Beauty and Aging

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Introduction

Aging affects all tissues of the body. However, it is at the facial area that more studies have focused on the aging process and on where the medical and surgical aesthetic treatments of rejuvenation are directed. The face does not age as one homogeneous object, but as a result of several dynamic components. Changes that occur with facial aging involve a complex interaction among the bone, skin, soft tissue, support ligaments, and septa. All these multiple phenomena must be considered for their proper correction.

Facial aging is a multifactorial process. Intrinsic aging includes histologic and physiologic changes resulting from cellular apoptosis and other genetically determined processes. Extrinsic aging results from long-term exposure to environmental aggressions like smoking, alcohol, UV radiation, dehydration, inadequate nutrition, extreme temperatures, traumatic damage, chemotherapy, or radiotherapy. The clinical signs of facial aging are related with changes in all structural layers (skin, fat, muscle, bone).

Proper facial analysis is the key to achieving optimal facial rejuvenation results. Undoubtedly, this analysis is based on an adequate knowledge of the underlying anatomy and the clinical implications that anatomy has for facial aging.

Skin

The skin undergoes aging changes such as thinning of the epidermis, collagen loss, and dermal elastosis that result in fine wrinkles, skin spots, and dryness. Aging causes a fragmentation of the dermal collagen matrix. Solar elastosis is the term used to describe the histologic appearance of the photoaged dermal extracellular matrix and is characterized by an accumulation of amorphous, abnormal elastin material surrounding a decreased volume and disorganized array of wavy collagen fibrils. The loss of this extracellular collagen is responsible for the loss of the structural integrity of the matrix and the decreased mechanical tension. There is a disability of fibroblasts to produce and organize new collagen. Hence, treatments that stimulate neocollagenesis can improve the appearance of the aged skin. Both extrinsic and intrinsic aging factors affect the ability of the skin to adjust to the aging loss of underlying soft tissue volume.

Ultraviolet A (UVA) and B (UVB) radiation causes direct and indirect skin damage. UVB light is almost completely absorbed by the epidermis, and thus dermal photodamage is solely caused by UVA. UVA directly induces DNA

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changes and indirectly causes cell damage by creating free radicals, leading to an increase in oxidative stresses and a degradation of the surrounding collagen. The photoaged dermis shows a histological aspect of chronic inflammation. The epidermis undergoes characteristic histological changes with sun damage, leading to increased thickness, slower keratinocyte turnover, and decreased melanocyte counts. However, there are also regions of increased melanocyte concentration, with increased capacity for melanin production and deposition to keratinocytes, which present as solar lentigines [1, 2].

The topography of wrinkles is not arbitrary and lymphatic vessels may be the primary anatomical structures that determine the position and location of cutaneous wrinkles. Repeated skin contractions over a fixed object (vessel or nerve) may lead to a surface configurational change [3].

Treatments that stimulate the production of new collagen include laser, topical retinoic acid, deep chemical peels, hyaluronic acid, collagen, and calcium hydroxyapatite. Stimulation of collagen production may lead to stimulation of fibroblasts by a direct mechanism of fibroplasia, or indirectly through increased extracellular matrix and stretching effect. Therefore, these treatments could both replace collagen and slow its loss [4]. Laser, topical retinoic acid, and peels are useful for treating the solar lentigines. Injection of uncrosslinked hyaluronic acid improves skin hydration.

Facial Fat

The facial adipose tissue is highly compartmentalized by septa that divide the fat in superficial and deep compartments relative to the superficial musculoaponeurotic system (SMAS) or mimetic muscles forming distinct anatomical units [5]. This compartmentalization facilitates the sliding of the facial muscles between compartments during facial motion. The vessels and nerves travel through the septa that form the transition zones between fat compartments. Many of the retaining ligaments that support facial soft tissue originate within the septal barriers between these compartments [6]. In addition, these fat compartments will undergo sequential changes during aging. In a young face the transitions between them are smooth. As we age, these transitions become more marked and furrows appear.

The facial fat can be divided into two layers (superficial and deep):

Superficial Fat Compartments (Fig. 1)

- *Nasolabial fat*: it is the most medial compartment of the cheek and the least modified with age.
- *Superficial cheek fat*:
  - *Medial cheek fat*: it is lateral to the nasolabial fat.
  - *Middle cheek fat*: it is anterior and superficial to the parotid gland. It is located between the medial and lateral superficial compartments of the cheek.
  - *Lateral-temporal cheek fat*: it is the most lateral compartment of the cheek and extends from the temporal region to the beginning of the neck.
- *Forehead compartments*: there are three compartments in the forehead.
  - *Central frontal fat*: it is in the midline region of the forehead and its inferior boundary is the nasal dorsum.
  - *Middle forehead compartments* (left and right): they are located between the central frontal fat and the lateral-temporal cheek fat on each side.
- *Orbital fat compartments*: they are three, inferior, superior, and lateral. The nasolabial, the superficial medial cheek, and the infraorbital fat pads are collectively referred to as the "malar fat."
- *Jowl fat compartments*: the superior and inferior jowl fat pads.

Deep Fat Compartments (Fig. 2)

- *Deep medial cheek fat compartment (DMC)*: it has two portions, one medial and one lateral (DLC). The medial portion is located poste-

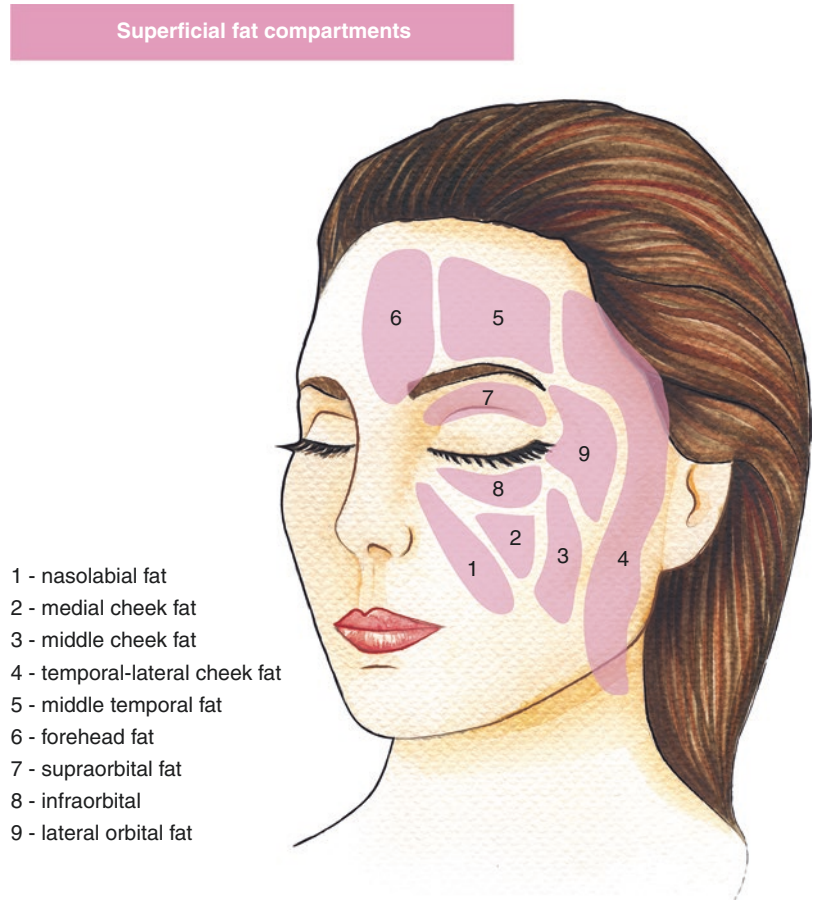

Fig. 1 Superficial facial fat compartments

rior to the nasolabial compartment and is bordered posteriorly by Ristow's space. It is one of the compartments most affected by aging.

- *Suborbicularis oculi fat (SOOF):* It is located behind the orbicularis oculi muscle and is divided into medial and lateral portions. The medial SOOF extends from the medial limbus of the iris to the lateral canthus. The lateral SOOF runs from the lateral canthus to the temporal fat compartment. The inferior boundary of the SOOF is the tear trough.
- *Retro-orbicularis oculi fat (ROOF):* it is located behind the orbicularis oculi muscle in the upper lid.
- *Intraorbital fat*: on the lower eyelid there are three fat pads, internal, middle, and external. On the upper eyelid there are two fat pads, middle and internal.
- *Buccal fat pad:* includes the Bichat pad and its superior extension that courses from the deep paramaxillary space to the superficial, subcutaneous plane inferior to the zygoma.

Aging Changes of the Fat Compartments

Classically the *gravitational theory* had been postulated as responsible for facial aging. This theory proposed that the vertical descent of the facial soft tissue was secondary to the ligamentous attenuation, leading to the sagging appearance of the aging face [7]. Repeated animation of facial mimetic muscles was also thought to contribute to this ligamentous attenuation. After the multiple studies that described the compartmentalization of facial fat, much has been

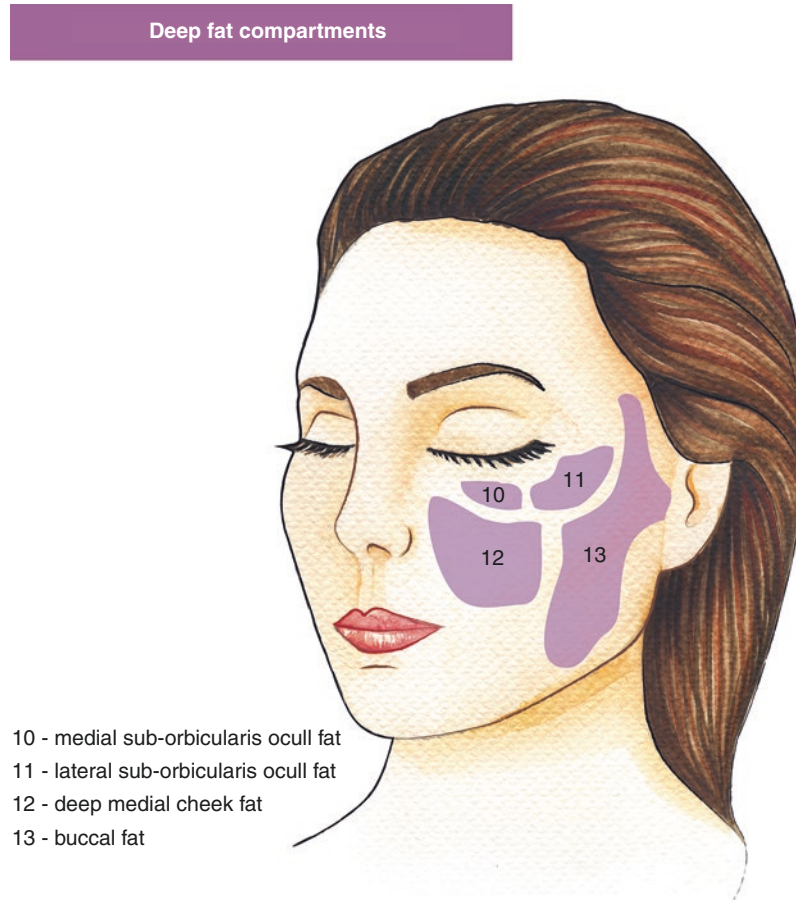

Fig. 2 Deep facial fat compartments

evolved in the theories of facial aging. At present, the most accepted theory for facial soft tissue aging is the *volumetric theory* [5, 8–14]. This theory proposes that the changing morphology of the face, especially the midface, is due to the relative deflation of certain fat pads rather than gravitational descent. Some compartments tend to deflate earlier than others (Fig. 3). These two theories are not mutually exclusive, and facial aging probably reflects a complex morphologic change that involves both elements of gravitational ptosis and volume deflation. Studies have shown that there is a relative hypertrophy of the superficial fat compartments (especially the inferior part of the nasolabial fat) and a pronounced atrophy of the deep compartments (especially DMC and buccal fat pad) with aging [15, 16]. The volumetric theory suggests that selective deflation of the deep fat pads with age leads to loss of support and descent of the overlying superficial fat, thereby contributing to the ptotic appearance of the aging face. This has led to the concept of "pseudoptosis," namely, that loss of volume in one area may lead to the development of folds in a neighboring area [4]. The cheek fat atrophy results in loss of the juvenile convexity of the middle facial third leading to a negative vector (Fig. 4). The negative vector means that the maximum projection point of the cheekbone is posterior to a tangential line to the cornea. Deflation of the deep periorbital fat contributes to the tear trough deformity and the nasojugal groove. The temporal fat compartment atrophy results in depression in the temporal region. The inferior part of the nasolabial compartment hardly suffers atrophy with aging.

But fat compartments not only suffer from deflation, midfacial fat compartments also exhibit an inferior migration and an inferior volume shift within the compartments [1, 15]

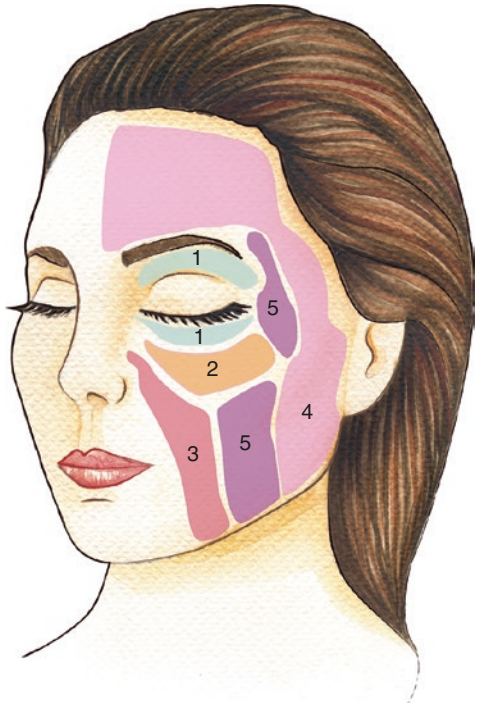

Fig. 3 Some facial fat compartments tend to deflate earlier than others with aging. Sequence of fat compartment deflation observed in facial aging

(Fig. 5). The young face is V-shaped, with a full middle facial third and a lower third with less volume. As the compartments descend, the middle third loses volume and the inferior one gains volume, which causes inversion of the facial youth V (Fig. 6). The inferior migration of fat compartments causes the transitions between compartments to become more marked. In the young face these transitions are smooth and the distances between compartments are short. In the aging face the transitions are pronounced giving rise to furrows (tear trough, nasojugal groove, etc.) and the distances between compartments are lengthened.

It has also been observed that the average adipocyte size is smaller in the deep cheek compartment than in the superficial cheek compartment [17]. Although the reasons for these variations are not clear, it seems that the mechanical environment of the two adipose layers of the midface could contribute to these adipocytes' morphological differences. The superficial compartments are adjacent to the muscles of the facial mime, while the deep compartments are in

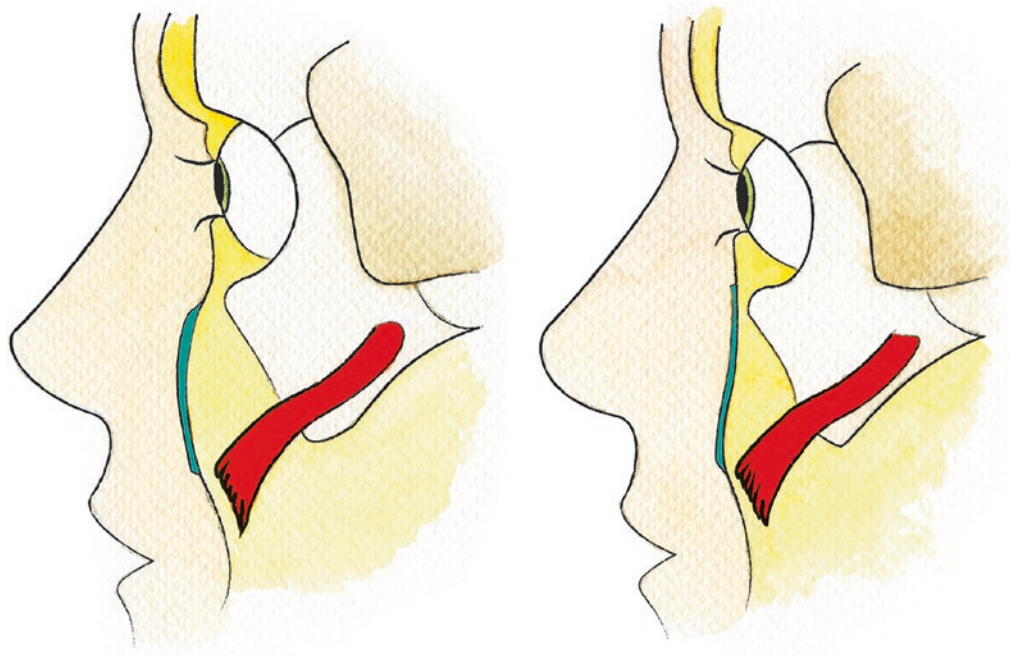

Fig. 4 The cheek fat atrophy results in loss of the juvenile convexity of the middle facial third leading to a negative vector

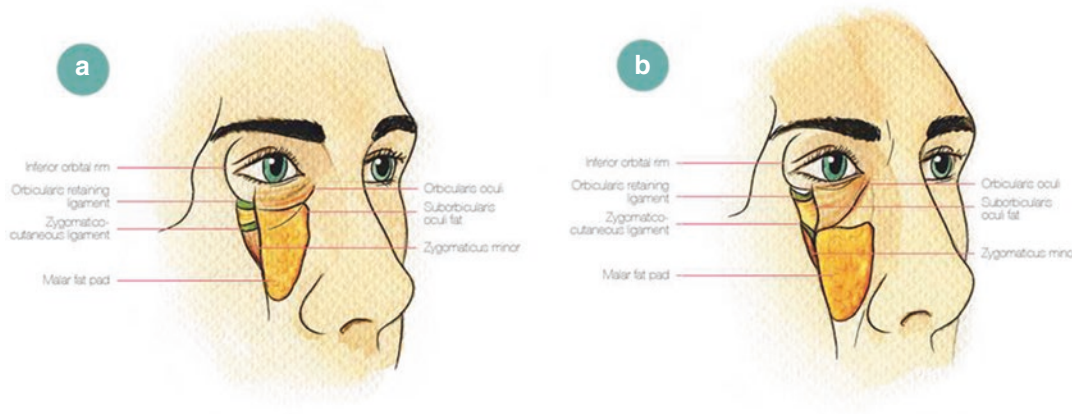

Fig. 5 Facial fat compartments not only suffer from deflation, but midfacial fat compartments also exhibit an inferior migration and an inferior volume shift within the compartments. Young face (**a**) and aged face (**b**)

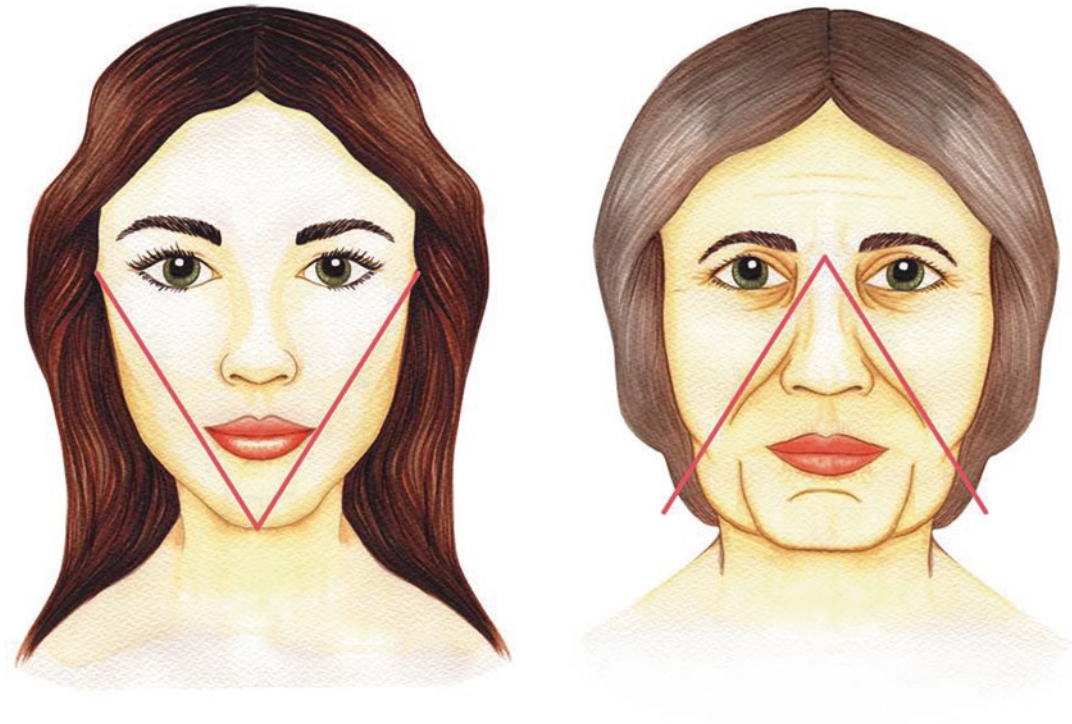

Fig. 6 The young face is V-shaped, with a full middle facial third and a lower third with less volume. As the midfacial fat compartments descend, the middle third loses volume and the inferior one gains volume, which causes inversion of the facial youth V

contact with the facial skeleton. This could be explained by the fact that the continuous compression of the deep compartments against the bone causes them to have a relatively inert role as space-filling interfaces over which the muscles of mastication slide and, hence, tend to more atrophy with time. In contrast, the superficial compartments are closer to the dynamic muscles of facial mime and this could make them more active metabolically [14].

Clinical Implications for Facial Rejuvenation

Due to the relevance that the facial fat compartments have acquired, there has been a change in thinking about facial rejuvenation, evolving from techniques aimed at lifting toward techniques aimed at filling. This evolution has allowed to rejuvenate the face in a more natural way compared to the classical lifting of skin and SMAS under the influence of gravitational theory, which leads to an unnatural appearance. The anatomical knowledge of the facial fat compartments allows us to focus facial rejuvenation techniques more precisely and directly. We can thus selectively increase the volume in the deep deflated compartments creating a more natural look rather than masking the creases of facial aging with superficial multilayering of fat or filler injection in nonspecific malar regions. This also allows economizing the product in the treatments with fillers. We can reduce successfully the nasolabial fold and restore the anterior projection of the cheek by injections into the DMC and Ristow's space [10]. The harsh transition between the lid-cheek junction and the tear trough deformity can be improved by injecting the medial SOOF and the DMC in a supraperiostal way. Augmentation of the lateral portion of the DMC can also increase the anterior cheek projection and smooth the transition between the anterior and lateral cheek. The superficial middle and lateral cheek compartment can be filled for final contour improvement [18].

The inferior migration of fat compartments and the deflation of midface fat lead to the inversion of the facial V. Hence, one should be conservative when filling the lower third (marionette lines, pre-jowls) because if the volume is increased in the lower third, the inversion of the facial V will be aggravated and we will not get an adequate rejuvenation. To restore volume in the middle facial third with fillers or autologous fat is one of the best ways to rejuvenate a face. However, in order to treat sagging of the lower third and jowls, it is preferable to perform treatments aimed at lifting and not only filling (threads, surgical facelift).

Muscle

The fat is positioned both above and below the facial mimetic muscles acting as an effective mechanical sliding plane. Over time, repeated contraction of the facial muscles contributes to changes in the facial fat distribution, expelling the underlying deep fat from beneath the muscle plane. This mechanism leads to a loss of the youthful curvilinear contour and an increase in the resting tone of the muscles. This dynamic muscular effect could explain why deep fat diminishes in favor of superficial fat overtime. With age, the facial mimetic muscles gradually straighten, changing its conformation from a broad convexity in the young face toward a rectilinear flatness in the older one. This convex contour becomes rectilinear as the underlying deep fat is expelled from behind the muscles and the superficial fat increases. The amplitude of movement of the

muscle also is greater in youth but it diminishes with aging and the face acquires a more rigid aspect [19, 20]. The contraction of the muscles of the facial mime is responsible for the appearance of expression wrinkles such as crow's feet, glabellar, and frontal wrinkles. The permanent contractures result in permanent skin wrinkling with a transformation of dynamic facial lines to static facial lines. This increased muscle resting tone with age explains why botulinum toxin treatments are so effective in facial rejuvenation. Young people usually have less strength in the depressor muscles like procerus, corrugators, and orbicularis oculi than in the levators like frontal muscle, and older people have a relatively higher strength in the depressor muscles than in the frontal muscle.

Facial Ligaments

The facial ligaments are composed of collagens, proteoglycans, glycosaminoglycans, and water. The major ligaments are robust and do not undergo significant primary aging changes in their passage from their deep origin to the SMAS. Most of the ligament change is in the multiple finer retinacular ligament branches from the SMAS through the subcutaneous layer to the dermis, which are more prone to being weakened over time by repetitive movement [18, 21, 22]. The most affected by aging are the zygomatic ligament, the orbital retaining ligament, and the mandibular retaining ligament.

The aging bony changes affect the points of origin and the firm adhesions of the ligaments to the skin, and other adjacent structures are getting affected as the position of the ligaments and thus their course are getting altered. The stability of the ligaments that serves as a hammock for the fat within each compartment (superficial or deep) show fatigue and bend along their course, promoting the appearance of sagging of the respective fat compartment and contributing to the appearance of the tear trough deformity, malar bags, and jowls.

Bone

Aging changes occur not only in the facial soft tissues but also in the underlying bony structure. Craniofacial bony remodeling is an important contributor to the facial aging process. The bony facial skeleton serves as the scaffolding for the overlying soft tissue, providing the framework on which the soft tissue envelope drapes. Selective bone resorption occurs in specific areas [9, 23–27]. The most relevant aging changes are bone resorption of the orbit, maxilla, and mandible (Fig. 7). There is an increase in orbital aperture size, more specifically, an increase in height of the superomedial and inferolateral orbital rim. The glabellar and the maxillary angles decrease with age, and there is a posterior displacement of the maxilla and a loss of its projection (Fig. 8). The pyriform aperture area increases over time causing a posterior displacement of the columella, the lateral crura, and the alar base. The anterior nasal spine also recedes, contributing to retraction of the columella, the drop of the nasal tip, and the apparent

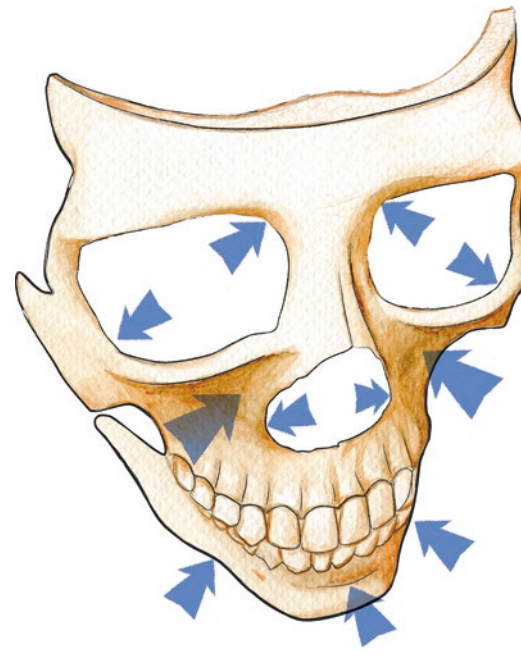

Fig. 7 One of the most relevant aging changes is bone resorption of the orbit, maxilla, and mandible

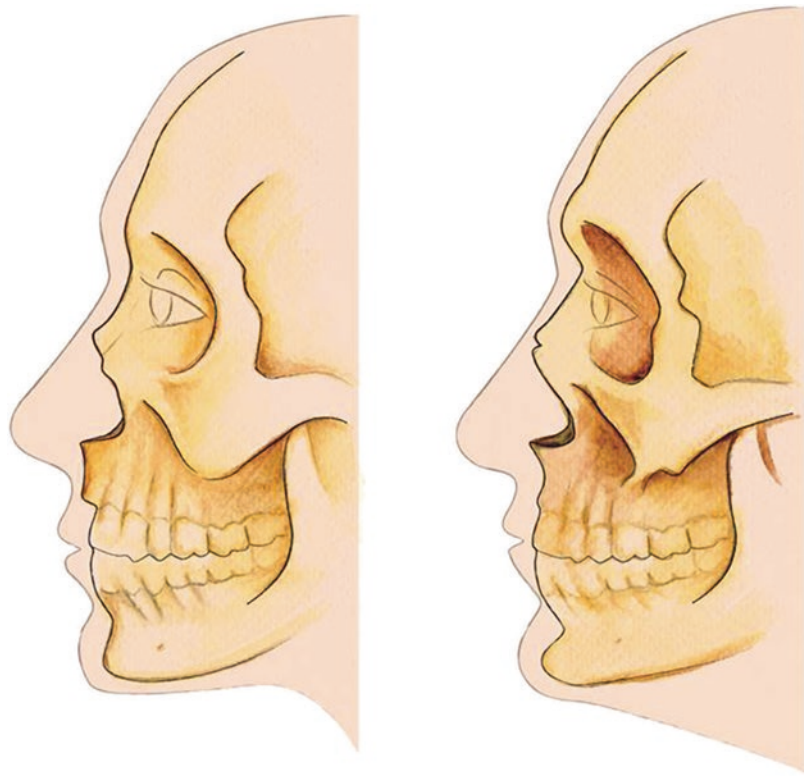

Fig. 8 Age-related bony changes (*right*): enlargement of the orbital and the pyriform apertures, posterior displacement of the maxilla, and shrinking of the mandible

lengthening of the nose. Mandibular ramus height and mandibular body height and length decrease, whereas the mandibular angle increases with age. These changes affect both sexes. Remodeling of the facial bone occurs regardless of the state of dentition, although the loss of dentition accelerates bony resorption of the maxilla and mandible.

Age-related bony remodeling causes a decrease in the space and support available for the soft tissue, especially the fat compartments, resulting in a folding of the soft tissue in a configuration that resembles an accordion [28]. The facial skeleton changes not only affect the overall facial shape but also affect the position of ligaments and septa. The expansion of the infraorbital rim causes an anterior positioning of the orbital septum and a pseudoprolapse of the intraorbital fat pads because the retaining capability of the orbital septum is reduced. The orbicularis retaining ligament loses it horizontal position toward a more inferior inclined alignment, causing loss of stability of the adjacent orbicularis oculi muscle and sagging of the ROOF and the SOOF. Superomedial orbital rim remodeling contributes to the unmasking of the medial upper lid fat, a change currently attributed to weakening of the orbital septum. The changes of the upper half of the orbit result in the soft tissues rolling into the orbit causing brow ptosis and lateral orbit hooding. The bone resorption in maxilla and pyriform area, the ligamentous fatigue, the laxity of the overlying skin, the altered muscle physiology, and gravity cause the loss of stability of the subcutaneous fat compartment superior to the nasolabial sulcus, and the fat has tendency to shift inferiorly. The zygomaticus major and buccinator muscles have strong adhesions toward the skin forming the nasolabial sulcus and border the nasolabial fat compartment inferiorly along with the terminating part of the SMAS. The fat is unable to migrate deep to the nasolabial fold inferiorly but is forced superiorly and thus a bulging of fat overlying the sulcus is clinically visible resulting in a

deep and sagged nasolabial fold. The tear trough deformity and the malar mounds are aggravated by the loss of projection of the maxilla with aging. Mandibular volume loss contributes to the laxity of the platysma and soft tissues of the neck, the loss of jawline definition, and the appearance of jowls (Fig. 9). People with poor facial skeletal support (midface hypoplasia, microgenia, and retrusive supraorbital rim) are predisposed to manifest aging changes prematurely.

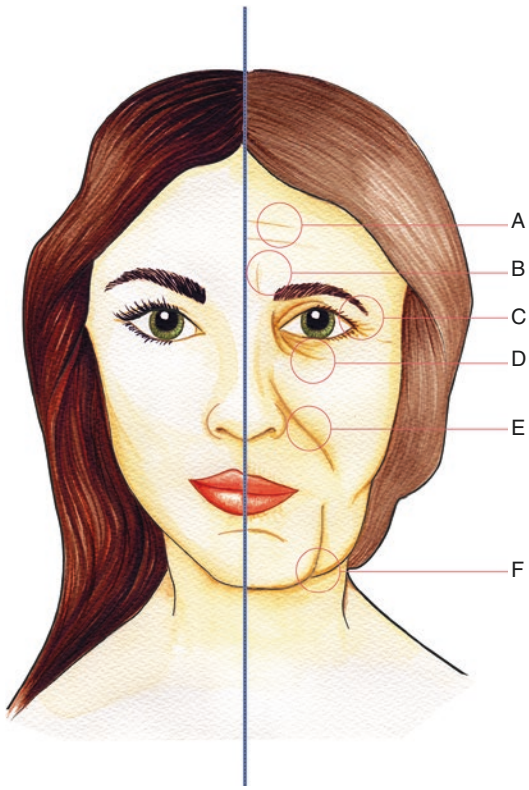

Fig. 9 The youthful (*left*) and the aged face (*right*). (**a**) Horizontal frontal wrinkles due to effect of frontalis muscle contraction. (**b**) Glabellar wrinkles due to the procerus and corrugator supercilii muscles contraction. (**c**) Periocular wrinkles due to orbicularis oculi muscle contraction. Eyebrow ptosis and sagging of the ROOF compartment due to laxity of the orbicularis oculi muscle and orbicularis retaining ligament, and bone resorption. (**d**) Tear trough deformity: aggravated by the orbital and maxillary bone resorption, the laxity of the orbital retaining and zygomatic ligaments, and changes in the SOOF compartment. (**e**) Nasolabial sulcus is formed by the overlying superficial nasolabial fat compartment and the traction of the underlying muscles of facial expression. Maxillary and pyriform bone resorption increases the sagging appearance of the nasolabial fold. (**f**) Jowl deformity. The mandibular ligament attaches the skin to the bone, and the superficial and deep fat compartments posterior to it are more loosely attached and can migrate inferiorly forming the jowls. The mandibular bone resorption contributes to the jowl deformity

Conclusions

To properly restore the youthful facial appearance, it is essential to understand the facial morphological changes over time. These changes affect facial skeleton, fat compartments, soft tissue, retaining ligaments, and skin in variable degrees depending on intrinsic and extrinsic factors. A balanced approach to facial rejuvenation between bone and fat volume augmentation and soft tissue envelope repositioning will avoid the distortions of either approach in isolation. Skeletal resorption can be improved with calcium hydroxyapatite injections or implants. The loss of volume in fat compartments can be treated with fillers or fat grafting in specific deflated soft tissue compartments. The SMAS, the retaining ligaments, and the lid structures can be modified with surgery. Botulinum toxin is useful to reduce the increased muscular resting tone present in aging. Skin rejuvenation can be performed with tretinoin, laser resurfacing, and peels. To return the characteristics of youth to the face, it is necessary to carry out an individualized and step-by-step approach.

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