The Anatomic Basis of Midfacial Aging

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Aging theories • Midfacial aging • Morphologic changes • Pathogenesis

> "Nature gives you your face at twenty. Life shapes your face at thirty. But the face you have at fifty is the face you have earned."
>
> Coco Chanel

Facial aging is a multifactorial, three-dimensional (3D) process with anatomic, biochemical, and genetic correlates. Many exogenous and endogenous factors can significantly impact the perceived age of an individual. Solar exposure [1–3], cigarette smoking [1, 2, 4, 5], medications [1], alcohol use [1], body mass index [2], and endocrinologic status [1, 6, 7] have all been implicated as factors that accelerate cutaneous and subcutaneous aging. These factors act in concert to create a variegated spectrum of facial morphologic aging changes, and thus, Mme. Chanel was partially correct in her statement from the last century.

Most of the aging changes that occur in the midface, however, occur predictably in the majority of individuals. Stigmata of midfacial aging typically appear by the middle of the fourth decade. Degenerative changes occur in nearly every anatomic component of the midface and include cranial bone remodeling, tissue descent secondary to gravity, fat atrophy, and deterioration in the condition and appearance of the skin. The lower eyelids and adjacent tissues are often the initial areas of patient concern.

This chapter reviews the morphologic changes that occur in the aging midface and discusses the pathogenesis of midfacial aging based upon its anatomic components. An integrated theory of facial aging will be presented.

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Morphology

Midfacial aging is directly related to changes that occur in the periorbital region and the cheek. The "youthful eye" is characterized by an upward slant from the medial to lateral canthus, a lower lid position 1–2 mm above the inferior edge of the limbus, and a smooth curve at the lid/cheek junction. The cheeks are volumized, appearing full and round, and the malar prominence is covered by malar fat (Fig. 2.1a). The midface conveys a heart-shaped appearance with the point of the "heart" at the chin, and the greatest prominence over the zygomatic arch extending to the inferolateral orbit. Overhead lighting accentuates this prominence, giving luster to the cheeks. The transition zones from the lower eyelids to the cheeks and lateral nasal wall are smoothly blended without obvious shadows or hollows in the youthful state.

Aesthetically undesirable aging changes begin to appear in the midface during the fourth decade, as the aesthetic subunits start to lose their homogeneity (Fig. 2.1b). The lateral nasal wall loses its smooth transition over the anterior maxilla and becomes convex. Orbicularis rolls appear in the pretarsal eyelid, along with fine rhytids in the lateral canthal area. A nasojugal groove, termed the "tear trough," forms below the lower eyelid, and the nasolabial fold grows in length and depth. The skin loses its sheen and becomes more sallow, pigmented, coarse, and wrinkled.

As aging continues into the fifth decade and beyond, bulges appear below the lower eyelid, consistent with protrusion of the temporal, nasal, and central lower eyelid fat pads. The tear trough elongates and the inferior orbital rim becomes visible. A hollow develops in the centromedial cheek below the tear trough, termed the "V" deformity. The cheeks lose their projection and hollow. Festoons, redundancy at the inferior margin of the orbicularis, may develop in the midcheek. The nasolabial fold becomes increasingly prominent and the midface appears to descend inferiorly and nasally. The corners of the lip become down-turned, as though weighed down by the ptotic midfacial fat, and melomental folds (marionette lines) develop. The midface, heart-shaped in youth, develops a pear shape (Fig. 2.2).

Periorbital Tissues

Visual tracking studies suggest that observers infer subjects' ages by visual cues around the eyes [8, 9]. Age-related changes in the periorbital tissues, such as under-eye bags and wrinkles, significantly impact the perceived age of an individual [8]. These visual morphologic cues, discussed above, develop insidiously.

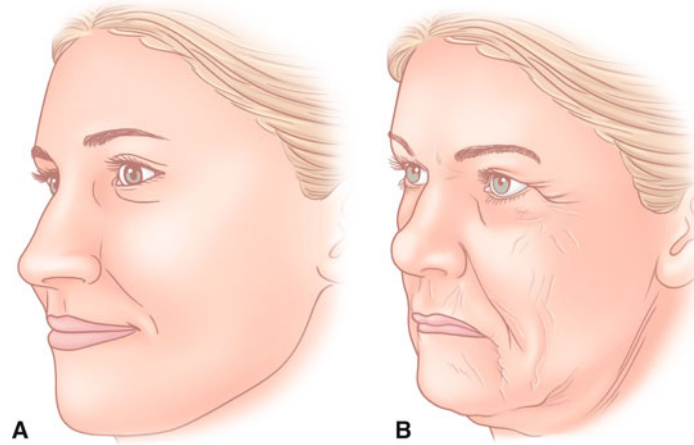

Fig. 2.1 (**a**) Youthful midface. Note the *round*, volumized cheek and smooth contours. (**b**) Aging midface. There is loss of homogeneity, as the tear trough and nasolabial folds deepen

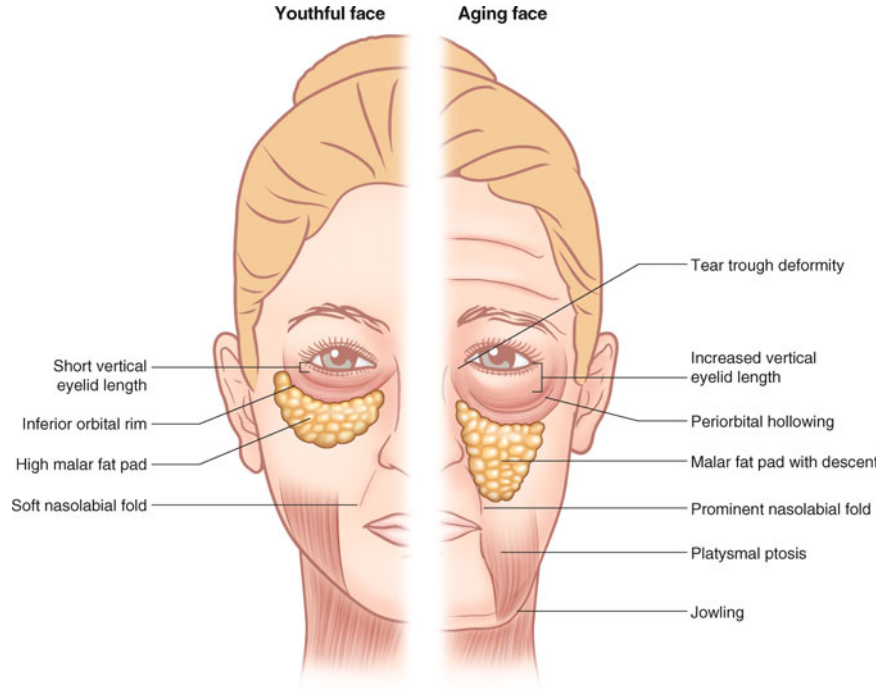

Fig. 2.2 Attributes of the youthful versus aged face. In the youthful face, malar fat overlies the malar prominence, conveying a heart-shaped appearance to the midface with the point of the "heart" at the chin. With age, the malar fat pad descends, baring the inferior orbital rim and contributing to the formation of the nasolabial fold. The vertical length of the lower eyelid appears to increase. Ptosis is also evident in the brow and the lower face

The tear trough, often the initial sign of midfacial aging, occurs in the lower eyelids as a hollow immediately below the lower eyelid fat prominences. Initially described by Loeb, the pathogenesis of the tear trough is ascribed to three coexisting anatomic factors (1) the solid fixation of the orbital septum to the inferomedial arcus marginalis, (2) a "triangular gap" formed by the junction of the orbicularis oculi, medial lip elevators, and levator alaeque nasi and, (3) the absence of fat and soft tissue from the central and the medial fat pads subjacent to the inferior orbicularis oculi [10]. Freeman showed that lack of fat at the level of the inferior arcus marginalis is the major contributing factor in the tear trough deformity, as demonstrated by intraoperative and cadaveric dissections of individuals with and without a tear-trough convexity [11].

In many individuals protrusion of fat becomes more apparent in the lower eyelids with age. Hamra referred to the alterations in the contour of the lower eyelid at this point of chronologic aging as the "double convexity deformity," which occurs as bulging lower eyelid fat is juxtaposed to the hollow of the tear trough [12]. The etiology of this increased fat prominence is controversial. Potential causes include orbital fat pseudoherniation or an increase in the amount of lower eyelid adipose tissue. In a study of orbital and facial computed tomography (CT) images from 167 patients, Chen et al. demonstrated that lower eyelid orbital fat herniation occurred with chronologic aging [13]. Attenuation of the orbital septum may contribute to the appearance of pseudoherniation [14–16]. De la Plaza suggested that distension of orbital supporting structures, such as the orbital septum, capsulopalpebral fascia, and lateral canthal tendon, leads to descent of the globe with resultant compression and anterior displacement of inferior orbital fat [14]. Alternatively, downward stress on

the septum by "sagging" volumes in the midface may traction forward the septum and the fat immediately posterior to it, thus increasing the visibility of the lower eyelid fat [17].

Some authors have proposed that the volume of orbital fat increases in the aging orbit. Darcy et al. analyzed high-resolution orbital magnetic resolution images (MRI) in 40 patients and demonstrated that orbital fat expansion occurs with age, displacing the soft tissues of the lower eyelid anteriorly [18]. However, the findings were limited, as single MRI cuts were used to extrapolate orbital volume. The authors postulated that adipocyte hyperplasia/hypertrophy or chronic fluid accumulation with age caused the orbital fat volume to increase.

In all likelihood, the most important change that occurs in the lower eyelids is the unveiling of deep eyelid contour that results from descent of midface fat and focal volume loss at the inferior orbital rim [19]. The orbital aperture appears to enlarge vertically as the midface volumes descend and exert downward traction on the septum and arcus marginalis [17], exposing the tear trough and eventually the inferior orbital rim. Lower eyelid fat becomes prominent due to the absence of the malar fat that covers it in youth. This is consistent with Lambros' theory of volume deflation [20], discussed later in this chapter.

Midface Skeleton

As aging progresses, the bony midface "collapses": the inferior orbital rim remodels and loses anterior projection, the midface loses vertical height, and the pyriform aperture recesses posteriorly [17, 21–25] (Fig. 2.3). Studies of normal CT scans, grouped by patient age, show that skeletal changes predictably occur at the orbital rim and the maxilla. Involution of the midface skeleton begins in the sixth decade and is observed more commonly in women than in men [27]. Increased bone resorption during perimenopause may contribute to the greater degree of bony involution observed in women [28].

Kahn and Shaw, using 3D CT reconstruction with volume rendering, found a significant increase in the orbital aperture and width correlated to advancing age in both male and female patients [25]. Woodward et al. corroborated these results in a review of consecutive facial CT scans in 50 females and 50 males [17]. Their analyses demonstrated that the angles of the pyriform process and the inferior orbital rim retruded with age, while the anterior lower eyelid fat pads appeared to become more prominent. The authors hypothesized that inferior and downward displacement of the orbital rim may pull the orbital

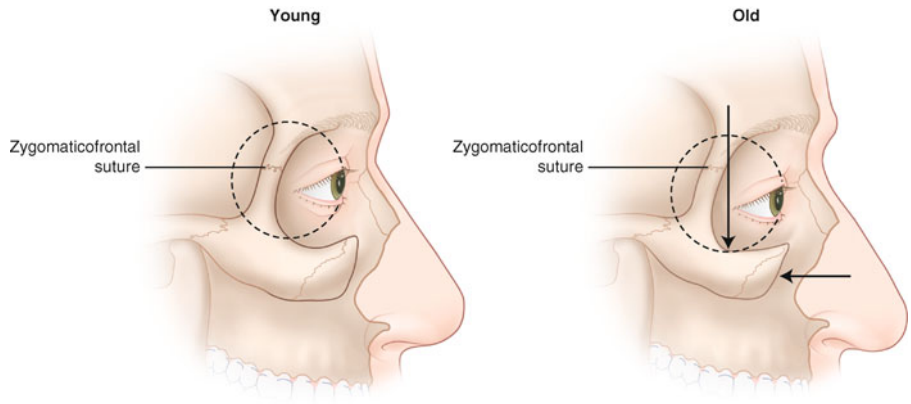

Fig. 2.3 With aging, the inferior orbital rim remodels and loses anterior projection. Adapted from Pessa [26]

fat anteriorly due to "shared attachments of the orbital septum and lower eyelid retractors." They also speculated that this inferior displacement exerted downward traction on the lower eyelid, resulting in the relative lower eyelid retraction seen with aging [17].

In a study of facial CT images from 30 male patients, Pessa et al. showed that the maxilla vertically shortened in proportion to the orbit with aging and attributed these changes to skeletal remodeling [23]. The authors proposed that the enlarged orbital aperture in combination with the shortened vertical maxilla results in a "collapse" of the surface area available in the midface to support the overlying soft tissues. This phenomenon was termed the "concertina effect" due to the decreased ability of the bones to support and volumize the overlying soft tissues [23]. In another study, Pessa et al. demonstrated that the globe becomes relatively proptotic with respect to the aging alterations of the inferior orbital rim and the cheek mass [22]. The authors have speculated that this relative maxillary retrusion might contribute to the prominence of the nasolabial fold in aging [29].

Dentition

Bony involution of the upper jaw, as described above, may be related to the loss of alveolar bone engendered by loss of dentition [30]. Bartlett et al. studied 160 human skulls and found that a reduction in facial height, especially in the maxilla and mandible, strongly correlated with tooth loss [31]. The loss of teeth alone affects the thickness of cortical bone throughout the facial skeleton; significant cortical bone loss and alveolar ridge absorption are observed in the edentulous [32]. The loss of load-bearing stresses in the edentulous state may result in maxillary ridge resorption, which occurs in the craniofacial skeleton throughout life [33]. Alternatively, the absence of trophic factors related to a reduction in tooth vascularization may result in a decrease in metabolic demand and decreased osteoblastic activity. This process is analogous to that of the anophthalmic orbit [34]. In general, loss of teeth affects the mandible more than the maxilla, and more bone loss is observed in women than in men [35].

Musculature

Aging causes a decline in muscle mass and muscle strength throughout the body [36–39], and these muscular changes have been studied in the midface [40, 41]. The orbicularis oris thins with age [42], whereas the orbicularis oculi does not [43].

Analyses of MRI images from patients of varying ages suggest that the aging muscles of the midface shorten and straighten, as though they are in spasm. Le Louarn has hypothesized that this spasm may prolapse the deep midfacial fat superficially with the continuous repetitive facial contractions that occur over a lifetime [44, 45]. Owsley and Roberts' analyses of histologic specimens and MRI images of the midface suggest that repeated contractions of the levator labii during animation may produce increased tissue expansion forces in the overlying cheek fat pad, contributing to its downward migration [46]. These findings support Le Louarn's hypothesis.

Adipose Tissue

Recent studies authored by Rohrich and Pessa have sought to define the fat compartments of the face and describe their clinical importance [47–52]. Superficial midfacial fat pads include the medial, middle, and lateral cheek fat compartments [51]. The deep medial fat pad underlies the superficial middle fat pad [52]. Inflation of the deep medial fat pad with saline in cadavers has been shown to eliminate the V deformity, reduce the size of the nasolabial fold, and diminish the appearance of the tear trough [52]. Based upon this finding and other anatomic observations, the authors have proposed that volume loss in the deep midfacial fat compartment may be one of the primary determinants of the morphologic appearance of the aging midface. The loss of midfacial projection caused by volume atrophy

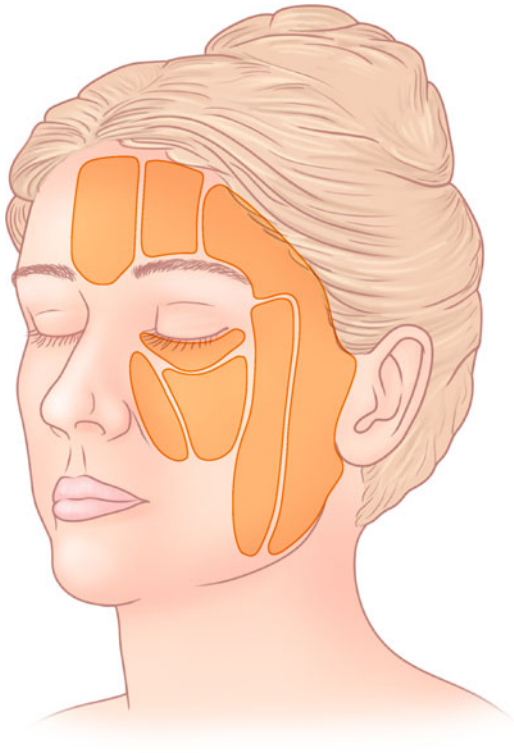

Fig. 2.4 Subcutaneous fat compartments of the face, as described by Rohrich et al. The subcutaneous fat in the face lies within relatively consistent anatomic compartments separated by connective tissue laminae. Adapted from Rohrich and Pessa [51]

unmasks the nasolabial fold and malar mounds, and the observed ptosis of soft tissues is actually "pseudo-ptosis" created by deflationary changes.

These studies on the organization of midfacial fat suggest that facial aging is compartment dependent (Fig. 2.4). That is, the face does not age as a confluent mass; rather, each compartment changes relatively independently over time [51]. Shearing forces between adjacent compartments may also contribute to the soft tissue malposition seen in aging [51]. Shifting of the deep fat compartments, along with volume loss within them, may lead to the pseudo-ptosis associated with aging. This leads to the appearance of "folds" (such as the nasolabial fold) at the junction of the superficial and deep fat compartments [48].

Gosain et al. performed high-resolution MRI in 20 "old" and "young" female subjects in a study evaluating volumetric changes in the aging midface [53]. Based upon their findings, the authors postulated that there is selective hypertrophy of the superior portion of the superficial fat pad in aging patients, and that a generalized redistribution of fat occurs with aging, perhaps related to actions associated with animation, that makes the nasolabial fold more prominent [53]. The study was performed using MRI in a supine position, which potentially mars the accuracy of their observations because it negates the effect of gravity as encountered in the upright position.

Owsley and Roberts discussed their extensive experience with midface lifting and used Gosain's observations to posit a theory as to the etiology of the nasolabial folds [46]. They proposed that the dynamic nature of facial animation leads to changes in fat position, resulting in downward migration of the fat pad. Prolonged muscle shortening over a lifetime causes radial expansion and elongation of the septations within the inferior malar fat pads. The lengthening of septations within the fat results in downward migration of the cheek and formation of the nasolabial fold [46].

An alternative theory proposed by Lambros suggests that the aging changes seen in the face, and in particular the midface, are due to volume loss [20]. According to Lambros' theory, the nasolabial fold deepens with age due to volume loss and inferior displacement of the deflated tissue envelope, not as a result of gravitationally induced soft-tissue descent [20]. This contradicts the theory of Owsley and Roberts, who suggest that fat atrophy in the midface plays a secondary role to gravitational descent of the cheek fat [46].

It is clear that changes in the subcutaneous and deep cheek fat over time contribute to the pathogenesis of midfacial aging, though the underlying mechanism of these changes (fat displacement versus atrophy) remains open to debate. In all likelihood, both mechanisms in yet-to-be-defined combination give rise to the aesthetic alterations seen in midfacial aging.

Superficial Muscular Aponeurotic System

Lockwood et al. have described a subcutaneous fasciomuscular system throughout the body [54]. This system, as it pertains to the face, was initially described by Mitz and Peyronie [55] and has been further detailed by Rohrich et al. [47–52, 56]. The superficial muscular aponeurotic system (SMAS) is described as a mid-level fibromuscular layer separating the deep facial fat from the superficial facial fat. Essentially, the subcutaneous fat that exists in the face lies within relatively consistent anatomic compartments separated by connective tissue laminae (Fig. 2.4). The blood supply to the fat and dermis runs within this fibrous connective tissue in a vertically oriented network, and the connective tissue is thought to lend support to the vessels [56].

Numerous studies have emerged that are insightful in understanding the anatomy of the SMAS and its role in facial aging [57–62]. Dissections have shown that considerable variability exists in the SMAS at different facial regions within a single individual, as well as at given anatomic locations among individuals [57, 60]. Macchi et al. postulated that the SMAS forms a 3D network with subcutaneous connective tissue fibers that ultimately have a connection to the dermis. In their estimation, changes in the viscoelastic properties of the SMAS and its 3D reticular network ultimately resulted in ptosis of facial volumes [58]. Histologic analyses by Owsley and Roberts have also shown that degenerative loss of elastin in both the superficial fascia and the skin contributes to the aging changes in the midface and the nasolabial folds [46].

In the midface, the SMAS is less distinct as compared to other facial regions, and its dermal attachments are not as easily demonstrated at surgery or in cadaveric dissections. Studies have shown that the SMAS attenuates from thickest in the parotid region to thinnest over the nasolabial fold, where it is virtually undetectable. While the literature is replete with articles pertaining to the SMAS, not all are in agreement regarding its existence, particularly in the midface. A study using tissue plastination techniques did not demonstrate a clear SMAS layer in the face, except adjacent to the parotid; in particular, SMAS was not demonstrated in the midface or the neck [62]. It is plausible that either lack of a SMAS layer in the midface, or the attenuation of the SMAS with aging, may contribute to the early and progressive aging process observed in the midface. Further studies are needed to elucidate the extent and the role of the SMAS in midfacial aging.

Ligaments

The ligaments of the face were first characterized by Furnas, who noted that fibrous condensations encountered in facelift dissections were in fact osseocutaneous and musculocutaneous supporting structures [63, 64]. The relationships of these ligaments to the SMAS, lower eyelid and lateral canthus have been extensively described, though the nomenclature describing them is inconsistent. Fundamentally, the orbicularis is supported by adhesions to the temporalis, and the cheek is suspended by the zygomaticomalar ligaments. The superficial and the deep fat pads in the infraorbital area are suspended by a ligament originating at the arcus marginalis, termed the "orbicularis retaining ligament" [65, 66], or in ophthalmic parlance, the "orbitomalar ligament" [67] (Fig. 2.5). Pessa described an inferior orbicularis ligament, which has also been termed the "orbitomalar ligament" [68]. It is thought to account for the delimitation of cellulitis and cheek hematomas.

Mendelson has extensively detailed the ligaments, septae, and adhesion zones of the brow, periocular region, and midface [65, 69–71]. He postulated that ligamentous laxity may be primarily responsible for facial aging. Mendelson proposed that the ligaments serve to stabilize the face in youth, but continuous muscular activity in combination with intrinsic aging changes lead to weakness of ligamentous support [71]. Thus, the transient soft tissue displacement occurring over the course of a lifetime with facial animation produces stretching of facial ligaments and causes subsequent ptosis of the soft tissues of the face.

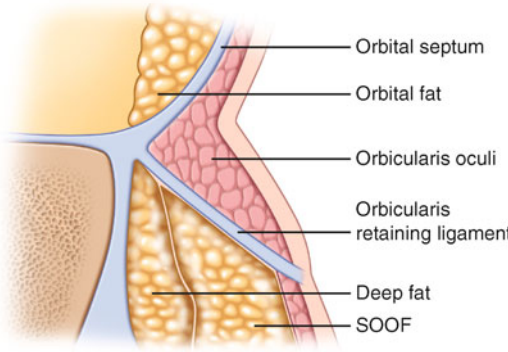

Fig. 2.5 The orbicularis retaining ligament, as seen in cross-section, is a bilaminar membrane separating the preseptal and prezygomatic spaces. It suspends the superficial and deep fat pads in the infraorbital area. Central laxity of the orbicularis retaining ligament allows the lower eyelid fat to descend into the upper cheek, contributing to the "V" deformity at the lid–cheek junction. Adapted from Owsley and Roberts [46]

Skin

Aging changes in facial skin begin in the fourth decade and include fine wrinkles in the lower eyelids and lateral orbital area, dyschromias, and alterations in texture and pigmentation. Genetics, hormonal influences, and environmental factors all contribute to the aging process in facial skin. In intrinsic skin aging, the subcutaneous vasculature loses its papillary organization, the dermal/epidermal interface flattens, and the dermis and subcutaneous tissues atrophy. Microscopically, elastin and collagen lose their organization, decrease in relative amount, and show histologic signs of degeneration [72]. The production of types I and III collagen by fibroblasts is reduced [73]. Fibroblasts, particularly within the papillary dermis, show selective diminution in function and number as compared to fibroblasts in reticular dermis [74].

Exposure to ultraviolet (UV) light is the principal cause of extrinsic aging. The midface is inevitably exposed to the sun and consequently subject to "photoaging," as termed by Kligman and Kligman [75]. Photoaging produces coarse, deeply wrinkled, leathery skin with multiple cutaneous premalignancies and telangiectasias. This is independent of and in addition to intrinsic aging, where the skin becomes dry and atrophic, with fine wrinkles and increased laxity. Both UVA and UVB light cause radiation changes at the basal epithelial level, dermal epidermal junction, and in the subcutaneous tissues. UVA induces solar elastosis [76], whereas UVB causes dysplasia and neoplasia by producing dermal and epidermal injury [77]. Skin that is constantly exposed to UV light is metabolically overactive, with an increased production of abnormal elastin and glycosaminoglycans [75]. Inflammation, angiogenesis, and abnormal collagen production and degradation are noted to a greater extent in sun exposed skin as compared to skin protected from solar exposure [75]. Other solar induced changes include a decrease in hormonal levels, which may contribute to selective fat loss and fat deposition [3]. Elevated blood glucose levels cause glycosylation of skin proteins, intensified by the presence of ultraviolet light [6].

Epidemiologic studies have shown tobacco to have a deleterious effect on the skin, causing premature skin wrinkling [4, 5, 78, 79]. Wrinkle development in one study was significantly correlated to the number of pack years, even after controlling for sun exposure, age, and sex [4]. Sun exposure was also a risk factor for developing wrinkles, with a multiplicative effect found when sun exposure coexisted with a long history of smoking [80]. In vitro studies suggest that tobacco smoking contributes to premature skin aging by impairing collagen synthesis, upregulating metalloproteinases, and causing an increase in the production of abnormal elastin and a reduction in proteoglycans [5]. UV light and smoking independently cause wrinkling of the skin by upregulation of fibroblast metalloproteinases and together have a synergistic negative effect [4].

The Volumetric Theory of Facial Aging

Lambros has carefully and convincingly developed a theory of midfacial and periorbital aging that seems to integrate all the anatomic components involved in the midfacial aging process. His seminal paper of 2007 described his practice of

superimposing his patients' youthful photographs on top of a current photograph, and "animating" the images to fade from one to the other, in order to visualize changes in the face over time [20].

Lambros' analyses suggested that the lid–cheek junction remains stable over time. Comparisons of the lid–cheek position were made in 83 patients, and in only 3 did the lid–cheek junction appear to descend over time. Using photographic comparison, Lambros showed that skin landmarks, such as moles and wrinkles, present at specific anatomic locations in the periorbital and upper midface, did not descend over the time course of the photographs. Based upon these findings, Lambros hypothesized that vertical descent of skin and subcutaneous tissue was not a major component of the midfacial aging process. He suggested that, if the face actually sagged, one would expect to see downward migration of skin landmarks.

Lambros attributed the conspicuity of the aging lid–cheek junction to shadowing created by fat protrusion in the lower eyelid, which increases the apparent vertical length of the lid and exaggerates the growing indentation at the tear trough. Additionally, the increased contrast between the lid and cheek was ascribed to accentuation of lower eyelid skin as it becomes thinner and darker with aging. The apparent descent of the lower lid is therefore an illusion perpetrated by variations in skin color, skin contrast, and tissue projection occurring over time. Lambros speculated that relative anteroposterior shifts in volume played a more dominant role in midfacial aging than soft-tissue descent [20]. Lambros' theory is corroborated by the work of Pessa, who suggested that craniofacial skeletal remodeling might primarily explain volume changes in the midface [26].

The Gravitational Theory of Facial Aging

It is possible to accept Lambros' observations and to reconcile them with the observations of numerous authors who feel that gravitational changes can be implicated in the morphogenesis of facial aging.

It is our observation that inversion photographs of aging patients (either in a supine or Trendelenburg position) demonstrate an appearance consistent with that of photographs taken approximately 10–15 years prior (Fig. 2.6). The tear trough diminishes, orbital fat prolapse is no longer apparent, and cheek nevi ascend to their original positions. Although volume loss does become more apparent in the lower eyelids and in the lips, many of the other features that give rise to the aging face are improved with supine positioning. Aging *is* therefore partially gravitational: much of the volume

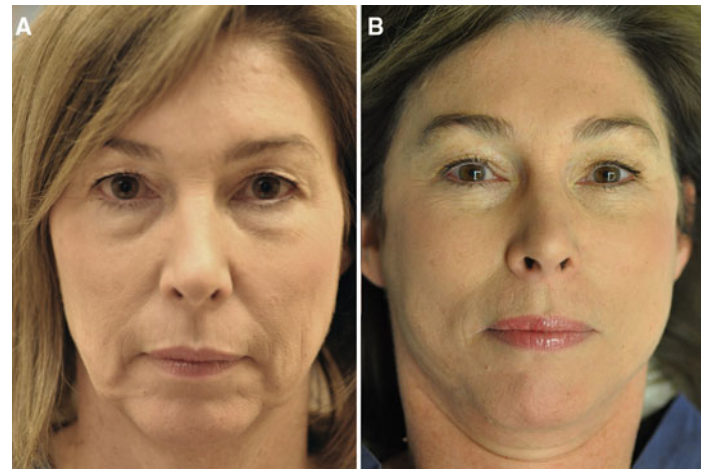

Fig. 2.6 A 51-year-old female in (**a**) prone and (**b**) supine position. With supine positioning, the tear trough is diminished, orbital fat prolapse is reduced, and the nasolabial folds are softened

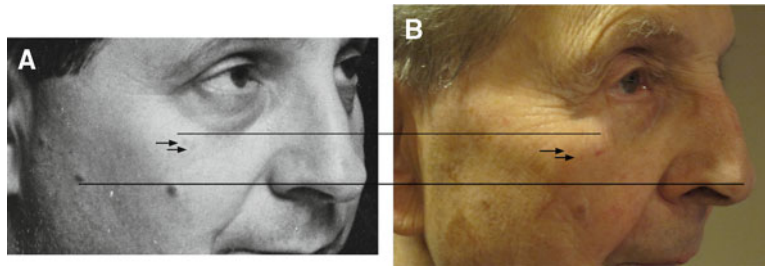

Fig. 2.7 Three-quarters view of a male in his (**a**) fifth decade and (**b**) eighth decade. In each photograph, the *upper line* corresponds to the most anterior projection of the malar prominence, and the *lower line* is placed at the superior border of the nares. Two *black arrows* denote faint nevi located near the lid–cheek junction. These nevi do appear to descend with age

in the face must be retained and not lost, because volumes drift back into position when the supine position is assumed.

We concur with Mendelson in our belief that facial ligaments attenuate with age. These ligaments create a supportive system that suspends the midfacial fat compartments, and attenuation of the ligaments results in gravitational descent of facial fat volumes. Preliminary observations in our patients demonstrate that malar nevi *do* descend over time and that the skin is not immobile in contrast to what has been described by Lambros (Fig. 2.7). Therefore, gravity, and not volume loss alone, may be responsible for many of the changes seen in the aging midface.

Gravity affects the entire face, but it does so non-uniformly. Gravitational changes are not as apparent where the strong anchor points of facial ligaments essentially tether the dermis to the periosteum. Facial volumes can descend without apparent changes at the lid–cheek junction because a dense network of orbital retaining ligaments exists around the periorbit that continue to suspend the skin. The skin at lid–cheek junction is supported medially and centrally by the dense orbicularis retaining ligament, laterally by the lateral canthal thickening, and is suspended between two solid points, the medial and the lateral canthi. These connective tissue attachments act as "hammocks" or "slings" that suspend the skin. As such, this area is more resistant to gravitational displacement than tissues of the mid- and lower cheek. Therefore, the cardinal sign of aging at and above the lid–cheek junction is not volume descent, but rather redundancy of skin and orbicularis suspended in the "hammock" discussed above. Similarly, laxity of the orbital septum allows orbital fat volumes to prolapse down and forward, delimited inferiorly by the orbicularis retaining ligament.

The ligamentous network supporting the mid- and lower cheek, however, is more discrete and compact. As the ligaments stretch over time, facial volumes descend, and the skin descends along with them. Immediately below the lid–cheek junction, and especially laterally, where the orbicularis retaining ligament is less firmly attached, one sees a downward descent of fat volumes. Attenuation of the zygomatic ligaments and muscular or SMAS attachments to skin also contributes to this downward drift.

The visual stigmata of midfacial aging often correspond to the areas of ligamentous attachments: the hollows that develop occur in the areas of the osseous origins of the ligaments (Fig. 2.8a, b), and the volumes and bulges below them represent areas of ligamentous attenuation where the fat compartments of the face have been displaced (Fig. 2.8c). The facial hollows and bulges are most visible in the presence of intense overhead lighting, which creates shadows established by the concavities that disappear with the redistribution of fat volumes when the patient is placed in the supine position (Fig. 2.6).

This theory is supported by the histologic observations of Lucarelli et al. who noted attenuation of

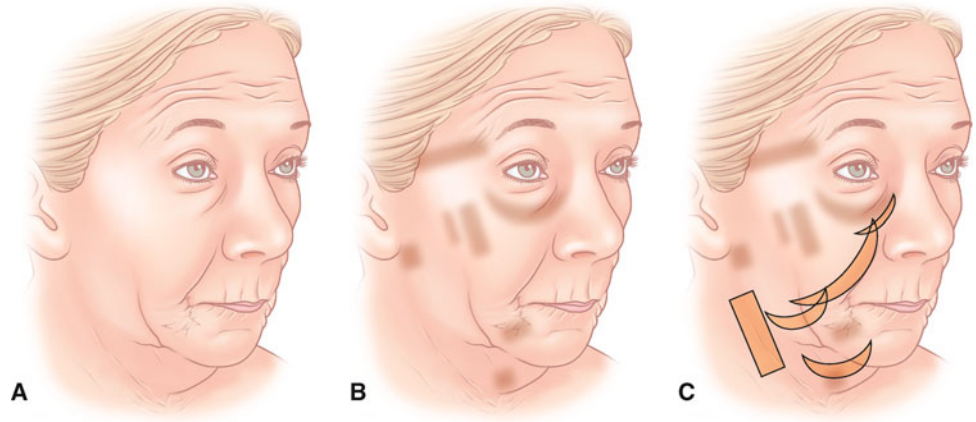

Fig. 2.8 (**a**) This woman demonstrates salient features of aging, including tear trough deformity, hollowing of the cheek, nasolabial folds, melomental folds, and jowling. (**b**) The hollows that develop in the face with age tend to occur in areas of the osseous origins of the ligaments. (**c**) Bulges in the aging face (as represented by *orange crescents* and *rectangles*) form in areas of ligamentous attenuation, where the fat compartments have been displaced due to lack of support

the orbitomalar ligament in eight of ten specimens with midfacial ptosis in their study [81]. Additionally, subcutaneous components of the zygomatic and masseteric cutaneous ligaments were either attenuated or not identifiable in 40% and 30% of cadavers with midfacial ptosis, respectively [81]. A study by Raskin and Latrenta adds further support to our postulate. Attenuation of the secondary supporting ligaments correlated to cadaver age in ten cadaver dissections. They additionally reported histologic evidence of distal attenuation of the ligaments within the malar fat [82].

Stuzin, Baker, and Gordon have described the anatomic relationships between facial fascias, muscles, fat pads, and skin in their precis based upon seven cadaver dissections and experience with hundreds of facelifts [57]. The authors concluded:

> The importance of the zygomatic ligaments lies in their ability to suspend malar soft tissue over the zygomatic eminence. In aging, an attenuation of malar support is commonly seen, leading to an inferior migration of malar soft tissue. This soft tissue ptosis occurs adjacent to the line of muscular fixation along the nasolabial fold. It is not that the fold deepens with aging, but rather malar soft tissue lateral to the nasolabial line accumulates, accounting for fold prominence in the aging face. (p 447) [57]

Owsley and Roberts have postulated that the descent of the malar fat pad, caused by attenuation in the dermal septal attachments, creates the permanent nasolabial fold associated with facial aging [46]. This corroborates Stuzin, Baker, and Gordon's explanation of the midface ptosis seen with aging. Similarly, Mendelson and Jacobson offered an explanation for why the lid–cheek contour changes over time with a relatively constant position of the lid–cheek junction [69]. They proposed that the orbicularis retaining ligament becomes attenuated over time, but still retains its attachments to the anterior lamella and the inferior orbital rim, and as a consequence, orbital fat appears to bulge even as the lid–cheek junction remains stable [69]. In conjunction with bony volume loss of the inferior rim, volume descent and traction on facial volumes may be responsible for the morphologic changes seen in the lower eyelids and the tear trough [17].

The concept of ligamentous dehiscence reconciles Lambros' findings with our observations that the fat volumes and the soft tissues of the midface appear to descend with age. As noted by Lambros, the skin tends to remains in place, particularly at the lid–cheek junction, because it is a passive envelope held in place by a nexus of

orbital supporting structures. However, the facial volumes beneath the dermis do sag with age. Facial volumes descend because of the attenuation of their surrounding connective tissue compartments and the ligaments that support them. Bony volume loss and selective atrophy and/or displacement of the midfacial deep and superficial fat compartments act in conjunction with gravity to cause the facial changes associated with midfacial aging.

Conclusions

Midfacial aging is a multifactorial, three-dimensional process that involves volume loss, volume descent, and skin alterations. Every anatomic component of the midface is affected. This chapter discussed the morphologic consequences of midfacial aging and reviewed the salient literature relating to the numerous factors that contribute to an aged appearance.

References

1. Guyuron B, Rowe DJ, Weinfeld AB, et al. Factors contributing to the facial aging of identical twins. Plast Reconstr Surg. 2009;123(4):1321–31.
2. Rexbye H, Petersen I, Johansens M, et al. Influence of environmental factors on facial ageing. Age Ageing. 2006;35(2):110–5.
3. Makrantonaki E, Zouboulis CC. Molecular mechanisms of skin aging: state of the art. Ann NY Acad Sci. 2007;1119:40–50.
4. Yin L, Morita A, Tsuji T. Skin aging induced by ultraviolet exposure and tobacco smoking: evidence from epidemiological and molecular studies. Photodermatol Photoimmunol Photomed. 2001;17(4):178–83.
5. Morita A. Tobacco smoke causes premature skin aging. J Dermatol Sci. 2007;48(3):169–75.
6. Danby FW. Nutrition and aging skin: sugar and glycation. Clin Dermatol. 2010;28(4):409–11.
7. Makrantonaki E, Zouboulis CC. Androgens and ageing of the skin. Curr Opin Endocrinol Diabetes Obes. 2009;16(3):240–5.
8. Nkengne A, Bertin C, Stamatas GN, et al. Influence of facial skin attributes on the perceived age of Caucasian women. J Eur Acad Dermatol Venereol. 2008;22(8):982–91.
9. Rexbye H, Povlsen J. Visual signs of ageing: what are we looking at? Int J Ageing Later Life. 2007;2(1):61–83.
10. Loeb R. Naso-jugal groove leveling with fat tissue. Clin Plast Surg. 1993;20(2):393–400.
11. Freeman MS. Transconjunctival sub-orbicularis oculi fat (SOOF) pad lift blepharoplasty: a new technique for the effacement of nasojugal deformity. Arch Facial Plast Surg. 2000;2(1):16–21.
12. Hamra ST. Repositioning the orbicularis oculi muscle in the composite rhytidectomy. Plast Reconstr Surg. 1992;90(1):14–22.
13. Chen YS, Tsai TH, Wu ML, Chang KC, Lin TW. Evaluation of age-related intraorbital fat herniation through computed tomography. Plast Reconstr Surg. 2008;122(4):1191–8.
14. de la Plaza R, Arroyo JM. A new technique for the treatment of palpebral bags. Plast Reconstr Surg. 1988;81(5):677–87.
15. Huang T. Reduction of lower palpebral bulge by plicating attenuated orbital septa: a technical modification in cosmetic blepharoplasty. Plast Reconstr Surg. 2000;105(7):2552–8.
16. Mühlbauer W, Holm C. Orbital septorhaphy for the correction of baggy upper and lower eyelids. Aesthetic Plast Surg. 2000;24(6):418–23.
17. Richard MJ, Morris C, Deen BF, Gray L, Woodward JA. Analysis of the anatomic changes of the aging facial skeleton using computer-assisted tomography. Ophthal Plast Reconstr Surg. 2009;25(5):382–6.
18. Darcy SJ, Miller TA, Goldberg RA, et al. Magnetic resonance imaging characterization of orbital changes with age and associated contributions to lower eyelid prominence. Plast Reconstr Surg. 2008;122(3):921–9.
19. Goldberg RA. The three periorbital hollows: a paradigm for periorbital rejuvenation. Plast Reconstr Surg. 2005;116(6):1796–804.
20. Lambros V. Observations on periorbital and midface aging. Plast Reconstr Surg. 2007;120(5):1367–76.
21. Zadoo VP, Pessa JE. Biological arches and changes to the curvilinear form of the aging maxilla. Plast Reconstr Surg. 2000;106(2):460–6.
22. Pessa JE, Desvigne LD, Lambros VS, Nimerick J, Sugunan B, Zadoo VP. Changes in ocular globe-to-orbital rim position with age: implications for aesthetic blepharoplasty of the lower eyelids. Aesthetic Plast Surg. 1999;23(5):337–42.
23. Pessa JE, Zadoo VP, Yuan C, et al. Concertina effect and facial aging: nonlinear aspects of youthfulness and skeletal remodeling, and why, perhaps, infants have jowls. Plast Reconstr Surg. 1999;103(2):635–44.
24. Levine RA, Garza JR, Wang PT, Hurst CL, Dev VR. Adult facial growth: applications to aesthetic surgery. Aesthetic Plast Surg. 2003;27(4):265–8.
25. Kahn DM, Shaw Jr RB. Aging of the bony orbit: a three-dimensional computed tomographic study. Aesthet Surg J. 2008;28(3):258–64.
26. Pessa JE. An algorithm of facial aging: verification of Lambros's 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(2):479–88.

27. Doual JM, Ferri J, Laude M. The influence of senescence on craniofacial and cervical morphology in humans. Surg Radiol Anat. 1997;19(3):175–83.
28. Glowacki J. Impact of postmenopausal osteoporosis on the oral and maxillofacial surgery patient. Oral Maxillofac Surg Clin North Am. 2007;19(2):187–98.
29. Pessa JE, Zadoo VP, Mutimer KL, et al. Relative maxillary retrusion as a natural consequence of aging: combining skeletal and soft-tissue changes into an integrated model of midfacial aging. Plast Reconstr Surg. 1998;102(1):205–12.
30. Xie Q, Wolf J, Ainamo A. Quantitative assessment of vertical heights of maxillary and mandibular bones in panoramic radiographs of elderly dentate and edentulous subjects. Acta Odontol Scand. 1997;55(3):155–61.
31. Bartlett SP, Grossman R, Whitaker LA. Age-related changes of the craniofacial skeleton: an anthropometric and histologic analysis. Plast Reconstr Surg. 1992;90(4):592–600.
32. Schwartz-Dabney CL, Dechow PC. Edentulation alters material properties of cortical bone in the human mandible. J Dent Res. 2002;81(9):613–7.
33. Dechow PC, Wang Q, Peterson J. Edentulation alters material properties of cortical bone in the human craniofacial skeleton: functional implications for craniofacial structure in primate evolution. Anat Rec. 2007;293(4):618–29.
34. Kennedy RE. The effect of early enucleation on the orbit in animals and humans. Adv Ophthalmic Plast Reconstr Surg. 1992;9:1–39.
35. Xie Q, Ainamo A, Tilvis R. Association of residual ridge resorption with systemic factors in home-living elderly subjects. Acta Odontol Scand. 1997;55(5):299–305.
36. Grimby G, Saltin B. The ageing muscle. Clin Physiol. 1983;3(3):209–18.
37. Stalberg E, Borges O, Ericsson M, et al. The quadriceps femoris muscle in 20-70-year-old subjects: relationship between knee extension torque, electrophysiological parameters, and muscle fiber characteristics. Muscle Nerve. 1989;12(5):382–9.
38. Brooks SV, Faulkner JA. Skeletal muscle weakness in old age: underlying mechanisms. Med Sci Sports Exerc. 1994;26(4):432–9.
39. Thomason DB, Booth FW. Atrophy of the soleus muscle by hindlimb unweighting. J Appl Physiol. 1990;68(1):1–12.
40. Abe Y, Inamura H, Koshu H, et al. Effects of aging on facial nerve and facial mimetic muscles-investigation of facial wrinkles and CMAPS. Facial Nerve Res. 2005;25:29–41.
41. Wohlert AB, Smith A. Spatiotemporal stability of lip movements in older adult speakers. J Speech Lang Hear Res. 1998;41(1):41–50.
42. Penna V, Stark GB, Eisenhardt SU, Bannasch H, Iblher N. The aging lip: a comparative histological analysis of age-related changes in the upper lip complex. Plast Reconstr Surg. 2009;124(2):624–8.
43. Pottier F, El-Shazly NZ, El-Shazly AE. Aging of orbicularis oculi: anatomophysiologic consideration in upper blepharoplasty. Arch Facial Plast Surg. 2008;10(5):346–9.
44. Le Louarn C, Buthiau D, Buis J. Structural aging: the facial recurve concept. Aesthetic Plast Surg. 2007;31(3):213–8.
45. Le Louarn C. Muscular aging and its involvement in facial aging: the Face Recurve concept. Ann Dermatol Venereol. 2009;136 Suppl 4:S67–72.
46. Owsley JQ, Roberts CL. Some anatomical observations on midface aging and long-term results of surgical treatment. Plast Reconstr Surg. 2008;121(1):258–68.
47. Rohrich RJ, Ahmad J, Hamawy AH, Pessa JE. Is intraorbital fat extraorbital? Results of cross-sectional anatomy of the lower eyelid fat pads. Aesthet Surg J. 2009;29(3):189–93.
48. Rohrich RJ, Arbique GM, Wong C, Brown S, Pessa JE. The anatomy of suborbicularis fat: implications for periorbital rejuvenation. Plast Reconstr Surg. 2009;124(3):946–51.
49. Rohrich RJ, Pessa JE. The anatomy and clinical implications of perioral submuscular fat. Plast Reconstr Surg. 2009;124(1):266–71.
50. Rohrich RJ, Pessa JE. The retaining system of the face: histologic evaluation of the septal boundaries of the subcutaneous fat compartments. Plast Reconstr Surg. 2008;121(5):1804–9.
51. Rohrich RJ, Pessa JE. The fat compartments of the face: anatomy and clinical implications for cosmetic surgery. Plast Reconstr Surg. 2007;119(7):2219–27.
52. Rohrich RJ, Pessa JE, Ristow B. The youthful cheek and the deep medial fat compartment. Plast Reconstr Surg. 2008;121(6):2107–12.
53. Gosain AK, Klein MH, Sudhakar PV, Prost RW. A volumetric analysis of soft-tissue changes in the aging midface using high-resolution MRI: implications for facial rejuvenation. Plast Reconstr Surg. 2005;115(4):1143–52.
54. Lockwood TE. Superficial fascial system (SFS) of the trunk and extremities: a new concept. Plast Reconstr Surg. 1991;87(6):1009–18.
55. Mitz V, Peyronie M. The superficial musculo-aponeurotic system (SMAS) in the parotid and cheek area. Plast Reconstr Surg. 1976;58(1):80–8.
56. Schaverien MV, Pessa JE, Rohrich RJ. Vascularized membranes determine the anatomical boundaries of the subcutaneous fat compartments. Plast Reconstr Surg. 2009;123(2):695–700.
57. Stuzin JM, Baker TJ, Gordon HL. The relationship of the superficial and deep facial fascias: relevance to rhytidectomy and aging. Plast Reconstr Surg. 1992;89(3):441–9.
58. Macchi V, Tiengo C, Porzionato A, et al. Histotopographic study of the fibroadipose connective cheek system. Cells Tissues Organs. 2010;191(1):47–56.
59. Thaller SR, Kim S, Patterson H, Wildman M, Daniller A. The submuscular aponeurotic system (SMAS): a

histologic and comparative anatomy evaluation. Plast Reconstr Surg. 1990;86(4):690–6.
60. Gosain AK, Yousif NJ, Madiedo G, et al. Surgical anatomy of the SMAS: a reinvestigation. Plast Reconstr Surg. 1993;92(7):1254–63.
61. Ghassemi A, Prescher A, Riediger D, Axer H. Anatomy of the SMAS revisited. Aesthetic Plast Surg. 2003;27(4):258–64.
62. Gardetto A, Dabernig J, Rainer C, et al. Does a superficial musculoaponeurotic system exist in the face and neck? An anatomical study by the tissue plastination technique. Plast Reconstr Surg. 2003;111(2):664–72.
63. Furnas DW. The retaining ligaments of the cheek. Plast Reconstr Surg. 1989;83(1):11–6.
64. Furnas DW. Festoons, mounds, and bags of the eyelids and cheek. Clin Plast Surg. 1993;20(2):367–85.
65. Muzaffar AR, Mendelson BC, Adams Jr WP. Surgical anatomy of the ligamentous attachments of the lower lid and lateral canthus. Plast Reconstr Surg. 2002;110(3):873–84.
66. Ghavami A, Pessa JE, Janis J, et al. The orbicularis retaining ligament of the medial orbit: closing the circle. Plast Reconstr Surg. 2008;121(3):994–1001.
67. Kikkawa DO, Lemke BN, Dortzbach RK. Relations of the superficial musculoaponeurotic system to the orbit and characterization of the orbitomalar ligament. Ophthal Plast Reconstr Surg. 1996;12(2):77–88.
68. Pessa JE, Garza JR. The malar septum: the anatomic basis of malar mounds and malar edema. Aesthet Surg J. 1997;17(1):11–7.
69. Mendelson BC, Jacobson SR. Surgical anatomy of the midcheek: facial layers, spaces, and the midcheek segments. Clin Plast Surg. 2008;35(3):395–404.
70. Mendelson BC, Muzaffar AR, Adams Jr WP. Surgical anatomy of the midcheek and malar mounds. Plast Reconstr Surg. 2002;110(3):885–96.
71. Mendelson BC. Surgery of the superficial musculoaponeurotic system: principles of release, vectors, and fixation. Plast Reconstr Surg. 2001;107(6):1545–52.
72. Uitto J. The role of elastin and collagen in cutaneous aging: intrinsic aging versus photoexposure. J Drugs Dermatol. 2008;7(2 Suppl):s12–6.
73. Varani J, Dame MK, Rittie L, et al. Decreased collagen production in chronologically aged skin: roles of age-dependent alteration in fibroblast function and defective mechanical stimulation. Am J Pathol. 2006;168(6):1861–8.
74. Mine S, Fortunel NO, Pageon H, Asselineau D. Aging alters functionally human dermal papillary fibroblasts but not reticular fibroblasts: a new view of skin morphogenesis and aging. PLoS One. 2008;3(12):e4066.
75. Kligman LH, Kligman AM. The nature of photoaging: its prevention and repair. Photodermatol. 1986;3(4):215–27.
76. Cavarra E, Fimiani M, Lungarella G, et al. UVA light stimulates the production of cathepsin G and elastase-like enzymes by dermal fibroblasts: a possible contribution to the remodeling of elastotic areas in sun-damaged skin. Biol Chem. 2002;383(1):199–206.
77. Hachiya A, Sriwiriyanont P, Fujimura T, et al. Mechanistic effects of long-term ultraviolet B irradiation induce epidermal and dermal changes in human skin xenografts. Am J Pathol. 2009;174(2):401–13.
78. Morita A, Torii K, Maeda A, Yamaguchi Y. Molecular basis of tobacco smoke-induced premature skin aging. J Investig Dermatol Symp Proc. 2009;14(1):53–5.
79. Yin L, Morita A, Tsuji T. Tobacco smoke extract induces age-related changes due to modulation of TGF-beta. Exp Dermatol. 2003;12 Suppl 2:51–6.
80. Kadunce DP, Burr R, Gress R, et al. Cigarette smoking: risk factor for premature facial wrinkling. Ann Intern Med. 1991;114(10):840–4.
81. Lucarelli MJ, Khwarg SI, Lemke BN, Kozel JS, Dortzbach RK. The anatomy of midfacial ptosis. Ophthal Plast Reconstr Surg. 2000;16(1):7–22.
82. Raskin E, Latrenta GS. Why do we age in our cheeks? Aesthet Surg J. 2007;27(1):19–28.

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