

PART ONE

ANATOMY
AND
THE AGING
PROCESS

CHAPTER 1

Anatomy and the Aging Changes of the Face and Neck

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With aging, all facial elements undergo specific modifications. This results in an appearance typical for a specific age group, well recognizable by others. These signs of aging, most of which are demonstrated by Figure 1.1, which shows, split-face, the same man at ages twenty-three and fifty-one, include the following:

- loss of forehead skin elasticity and subcutaneous fat, which, along with increased depressor muscles tonus, results in apparent skin redundancy and pronounced frown lines
- brow ptosis
- wider and deeper orbital appearance
- distortion of the superolateral upper orbital rim with excess upper eyelid skin and fat (hooding)
- distortion of the inferomedial orbital rim: protrusion and sagging of fat, muscles, and skin
- prominent nasolabial folds
- deeper and more vertically sloped nasolabial crease
- loss of jawline contour with formation of jowls due to skin laxity and fat ptosis
- loss of submental cervical angle: midline platysma separation and band formation, skin ptosis

These changes result in loss of the arches of the face that define the youthful appearance noted in Figure 1.2.

Such massive structural and morphological changes involve all the tissues, but each in a different way. Laxity of the skin and subcutaneous tissues accounts only for a part. Loss of volume, due to fat atrophy and bone remodeling, also contributes significantly to the aging process. Let us examine the roles of bone, fat, and muscle changes during the aging process and their consequences on appearance.

ROLE OF BONE

Human bone goes through remodeling throughout the lifetime. Maximum bone mass is reached between fifteen and

twenty-five years of age, at which age, women have about 20% less bone mass than men. By the age of sixty, 25% of bone mass will be lost. There is an acceleration of bone disappearance in women at menopause, then a slowing down of the process, while men show a steady decrease in bone mass. Men catch up gradually to women, and both end with the same loss.

Several studies have shown that craniofacial bones do not undergo disappearance, but rather, they show continuous growth throughout life, with enlargement of facial height and width. However, bone of dermal origin, like facial bone, differs in its evolution from bone of endoskeletal origin (postcranial skeleton). They present with permanent zones of bone deposition or resorption that modify their shape, as can be seen with the global transverse enlargement at the malar and zygomatic arch levels.

Studies of the evolution of facial dimensions and structures with age give contradictory results. In transversal studies (Pessa 2000; Shaw and Kahn 2007), individuals of different age groups are studied at a given time. The authors' conclusions are that there is a marked decrease of the midfacial vertical maxillary dimension and posterior sliding, associated with a deepening of the inner part of the inferior orbital rim. This allows Pessa (2000) to describe a clockwise rotation of the face.

These conclusions are totally contradicted by a longitudinal study based on Behrents's atlas, from the original work of Bolton, who followed almost six thousand subjects yearly from 1928 to the 1970s. Levine et al. (2003) states that bone resorption at the orbital rim and maxilla ridge is responsible, through disappearance of the maxillary concavity, for an increase in the vertical length of the maxilla with anterior maxillary wall displacement, under the forces generated by the descent of the soft tissues of the midface.

Clinical and therapeutic conclusions are therefore completely different. In the first case, bone posterior displacement would be responsible for the sliding downward and forward of the malar fat pad, increasing the thickness and

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FIGURE 1.1: *The signs of aging: same man, age twenty-three and age fifty-one.*



FIGURE 1.2: *Loss of convexities of the once youthful curvatures of the face.*

volume of the nasolabial fold, thus inducing a reactional muscular retraction. Youthful appearance would then be best achieved by restoring the bone dimensions of youth, with bone apposition or osteotomies and deep, subperiosteal face lifts.

In contrast, according to Levine et al. (2003), it is the loss of soft tissue volume and position, unmasking underlying skeletal contours, that is responsible for the aging appearance. Muscle tone is increased by the pull of soft tissues and the lengthening of underlying bones. Rejuvenation will be best achieved by restoring soft tissue volume and repositioning, hence the role of facial fillers and superficial face lifts.

We know that bone remodeling is induced by forces applied on the periosteum, and the rotation effect described might be an illusion, as it could be based, not on the backward movement of the lower maxilla, but on its vertical descent. This could explain why we do not see the functional consequences such a clockwise rotation should have on the nasopharyngeal area, which should be diminished and impaired.

The true consequences of bone remodeling on facial aging are still to be demonstrated and quantified. Longi-

tudinal studies are currently under way to try to shed light on this very important issue.

ROLE OF FAT

There are two layers of subcutaneous fat. The first is a superficial one, between the dermis and fascia superficialis (upper layer of the SMAS), corresponding to the hypodermis and being fairly evenly distributed. Its role is protective, mechanically and chemically. A deeper layer, deep in the SMAS, is found around or under the muscles. It improves the gliding of tissue planes during muscle movement (Dumont et al. 2007). These layers are further divided into several fat compartments well limited by septa, which can fuse in a membrane corresponding to the facial retaining ligaments and within which can be found the feeding vessels to the skin (Rohrich and Pessa 2007).

Aging seems to occur differently between these layers. The deep, perimuscular fat layer is not very much affected by variations in weight of individuals, whereas the volume of the superficial, subcutaneous layer relates more to overall body fat content and weight variations. The variations in fat volume present in the superficial compartments are

responsible for the thin (skinny) or heavy (fatty) appearance of an aging face.

Muscles also play a part in fat distribution, as with aging, fat is displaced from being deep to superficial around the muscles in the deep layer, and this accentuates fat and skin sliding in the superficial layer, and all of this in a different way for each compartment.

Fat distribution, including fat distribution around the muscles, is very important to understand. The true goal is to re-create the appearance of youth, not just to blindly add volume to an aging face. When injecting a dermal or subdermal filler, including autologous fat, to produce the desired effect, the depth of this injection is of primary importance to re-creating the appearance of youth, and not just its volume.

ROLE OF MUSCLES

Although it seems common sense that there is lengthening of facial muscles with age, numerous studies show that the actual muscle tone increases, with shortening of the amplitude of movement, muscle tone at rest staying closer to maximum contracture tone. According to some authors, this is due to a reactional adaptation to bone resorption, and according to others, it is due to the lengthening of the bone bases. There is also a marked decrease in muscle mass, well proven in the lower eyelid. It is possible that this might increase the tone of the remaining muscle mass. Whatever the cause, Le Louarn (2007) has well demonstrated this tightening of the muscles of the face, the permanent contracture of which results in shifting of the underlying fat, accentuation of skin creases, and permanent skin wrinkling.

This is well observed in facial nerve palsy, in which the absence of movement results in the disappearance of wrinkles and the nasolabial fold. The best way to treat this contracture could be through the use of botulinum toxin, but it cannot be used easily or without unpleasant side effects everywhere in the face.

CONSEQUENCES FOR EACH FACIAL AREA

Forehead

There is bone loss with a verticalization of the forehead and a flattening of the supraorbital ridge. This results in an apparent lengthening of the forehead skin, further increased by recession of the hairline. The thinning of the subcutaneous fat in all compartments contributes to the thinning of the skin and the marked hollowing of the temple area. Increased muscular tone results in permanent wrinkling, horizontal lines by the frontalis, and glabellar frown lines by the corrugators and superior orbicularis oculi.

Botulinum toxin is efficient for treating the wrinkles, with care being taken to leave the inferior third of the frontalis muscle untreated. If skin laxity is important, with

marked descent of the eyebrows, a forehead lift is indicated, as releasing the muscle contracture will further accentuate drooping eyebrows. Superficial fillers will give good results for suprazygomatic and temple hollowing.

Upper and Lateral Orbit

Bone changes are major here, with flattening of the supra-orbital ridge, bone loss at the nasion, and oblique lengthening of the orbital diameter, with bone resorption on the superolateral rim contributing to brow ptosis. A combination of all factors – bone resorption, skin relaxation, muscle contracture and volume loss, and fat displacement – results in ptosis of the brow and upper eyelid, with hollowing of the upper part of the eyelid and protrusion of the inner orbital fat, medial to the levator muscle. Contracture of the lateral orbicularis oculi muscle results not only in crow's feet, but also, at its lower external portion, in marked displacement of the suborbicularis oculi fat pad (SOOF).

Lower Eyelid and Nasolabial Fold

There is marked bone resorption on the inferomedial orbital rim and major changes of the anterior maxilla, with resorption and verticalization. Contracture of the palpebral part of the orbicularis oculi is responsible for wrinkling of the palpebral skin, while the orbital, peripheral part of the muscle pushes away the deep fat pads, some parts of the muscle showing marked volume loss. The orbital septum slackens, together with the orbicularis retaining ligament, resulting in orbital fat herniation and lower-eyelid lengthening. The subcutaneous fat superficial to the orbital part of the orbicularis muscle slides downward and inward, deepening the tear trough. It pushes on the nasolabial fat compartment, thus creating or thickening the nasolabial fold and deepening the crease. Laterally, sliding of the SOOF, together with skin and muscle, is stopped by the superficial malar fat compartment, resulting in so-called malar bags.

Orbital rim changes can be treated with deep permanent or long-lasting fillers to replace orbital rim losses, lifting up the brows and filling the tear-trough deformity. Muscle contracture is released with botulinum toxin, but skin excess and displacement need tightening, repositioning, or resection, and fat protrusion is best treated with surgical procedures. For the eyelids proper, in the opinion of the author, there is no indication for superficial fillers since a variety of complications can result from superficial placement of the fillers, whether subcutaneous or injected within the orbicularis muscle.

Displaced malar and lower orbital subcutaneous fat should be repositioned surgically. Fillers can only be used to replace volume loss. If fillers are injected superficially without prior repositioning of the sagging deeper tissues, the result is an abnormal-looking cheek and malar volume when smiling. The problem can be avoided by injecting

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the filler deep, under the muscular layer, around the periosteum of the maxilla, so that the filler is not subject to muscle movements. The result is a natural, youthful appearance.

Lips and Perioral Region

Apart from major changes induced by tooth loss, normal aging is characterized by thinning of the lips due to loss of muscle volume; permanent superficial orbicularis oris contracture, resulting in vertical perioral wrinkles; and deepening of the chin crease through the combined effect of muscle and fat volume loss and bone resorption. These changes can be treated with fillers, skin resurfacing, and, if done very carefully, with neurotoxin.

Jowls and Marionette Lines

Marionette lines are due, in their superior part, to the permanent contracture of the depressor anguli oris, with a pull downward of the corner of the mouth, unbalanced by the weakened lip levator muscles. This can be released with botulinum toxin. In their lower part, like the nasolabial fold, they correspond to a ligamentous border separating the ptotic cheek skin and its fairly thick subcutaneous fat from the lip, where there is very little subcutaneous fat. This jowl fat seems to be within a distinct anatomical compartment. Therefore jowl treatment could probably be better achieved by surgery than by filling the lip or by liposuction of the fold.

Lower Face and Neck

Skin and muscle laxity are responsible for a sliding of the teguments of the cheek, with an anterior and downward rotation. As we have seen, the obstacle represented by the labiomental crease will stop this sliding, creating the anterior fold of the jowl. The contracture of the platysma increases this effect and is responsible for the submental vertical folds, or platysmal bands. Platysmaplasty with submental horizontal platysma muscle release and midline suture allows for the creation of a platysmal sling, which defines the cervicomental angle and repositions the medial submental fat pad. Platysmaplasty is even more critical for patients with visible platysmal bands due to a separation of the medial platysma borders. Deep placement of appropriate fillers or implants (subcutaneous or along the periosteum) is an excellent nonsurgical alternative for restoring the jawline and blunt jowls. Placement should be along the mandibular border, at the lower end of the labiomental

crease, corresponding to the ligament of Furnas. Botulinum toxin has been used to release the platysma bands, but results are short-lived, it is very costly, and it can have undesirable side effects; thus it has limited usefulness in this area. Numerous surgical techniques have been described to try to correct the platysmal muscle bands and tighten the skin in the anterior neck region, which means that none can be entirely satisfactory or helpful for all cases. Jowls and lateral neck laxity, however, respond far more predictably to lifting procedures.

The availability of new and improved fillers, neurotoxins, and various tissue tightening and skin resurfacing methods and devices, discussed in this textbook, can provide alternatives or supplementation to classic surgical techniques. In conclusion, a good understanding of anatomy and how it changes with the aging process, and careful analysis of each patient's morphology, is mandatory to define for each individual what will be the best combination of treatments.

SUGGESTED READING

- Levine RA, Garza JR, Wang PTH, et al. Adult facial growth: applications to aesthetic surgery. *Aesthetic Plast. Surg.* 2003;27:265–8.
- Pessa J. An algorithm of facial aging verification of Lambros' theory by three dimensional stereolithography with reference to the pathogenesis of midfacial aging, scleral show and the lateral suborbital trough deformity. *Plast. Reconstr. Surg.* 2000;106:479–88.
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- Shaw RB, Kahn DM. Aging of the midface bony elements: a three-dimensional computed tomographic study. *Plast. Reconstr. Surg.* 2007;119:675–81.

Suggested Reading about Fat

- Coleman SR, Grover R. The anatomy of the aging face: volume loss and changes in 3-dimensional topography. *Aesthetic Surg. J.* 2006;26(Suppl):S4–S9.
- Dumont T, Simon E, Stricker M, et al. Analysis of the implications of the adipose tissue in facial morphology, from a review of the literature and dissections of 10 half faces. *Ann. Chir. Plast. Esthet.* 2007;52:196–205.
- Rohrich RD, Pessa JE. The fat compartments of the face: anatomy and clinical implications for cosmetic surgery. *Plast. Reconstr. Surg.* 2007;119:2219–27.

Suggested Reading about Muscles

- Le Louarn C. Botulinum toxin and the Face Recurve® concept: decreasing resting tone and muscular regeneration. *Ann. Chir. Plast. Esthet.* 2007;52:165–76.

PART TWO

**ANESTHESIA
AND
SEDATION FOR
OFFICE
COSMETIC
PROCEDURES**

CHAPTER 2

Local Anesthetics

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BASICS OF LOCAL ANESTHESIA

Mechanism of Action

Local anesthetics block Na^+ influx during depolarization of the nerve cell membrane. The result is the blockade of the action potential and subsequent anesthesia.

Order of Blockade

The blockade proceeds from pain through temperature, touch, pressure, vibration, proprioception, and motor function:

- First affected are the small, unmyelinated C-type nerve fibers (which transmit pain and temperature).
- Last affected are the largest, myelinated A-type fibers (which transmit pressure sensations and motor fibers).

Patients may have anesthesia but still feel pressure and may have the ability to move because the A-type fibers are unblocked.

Composition

Anesthetics are weak organic bases that exist in two forms:

- The ionized form is an active form that blocks nerve conduction. The ionized form is water-soluble, therefore allowing injection. At physiologic pH, 80% is in the ionized form.
- The nonionized form is the lipid-soluble form that facilitates diffusion into tissues and nerve cell membranes.

Chemical Structure

The chemical structure has three components:

- The aromatic portion is usually composed of a benzene ring (lipophilic).

- The intermediate chain is either an ester or an amide linkage (determines class).
- The amine is hydrophilic (water solubility).

Lipid solubility is important because it enables diffusion through the lipophilic nerve membrane. Lipophilicity is directly related to potency.

Duration of Anesthesia

Duration is directly related to the degree of protein binding of the anesthetic receptors along the nerve cell membrane (determined by amine structure).

Potency

Potency is determined by lipid solubility.

Speed of Onset

Speed of onset relies on the pKa (the fraction of anesthetic that is active at physiologic pH). This is determined by the aromatic structure. An anesthetic with a low pKa will have a rapid onset of action. Alkalinization of the anesthetic solution with sodium bicarbonate will speed the onset of action.

TECHNIQUES FOR PAINLESS ANESTHESIA

- Verbal reassurance and distraction
- If anxious, could consider premedication
 - Ambien 5–10 mg, sl 30 minutes prior (short-acting, 4-hour effect)
 - Valium 5–10 mg, sl 30 minutes prior
 - Demerol, Vistaril
- Mechanical distraction
- Use small-gauge needle (30 gauge) and syringes (larger syringes result in more pressure and increase pain)

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- Cryoanesthesia: fluorethyl, frigiderm, ethyl chloride, ice cube, smart cool (-5°C)
- Warm anesthetic to body temperature
- Inject slowly, first subcutaneously and then dermally
- Inject through previously anesthetized tissue
- Increase pH with bicarbonate 1:10
- Plain lidocaine, if possible
- Preoperative EMLA
- prolongs anesthesia by 100% to 150%
- hemostatic up to 1:500,000 or 1:1,000,000
- available at premixed concentrations of 1:100,000 and 1:200,000 with lidocaine

ADDITIVES TO LOCAL ANESTHETICS

When choosing an additive, one should consider the desired effect and weigh the risk versus the benefits. Additives may potentiate hemostasis and/or prolong or shorten the duration or onset of anesthesia. Because additives alter the pH of the anesthetic, this may result in more or less patient discomfort. Tissue irritation is related to the acidity of the infiltrated solution. Epinephrine and bicarbonate affect acidity and alter the associated discomfort.

- Risks
 - systemic effects
 - possible tissue necrosis, increased if used for ring blocks
 - interacts with tricyclic antidepressants (HTN, tachycardia, ventricular tachycardia) and nonselective beta blockers (propranolol; hypertension and bradycardia)
 - increased risk for rapid absorption in vascular areas (e.g., glans penis)
 - concentrations greater than 1:200,000 are usually not necessary for dermatologic surgery and concentrations greater than 1:100,000 are at increased risk for side effects
 - should not exceed 1 mg (100 mL of 1:100,000)

	pH	Comments
1% lidocaine	5.5–7.0	• close to physiologic pH, resulting in less pain
1% lidocaine + epinephrine	3.5–5.0	• A more acidic pH is necessary to preserve the epinephrine; this results in increased pain
“Buffered” Anesthetic 1% lidocaine + epinephrine (10 parts) with 8.4% sodium bicarbonate (1 part) 10:1 ratio	7.3–7.5 (very close to physiologic pH)	<ul style="list-style-type: none"> • bicarbonate helps neutralizes pH, thereby decreasing pain • bicarbonate decreases hemostatic effect of epinephrine • good for only 1 week because bicarbonate degrades epinephrine, which results in loss of vasoconstriction after 1 week • stable at room temperature for approximately 24 hours (must be refrigerated) • faster onset but decreased duration of anesthesia
“Fresh” Anesthetic: 50 mL 1% lidocaine 0.025 mL of 1:1,000 epinephrine → 1% lidocaine with 1:200,000 epinephrine	50 mL of 1% lidocaine 0.5 mL of 1:1,000 epinephrine → 1% lidocaine with 1:100,000 epinephrine	Close to physiologic pH
		• does not have acidic preparations that manufacturers use to preserve epinephrine

Author's Tips: You may initially use buffered lidocaine + epinephrine to avoid the burning sensation, then follow this with nonbuffered lidocaine + epinephrine to improve hemostasis. So-called fresh anesthetic is preferred.

Additives to Local Anesthetics

Epinephrine

- Benefits
 - has vasoconstrictive effect
 - less anesthetic is required to obtain anesthesia, therefore there is less toxicity from the anesthetic

- pregnancy category C

Hyaluronidase (Widase)

- Mechanism
 - hydrolyzes hyaluronic acid in the connective tissue and facilitates diffusion of the anesthetic

- Benefits
 - increases the spread of anesthesia, decreases the duration of action of the anesthetics because it increases absorption
 - USE: to decrease distortion of the surgical site, the addition of hyaluronidase is useful for nerve blocks and procedures around the orbit
- usual dilution is 150 U in 30 mL of anesthetic
- Risks:
 - potential for allergy – contraindicated in patients with a known allergy to bee stings
 - contains the preservative thimerosal
 - recommended intradermal test dose
 - increases the potential for toxicity of the anesthetic

CLASSIFICATION OF ANESTHETICS

Anesthetics are classified into two groups, depending on the linkage in the intermediate chain: the ester group and the amide group. This structural difference affects metabolism and allergic potential.

Amides

- Two “i”s in their name
- Hydrolyzed in the liver by hepatic microsomal enzymes p450 3A4 (p450 3A4 inhibitors will increase half-life of anesthetic)
- Metabolites are excreted by the kidneys
- Patients with severe liver disease may be at increased risk of amide anesthetic toxicity

Generic name (trade name)	Relative Potency	Onset (min)	Duration (hours)	Max Dose “Plain” (mg, for 70-kg male)	Max Dose “with Epi” (mg, for 70-kg male)	Other
Bupivacaine hydrochloride (Marcaine)	8	2.0–10.0 slow	2–4 long	175	225	<ul style="list-style-type: none"> • cannot be buffered due to precipitation with bicarbonate • more intense injection burn • greater cardiac toxicity than lidocaine: ventricular arrhythmias and cardiovascular collapse • ideal for long procedure (bupivacaine 0.25% ± 1:200,000 epinephrine)
Etidocaine (Duranest)	6	3–5 med	3.0–5.0 long	300	400	
Leubopivacaine hydrochloride (Chirocaine)		2–10	2.0–4.0	150	not available	
Lidocaine (Xylocaine) 0.5%, 1%, 2%	2	Rapid	0.5–2.0 med	350	500 (3,500 dilute)	<ul style="list-style-type: none"> • maximum dose without epi = 4.5 mg/kg • maximum dose with epi = 7.0 mg/kg • tumescent = 55 mg/kg • buffered: 1 mL 8.4% NaHCO₃ + 10 mL anesthetic (increases pH to 7.3) • 2% lidocaine may produce a larger concentration gradient, promoting diffusion into the nerve
Mepivacaine (Carbocaine)		3–20	0.5–2.0	300	500	
Prilocaine hydrochloride (Citanest)	2	5–6	0.5–2.0	400	600	<ul style="list-style-type: none"> • metabolized to orthotoluidine metabolite, causing methemoglobinemia
Prilocaine/lidocaine (EMLA)			short			<ul style="list-style-type: none"> • topical
Ropivacaine (Naropin)		1–15	2.0–6.0	200	not available	
Dibucaine (Nupercaine)			short			<ul style="list-style-type: none"> • topical

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Esters

- metabolized/hydrolyzed by pseudocholinesterases in plasma
- metabolized to PABA and therefore may cross-react with sulfonamides, sulfonureas, PABA, paraphenylene diamine (PPD), PAS, thiazides
- excreted renally (therefore contraindicated with severe renal failure)
- allergic reactions are infrequent but more common than seen with amides

Generic Name (trade name)	Relative Potency	Onset (min)	Duration (hours)	Max Dose "plain" (mg, for 70-kg male)	Max Dose "with Epi" (mg, for 70-kg male)	Other
Benzocaine (Anbesol)			short			<ul style="list-style-type: none"> • topical • may induce methemoglobinemia
Chloroprocaine hydrochloride (Nesacaine)	1	5–6	0.5–2.0	800	1,000	
Procaine (Novocaine)	1	5	1.0–1.5	500	600	<ul style="list-style-type: none"> • risk of methemoglobinemia
Tetracaine (Pontocaine)	8	7	2.0–3.0	100	not available	

Author's Tips:

- Blepharoplasty: 1% lidocaine with epinephrine injected locally
- Lip augmentation with fillers: gingival mucosal block using 1% lidocaine ± epinephrine
- TCA chemical peel: supraorbital, infraorbital, and mental nerve blocks 2–3 mL (amide-type anesthetics; may use 2% lidocaine to enhance diffusion; may use vasoconstrictor [epinephrine 1:200,000] to slow absorption of anesthetic and prolong duration of anesthesia)

Risk of methemoglobinemia: (prilocaine >> lidocaine, procaine [Novocaine], benzocaine)

- Iron molecule in Hg oxidizes from ferrous 2+ to ferric 3+ state, which decreases ability to bind, transport, and deliver oxygen
- Increased risk in premature infants given >500 mg
- Treatment: stop drug, give oxygen, methylene blue 1–2 mg/kg over five minutes (mix 7 mg/kg ± IV glucose)
- Increased risk in patients taking Tylenol, sulfa, dapsone, benzocaine, chloroquine, phenobarbital, phenytoin
- Note that lidocaine and procaine may also cause it, but less commonly: 0.5% = 0.5 g/100 mL → 5 mg/cc 1% = 1g/100 mL → 10 mg/cc 2% = 2 g/100 mL → 20 mg/cc

Tumescent Anesthesia and Tumescent Liposuction Solutions

Tumescent anesthesia

Uses

- liposuction, face lifts, reconstruction, ambulatory phlebectomy, ablative laser resurfacing, hair transplantation, endovenous radiofrequency ablation

Benefits

- increases maximum safe dose of lidocaine to 55 mg/kg
 - dilute epinephrine results in vasoconstriction of subepidermal vessels, resulting in hemostasis and decreased absorption

Formula

Lidocaine 1%	50–100 mL
Epinephrine 1:1,000	1 mL
Sodium bicarbonate 8.4%	10 mL
Normal saline 0.9%	900–950 mL
Hyaluronidase 150 U/mL	6 mL (optional)
Triamcinolone acetonide 40 mg/mL	0.25 mL (optional)

- A final concentration of 0.05% to 0.1% lidocaine with 1:1,000,000 is prepared
- Peak plasma levels of lidocaine at 12 hours postinfusion (CNS toxicity at blood levels of 5–6 µg/mL)