrow region as the vessels lie deep within the glabellar complex of muscles (Figure 1.6). This area extending for 1.5 cm above the SOR should therefore be considered a 'no fly zone' for needle penetration below the skin. The minimal thickness of tissue from epidermis to bone in this region is approximately 2.9 mm [26]. The diameter of the SO and supratrochlear vessels can approach 1.8 mm, leaving an unmanageable margin of safety for product deposition under the skin [27].

The subgaleal/supraperiosteal plane superior to this 'needle no fly zone' remains relatively avascular, devoid of major vessels, as is evidenced by its frequent choice for bicoronal and endoscopic forehead lifts. The safer depth for filler deposition would logically be on bone of the forehead (supraperiosteal) above the 1.5 cm 'needle no fly zone' of the SOR region where vessel depth is indeterminate. In fact, because the glabellar vessels are terminal and have no collaterals, it must be stated that the entire no-fly zone should be considered a high risk area for filler injection. It should be noted that deep periosteal branches of the supratrochlear and SO arteries have been described [28], the former of which is intentionally preserved for increased circulation to forehead flaps designed for nasal reconstruction (Figure 1.7). These vessels should be of minimal consequence in supraperiosteal deposition of forehead contouring filler, as the majority of the superficial supratrochlear blood supply to the forehead skin would be unviolated, and retrograde flow of product in this deep branch is highly unlikely due to the small calibre and tortuosity of the vessel.

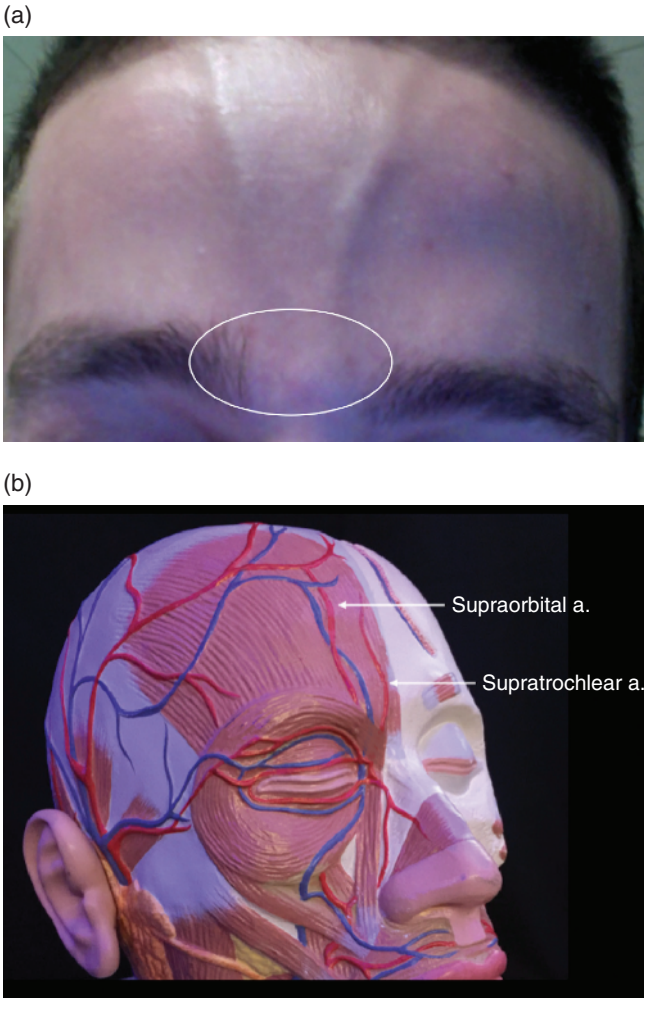


Figure 1.6 Upon exiting the orbit, the supraorbital and supratrochlear arteries pierce the overlying galea at the level of the supraorbital rim. They course deep within the glabellar complex of muscles in their initial 1.5 cm (indicated by the circle in (a)) as they ascend in the forehead to lie on the muscle and under its superficial fascia (b).

The supratrochlear nerve and superficial branches of the SO nerve follow the arterial cascade in their depth and distribution. The deep branch of the SO nerve, however, remains in a supraperiosteal location as it courses up the forehead on its way to 'supply' the posterior scalp with sensation [29]. Its supraperiosteal location 1–1.5 cm medial and parallel to the temporal fusion line (TFL) deep in the forehead is relatively consistent (Figure 1.8). Nerve impingement in this region by deep needle penetration on bone is not recommended in order to avoid causing referred pain in the posterior scalp. Furthermore, several cases have come to the attention of the authors

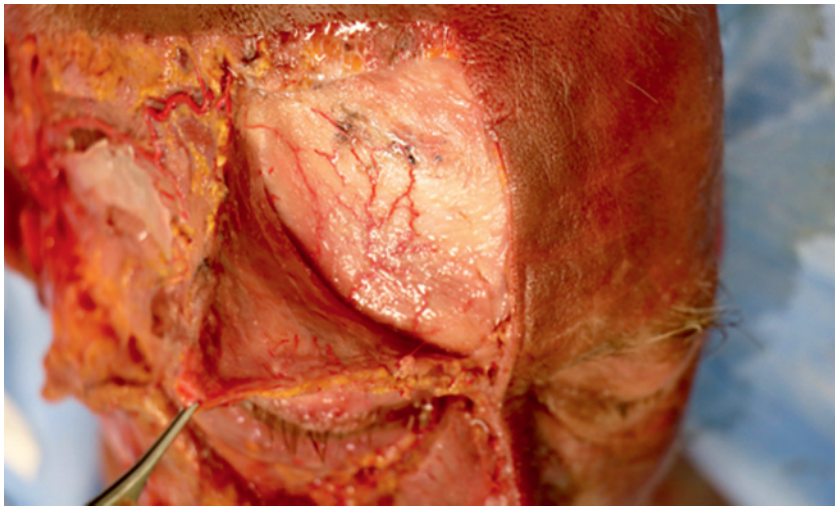


Figure 1.7 Tortuous deep periosteal branches of the supraorbital and supratrochlear arteries.

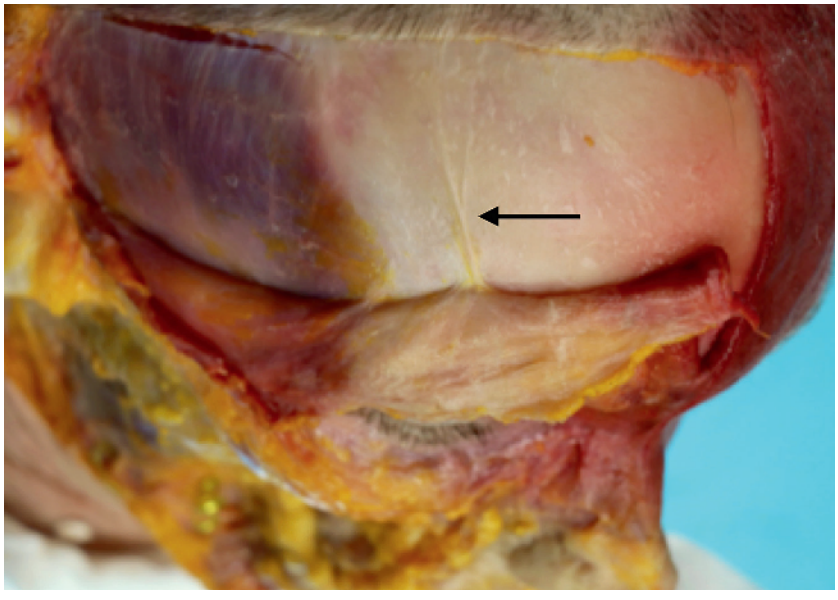


Figure 1.8 The deep branch of the supraorbital nerve (black arrow) is shown ascending the forehead on the periosteum medial to the temporal fusion line. The frontalis muscle is retracted inferiorly.

of accidental intraluminal injection of the nerve's accompanying artery, with a resultant thin vertical band of skin necrosis in the lateral forehead (S. Liew and S. Hart, personal communication). It is the authors' suggestion that depressions requiring contouring in the 1–1.5 cm zone medial

to the temporal crest (where the deep branch of the artery and nerve are located) be accomplished with more medial placement on the periosteum of the forehead and subsequent 'milking' of the product more laterally into the defect. This technique of adjacent fill and milking the product to the desired location is a general principle for treating many higher risk areas, but requires detailed knowledge of the fascial boundaries, anatomical planes, and the fat compartments of the face.

When filler is injected for persistent glabellar creases after toxin therapy, the injector must be highly cognizant of the depth of the needle in that it does not pass beneath the dermis of the glabellar region. As described above, the glabellar zone is a watershed area for the communication of the internal and external carotid systems, and represents a highway into the skull with resultant visual and intracranial adverse events. Monitoring the increased plunger resistance associated with intradermal injection is paramount to avoiding serious complications. If plunger resistance is lost during treatment, it must be assumed that the needle has passed below the restrictive distension of the dermis – injection must be ceased immediately and the needle withdrawn to a more superficial intradermal location. Again, it is vital that skin vascular integrity is constantly monitored in the glabellar region due to the preponderance of end-arterial vessels in the region.

In the event that volume restoration is indicated along the curved SO brow, a lateral approach with slow antegrade injection through a larger bore cannula (25 g or greater) is recommended. Encroaching in the radix area in a vertical direction parallel to the course of the supratrochlear vessels, even with the large gauge blunt cannulas used for autologous fat, can lead to intravascular cannulization and subsequent serious injury. A more prudent approach might involve lateral approach with antegrade injection of product. In this manner, the intravascular deposition of product will be minimal in the unlikely event of vessel penetration.

1.4 The Temple

Safe correction of overly scaphoid temporal hollows can be achieved only through a solid understanding of the injection anatomy of the region (Figure 1.9). The temporal fossa is bounded superiorly by the TFL or crest, which is most palpable at the lateral brow margin. The resident temporalis muscle of mastication is firmly anchored in its superior portion to the temporal bone, as it must exert significant pull through a musculotendinous insertion on the coronoid process of the mandible. The periosteum of the forehead continues over the temporalis muscle as the *deep* temporal fascia (an obvious misnomer), and splits into two sheaths (Figure 1.10).

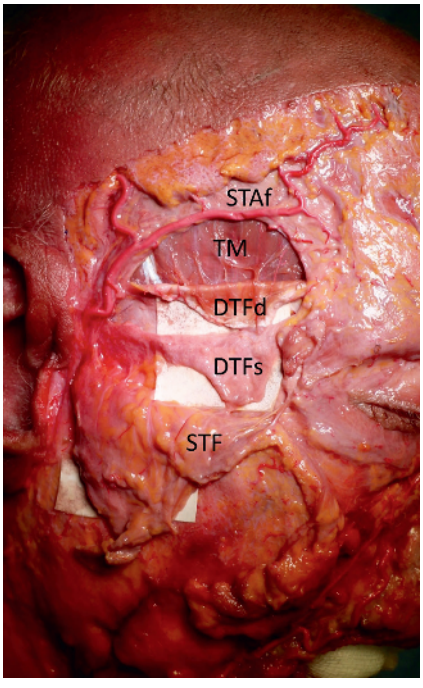


Figure 1.9 Particular anatomy of the temporal region: frontal branch of the superficial temporal artery (STAf); temporalis muscle (TM); deep layer of the deep temporal fascia (DTFd); superficial layer of the deep temporal fascia (DTFs); superficial temporal fascia (STF).

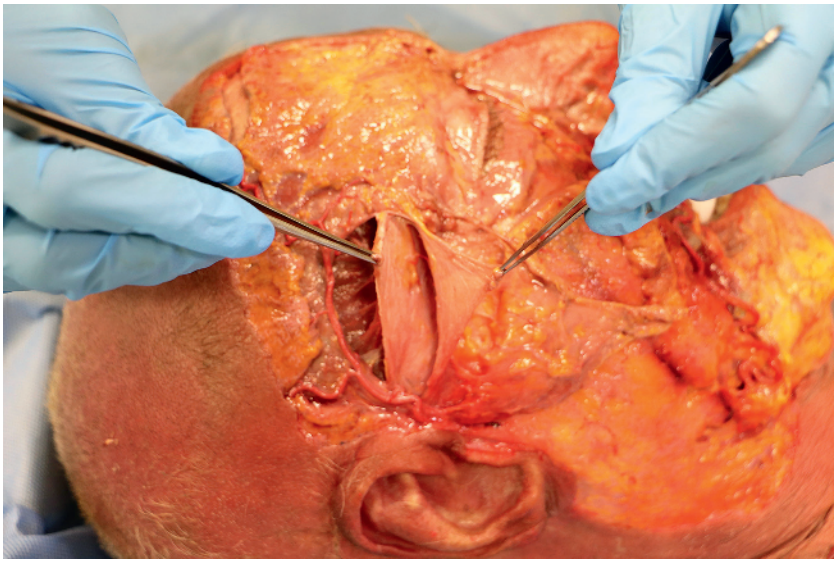


Figure 1.10 Cadaveric dissection demonstrating the two layers of the deep temporal fascia. The superficial temporal fascia containing the superficial temporal vessels is retracted to the right.

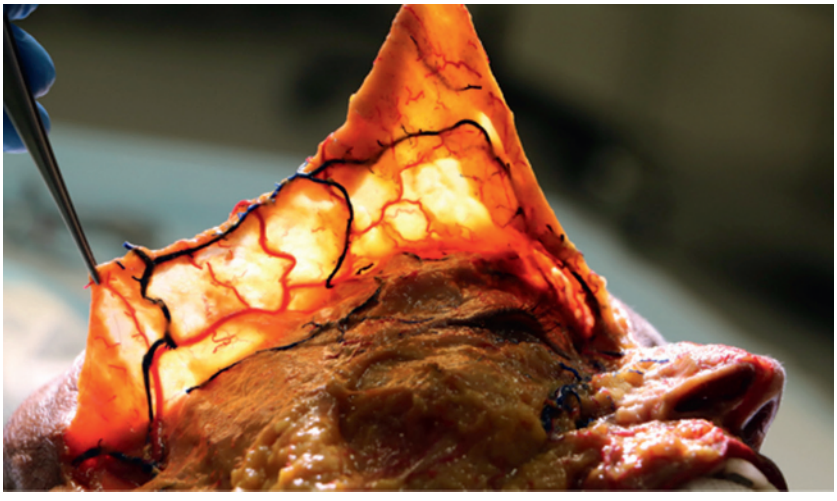


Figure 1.11 The superficial temporal fascia elevated showing the contained vascular arcade.

Similarly, the dense galea of the forehead, being unable to pass under the temporalis due to the latter's firm attachment to bone, continues on the muscle's surface over the deep temporal fascia as the superficial temporal fascia. The injection specialist can use this unusual anatomic anomaly to his/her advantage in allowing the anatomy to direct the flow of filler instilled in the region.

Every level of the temple contains vascular structures which must be avoided. The superficial temporal fascia, the deep leaves of which contain the ramii of the superficial temporal artery (Figure 1.11), lies on the deep temporal fascia. Superficial needle injection of filler in this region should be avoided, as inadvertent intravascular injection into this system of vessels can pass retrograde into the ophthalmic artery causing central retinal artery occlusion. The deep temporal arteries (anterior and posterior), branches of the second division of the internal maxillary artery, as well as the middle temporal artery, pass within the deep substance of the muscle (Figure 1.12), diminishing in diameter as they ascend the fossa [30]. The anterior deep temporal artery is located no closer than 1.8 cm from the orbital rim. A superficial plexus of veins, if not apparent through the temporal skin, can be better visualized and marked once engorged by placing the head in a forward position. The large middle temporal vein lies within the substance of the temporalis muscle 1 cm above the palpable zygomatic arch (Figure 1.13).

A single injection point *on temporal bone* is planned, 1 cm up the temporal crest and 1 cm lateral following the SOR. Digital pressure confirms the

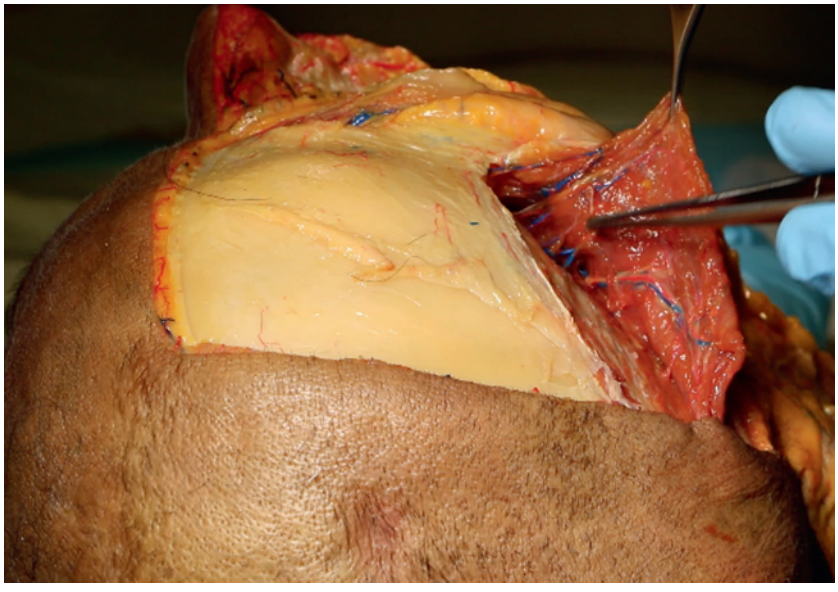


Figure 1.12 The anterior and posterior deep temporal arteries are shown on the deep surface of the temporalis muscle. The anterior deep temporal artery is located no closer than 1.8 cm to the lateral orbital rim.

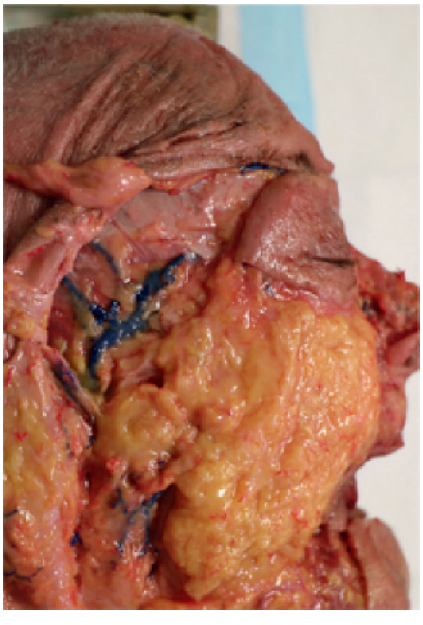


Figure 1.13 The middle temporal vein is consistently found running 1 cm above and parallel to the zygomatic arch. The transverse facial artery can be seen emerging from the parotid gland below the level of the zygomatic arch.

absence of a pulse at the injection site, and any obvious veins are avoided. A highly viscous (cohesive) or stiff (high G′) filler will ride up through the thin fibres of the temporalis muscle in this region, reaching the undersurface of the deep temporal fascia. Aspiration prior to injection is suggested, although the absence of blood reflux is not a guarantee of extravascular location of the needle tip. Maintaining the tip of the needle on bone is crucial to avoiding inadvertent intravascular injection. This location, selected high up near the temporal crest where the muscle fibres are sparse, remains relatively avascular in that any terminal branches of the more posterior deep temporal vessels are of no relative consequence to injection. The anterior deep temporal artery should not be violated as it is located 1.8 cm posterior to the crest. Slow steady injection results in subsequent spread of product which occurs in the less resistant plane between the overlying deep temporal fascia and the underlying muscle, extending circumferentially in all directions but limited medially by the fusion of the fascia at the temporal crest, and similarly inferiorly at the orbital rim. Positioning a flat finger posterior to the needle with the free hand during injection prevents the spread of filler beneath the hair-bearing skin where it has no aesthetic import. Further propagation of the filler will then result in the desirable inferolateral direction towards the arch of the zygoma (Figure 1.14). Maintaining the tip of the needle on bone is crucial to avoiding inadvertent intravascular

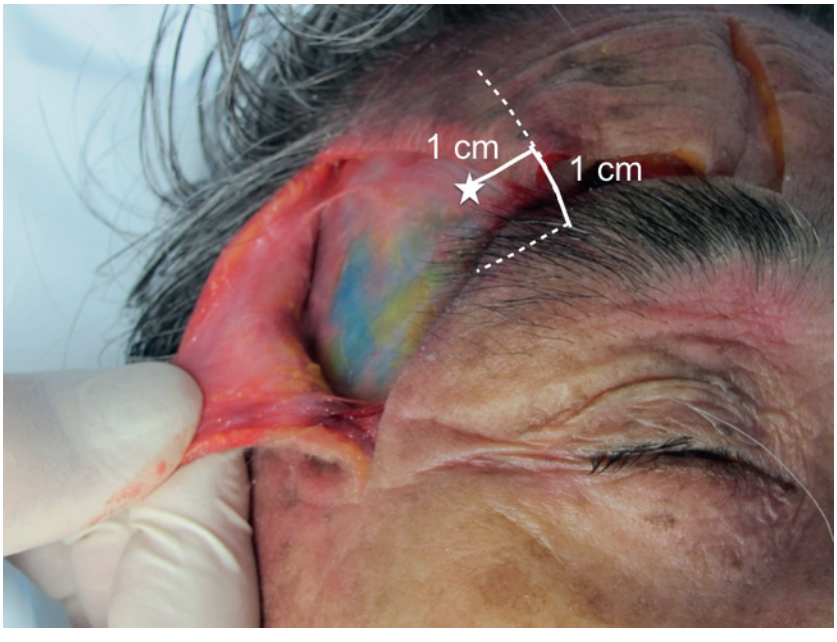


Figure 1.14 Temple injection of dyed product at the starred point (1 cm up, 1 cm over) on bone results in spread throughout the aesthetic temporal fossa.

injection. It is advisable to maintain pressure on the region for several minutes once the needle has been withdrawn, regardless of the appearance of blood at the skin's surface. This is because skewering of a deeper vessel (ex. the sentinel vein) may have occurred, and if not controlled at the time of treatment, the resultant bruising will likely appear in 24–48 hours. Inadvertent skewering of a more superficial artery or vein will result in deposition of product deep to the vessel on bone, and should limit any adverse events to possible bruising in the area. Again, in this region of the fossa, it is mandatory to be close to the TFL (1 cm lateral), anterior (1 cm superior to the SOR) and absolutely deep (the needle must be touching bone) to deposit filler in this relatively avascular plane that is devoid of any significant vessels. It cannot be overemphasized that maintaining the needle on bone during the entire *slow* injection period is fundamental for a safe outcome. It should be noted that a more inferior location for deep injection in the temple, where temporal bone is thinnest near the osseous suture line, has been associated with intracranial violation and deposition of product with a blunt cannula.

Superficial treatment of temporal hollows is also possible with the use of a lower G' product through a blunt cannula, preferably in a direction perpendicular to the superficial temporal arterial branches. Sharp needle penetration into the deeper portion of the temporal fossa above the zygomatic arch is strictly contraindicated, due to the presence of the branches of the second portion of the internal maxillary artery in the sphenopalatine fossa, the embolization of which has led to necrosis of the ipsilateral palate.

1.5 The Middle and Lower Face

The main arterial supply to the lower 2/3 of the face is via the facial artery, a branch of the external carotid artery in the upper neck. This artery courses medially below the jaw to cross the mandible 1 cm medial (i.e. anterior) to the anterior border of the masseter muscle, where its pulsations can be easily palpated. The artery then ascends the lower face in a tortuous course towards the pyriform region of the maxilla at the nasal alar base (Figures 1.15 and 1.16). The tortuosity is understandable, as a spring-like action is necessary to allow the artery to lengthen during wide opening of the mouth. The facial vein, on the other hand, although the thickest-walled vein in the face, is more elastic than its arterial counterpart, and takes a more direct course from the central face, lying posterior to the artery and coursing through the anterior belly of the masseter muscle. Both the facial artery and vein are located deep on the buccinator muscle lining the inside of the cheek, coursing beneath the mimetic muscles risorius and zygomaticus major, and either above or below zygomaticus minor.

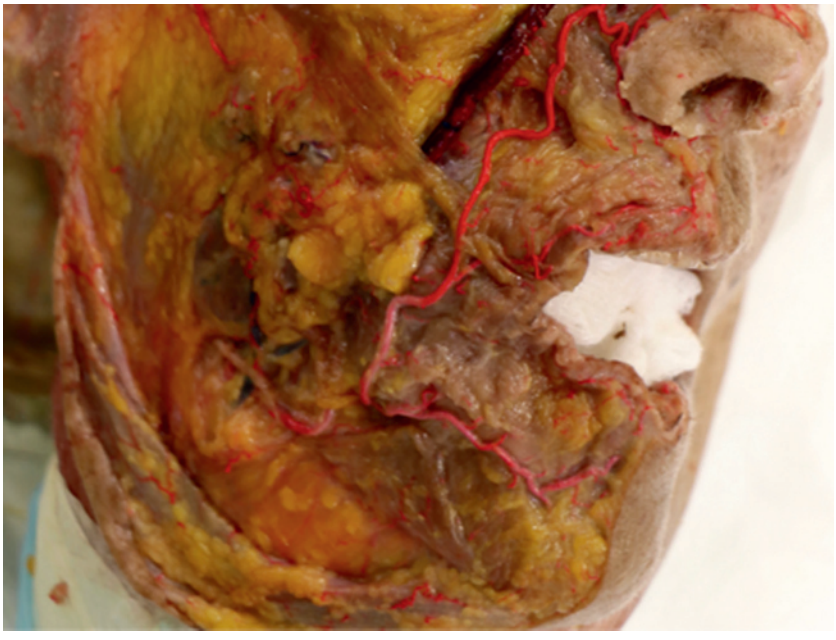


Figure 1.15 The tortuous facial artery is easily identified crossing the mandible medial to the anterior border of the masseter as it climbs towards the pyriform region of the maxilla. Occasionally, this artery may be found medial to the nasolabial fold in the pyriform fossa (see text).



Figure 1.16 The parotid gland and Stensen's duct, which is topographically located on a line drawn from the tragal notch of the ear to 1 cm above the oral commissure. The tortuous facial artery and straight vein crossing the border of the mandible are clearly displayed.

The transverse facial artery, originating from the superficial temporal artery below the zygomatic arch, exits the upper parotid gland as the emerging artery, and divides into a superficial (superior) and deep (inferior) branch (Figure 1.13). The submalar deep branch contributes to the vascular supply of the muscles of the lower face. Therefore, injections in the submalar region, below the palpable inferior border of the maxilla, are best performed superficially in the subcutaneous plane, with either needle or cannula, to avoid encountering these deeper major vessels. Pinching out of the superficial cheek fat with injection allows the distraction of the subcutaneous tissue without drawing up the deeper vessels, adding an additional level of security. This also allows for deposition of product without violating the parotid gland or its duct (Figure 1.16).

When jawline contouring is contemplated, *superficial* introduction of the cannula 1 cm medial to the anterior border of the masseter (i.e. overlying the facial artery) has the advantage of advancing the cannula along the jawline in either direction while moving away from the underlying danger. In the authors' opinions, this can be more safely performed above the parotid fascia posteriorly. Violation of the parotid with intragland injection should be avoided to prevent untoward complications of abscess, cyst, and sinus formation or Stenson's duct obstruction. Deposition of product on the boney gonial angle may be desirable to define or re-establish the L-shaped contour of the posterior jawline which is often lost with ageing (more commonly in the female). The tip of the needle (direct approach) or cannula (indirect approach) must, in all cases, be directly opposed to and remain on the periosteum of the gonial angle during injection.

The zygomaticomalar region of the cheek receives its arterial supply from the transverse facial artery (Figure 1.13). This branch of the superficial temporal artery courses medially through the substance of the parotid gland, exiting as the superficial and deep emerging arteries. The deep emerging artery courses inferiorly to supply the muscles of the lower face, deep to the superficial zone of injection described above. The superficial emerging artery arborizes extensively over the lateral cheek, similar to the plexus of veins located in the area. Injections in this region overlying palpable bone, bounded by the lateral iris medially and the inferior border of the maxilla below, can be performed superficial or deep, appreciating the increased risk of bruising possible. Aspiration and slow injection is particularly indicated in this region due to the presence of the zygomaticotemporal and zygomaticofacial foramina, whose arteries are the terminations of the lacrimal (and thus ophthalmic) vessels. The region also houses the zygomatic ligament, a true oseo-cutaneous ligament (Figure 1.17). Deposit of filler into the deeper non-mobile fat pads (ex. SOOF) underlying the

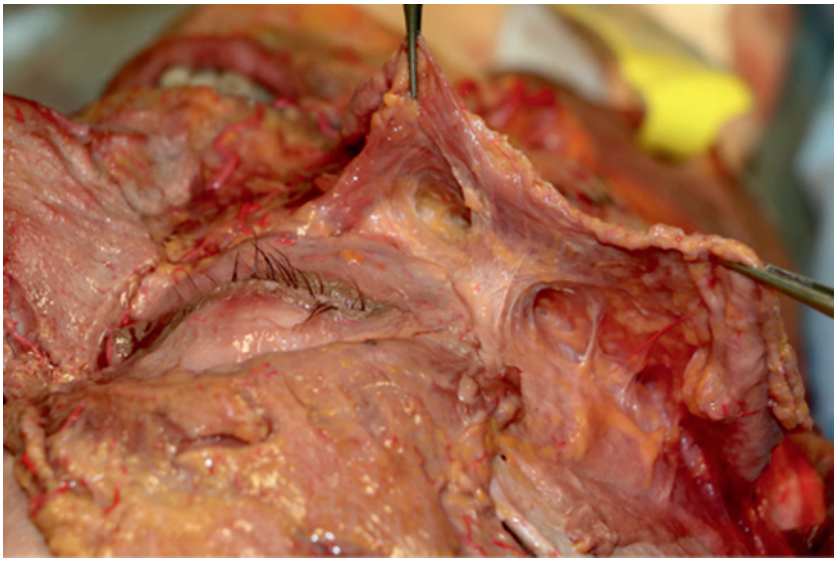


Figure 1.17 The zygomatic ligament (white) as viewed from above.

muscles of this area and pre-zygomatic space (Figure 1.18) is preferred in cases where the overlying soft tissue/skin envelope integrity is compromised (ex. the more mature patient) and unable to support the added weight of product over the long term.

The anteromedial cheek, commonly volume-deficient in the Asian population, is a high-risk area for subdermal filler due to the presence of the infraorbital artery (IOA) emerging 5–8 mm below the infraorbital rim. The artery's exit from the skull is more variable than its SO counterpart in that it can be located slightly more lateral. Often textbooks incorrectly describe it as lying on the 'mid pupillary line'. In fact, its exact location is easy to determine clinically, because the examiners finger will easily fall into the groove immediately medial to the medial border of the zygoma, about 5–8 mm below the infraorbital rim. Due to the IOA's variable depth as it supplies the overlying muscles and soft tissues, and its connections to the angular, facial and supratrochlear arteries (Figure 1.19), it is advisable to proceed with extreme caution when addressing aesthetic concerns in this region. The infraorbital foramen is angled inferiorly, commonly with a hood of bone protecting its superior margin. This exposes the artery in its foramen to deep inferior approach injections, which should be avoided. Utilization of blunt cannulas of significant gauge is highly recommended from a lateral approach while carefully monitoring patient pain and skin blanching during the procedure.



Figure 1.18 The prezygomatic space, seen here, is the preferred location for malar enhancement with fillers.

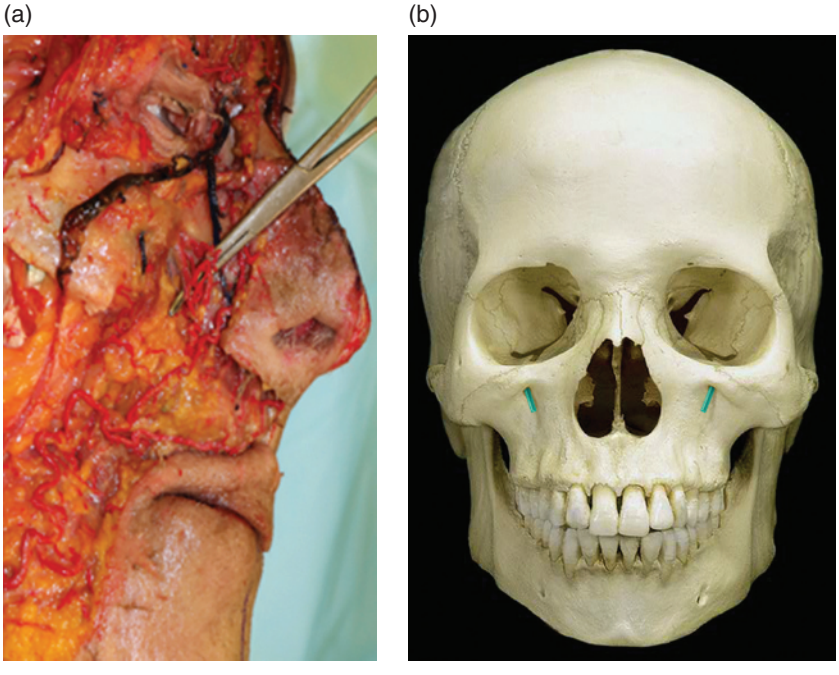


Figure 1.19 (a) The infraorbital artery is indicated and its vast communication with the surrounding vessels; (b) the angulated form of the infraorbital foramen, its overlying protective hood of bone, and the funnel like shape of the maxilla preclude an inferior approach to this region (see text).

The region of the tear trough superior to the infraorbital foramen is amenable to deep correction on the bone of the rim, although as before mentioned, this region is probably best approached lateral to the foramen, milking the product medially to the desired location. Prolonged, unresolving oedema of the infraorbital region post treatment is pathognomonic of inadvertent deposition of product behind the orbital septum, and ultimately requires hyaluronidase therapy for its resolution. Superficial subdermal 'sheeting' of filler that spreads thinly (so as to minimize the risk of Tyndall effect) may also be valuable with minimal risk of intravascular consequence.

The malar septum lies around the orbit, in the shape of a funnel, both above and below the orbit, ending deeply on the orbital margin superiorly and inferiorly, and about 5 mm lateral to the lateral canthal area on the bone. This funnel creates a potential site of complication if filler is injected superficially in this region, since it will cause premalar edema, which is just as problematic as injection deep to the orbital septum. In fact, this error is far more common, and the authors see this adverse event frequently. Treatment is again with hyaluronidase, but the problem is best avoided by injecting deep to the malar septum, and not in it. Only a tiny amount of HA filler deposited in this space (for example, a tiny droplet when withdrawing the needle) is enough to trigger the swelling.

The pyriform fossa of the maxilla, topographically located at the upper limit of the nasolabial fold and underlying the nasal alar base, is a frequent target of the injection specialist. Injections are typically placed under the alar base, medial to Ristow's space and avoiding the deep medial cheek fat compartment (Figure 1.20). Ageing of the midface is typified by retrusion of this region with splaying of the overlying alae and drooping of the nasal tip. Instillation of higher G' product as support will not only flatten the unsightly gutter, but also marginally narrow the alar base and slightly increase nasal tip projection. Unfortunately, this remains the most frequent area of intravascular injury due to the proximity of the facial artery and its frequent anastomosis to the angular artery [31]. Although most often located laterally, the subcutaneous facial/angular artery in the pyriform region (Figure 1.15) can variably course medial to the nasolabial fold in approximately 5% of Caucasians and 20% of Asians. It is therefore imperative to avoid the subcutaneous plane and place filler either intradermally for structure, or on bone for lifting effect.

Dorsal nasal enhancement is covered in more detail in Chapter 7. It is best accomplished on bone or cartilage in the midline, due to the more laterally located paired dorsal nasal arteries (Figure 1.21). Caution is paramount, especially in the radix area where the presence of an intercanthal artery within the procerus muscle can cause bruising or a more serious intravascular adverse event. Intradermal injections

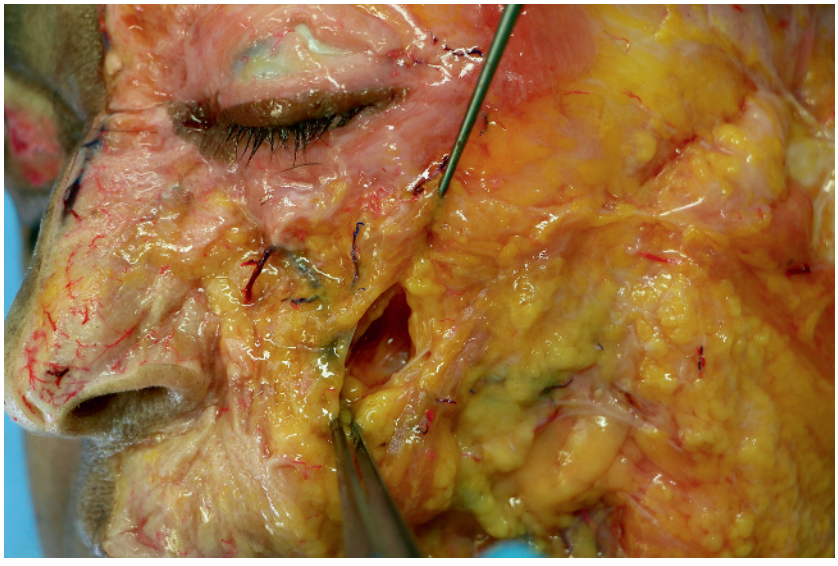


Figure 1.20 The deep medial cheek fat compartment and Ristow's space.

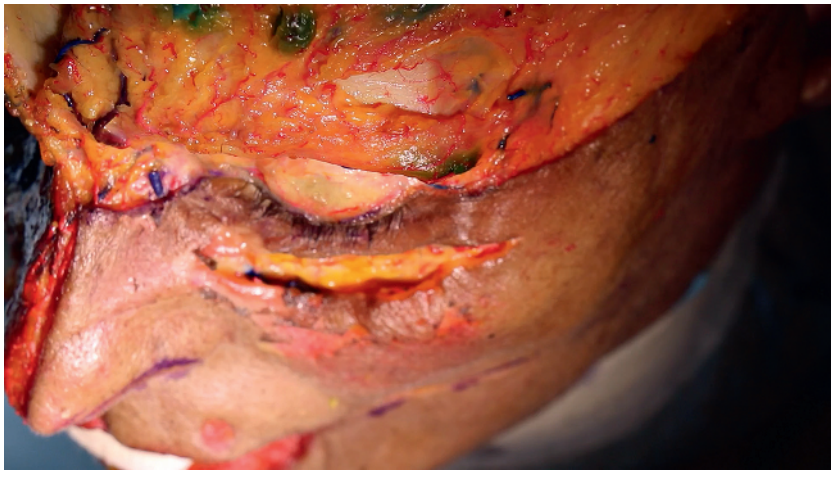


Figure 1.21 The paired dorsal nasal arteries are located off the midline.

in the nose are not advisable due to the abundance of end-arteries, the dermal occlusion of which may cause small ulcerations and skin necrosis. Every crease overlies an arterial vessel, thereby dictating off-crease puncture when nasal contouring to avoid excessive nasal swelling [32]. Deposition of filler with high G′ for the retracted columella is best performed on the nasal spine deep to the columellar artery located above the bone (Figure 1.22).



Figure 1.22 The columellar artery of the nose.

The perioral and chin region are supplied primarily by the paired superior (SLA) and inferior (ILA) labial arteries, and branches of the mental artery as it exits its foramen in the mandible. Although located most commonly opposite the first premolar of the lower dentition, the foramen is highly variable and can be found more lateral along the jawbone. The SLA and ILA are branches of the facial artery as it ascends approximately 1.5 cm lateral to the oral commissure (Figure 1.23). The very tortuous SLA has a mean external diameter of 1.8 mm and often has its origin above the labial commissure in 75% of patients. The less tortuous ILA has a mean external diameter of 1.4 mm and an origin below the labial commissure in 40% of patients. A common trunk origin for the SLA and ILA is found in 30% of patients above or below the oral commissure. The ILA can occasionally be found underlying the labiomental crease of the chin, thus explaining the reported case of blindness as a result of inadvertent intravascular injection for chin enhancement [21].

The lower lip blood supply is often augmented by the presence of the horizontal and vertical labial arteries originating from the facial artery, as well as the mental artery, thereby explaining the reduced incidence of clinically observed lower lip vascular injury (Figure 1.24). The SLA and ILA run on the undersurface of the lips both submucosally and intramuscularly. The vessels appear like dolphins, darting in and out of the muscle to anastomose with the opposite artery in the midline, but always no deeper than 4 mm beneath the vermilion at the level of its border with the white lip. Perioral rhytids and lip enhancement injections are therefore

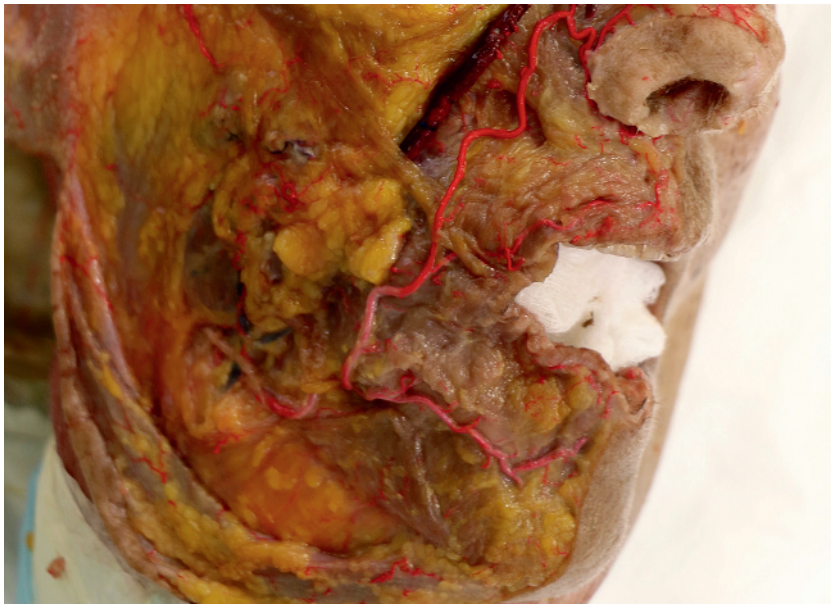


Figure 1.23 The superior and inferior labial arteries. Note that the inferior labial artery can be located quite low under the labiomental crease of the chin.

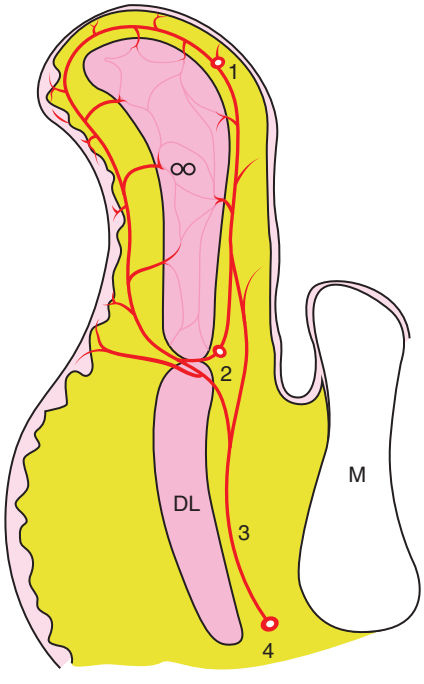


Figure 1.24 The lower lip arterial supply. 1. Inferior labial artery. 2. Horizontal labial artery. 3. Ascending vertical branch of the mental artery. 4. Mental artery.

performed with higher safety in the more superficial planes (i.e. dermal, subdermal, submucosal, and superficial intramuscular), above the underlying vessels [33].

Chin zone contouring (prejowl sulci; 3-D augmentation) can be accomplished at multiple depths, but most safely on bone in the midline.

1.6 Conclusion

The word anatomy is derived from the Greek word ana (on, upon) and temno (I cut); however, when dealing with non-surgical facial enhancement, there is obviously no cutting involved. Facial injection anatomy is therefore unique, and not typical of the 'surgical' anatomy to which surgeons are accustomed.

It is crucial for the injection specialist to have a complete appreciation of the *depth* of his needle tip in order to limit intravascular deposition of product. Once the needle penetrates the skin and disappears into the underlying tissue, it is impossible to avoid piercing a vessel. Facial vessels are extremely variable as they course and anastomose across the face from their origins to their terminal branches, but what can be fairly consistent is the depth at which they travel in defined zones.

Understanding the injection anatomy of the region and whether the needle tip resides in a vessel, above it, or below it, is critical to limiting the disastrous outcomes occasionally seen with facial fillers.

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