

Structural and Volumetric Changes in the Aging Face

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Abstract

Traditional views on facial aging and associated therapies focused on soft tissue descent and skin texture changes. Recently, revolumization for age-related lipoatrophy has become increasingly popular. Researchers are now reporting objective studies examining processes of structural and volumetric changes with age in increasing frequency as well. To better understand and treat facial aging, the surgeon should review all available data. Presented here are current evidence-based studies regarding age-related changes in the facial skeleton and soft tissue envelope. By reviewing these changes by each anatomic subsite, the surgeon will be better equipped to develop an appropriate treatment plan, individualized for each patient.

Keywords

- ▶ aging face
- ▶ lipoatrophy
- ▶ fat transfer
- ▶ facial skeleton
- ▶ facial rejuvenation

Facial volume loss as a part of the facial aging model has become increasingly accepted over the past decade. Although its popularity is a recent phenomenon, the concept is not new. Gonzalez-Ulloa and Flores described senile facial lipoatrophy nearly 50 years ago.¹ Measuring adipose thickness in various regions of the faces of newborn, young adult, and old cadavers, they found progressive volume loss with age. It is unclear whether their findings were not taken into further consideration because surgeons at the time did not accept the validity of the study, or there were inadequate techniques and materials to implement facial volume replacement at the time. With refinement of autologous fat transfer technique and availability of a variety of biomaterials and alloplastic implants, facial revolumization has rapidly expanded. Surgeons have reported improved outcomes when facial volume is addressed during facial rejuvenation.^{2–9}

Despite positive clinical outcomes, debate remains regarding the significance of volume loss and its contribution to the aged phenotype. Empirical investigations rather than observation are warranted to help answer this question. Furthermore, it stands to reason that surgeons would be better prepared to treat the aging face when equipped with objective information regarding facial aging mechanisms. With the knowledge of specific changes in each anatomic region, the surgeon can develop an algorithmic approach to properly

assess each patient according to their aging status and develop an appropriate treatment plan. The aim of this review is to present evidence-based research on age-related structural and volumetric alterations seen in each anatomic subsite of the face and discuss how these changes contribute to the aged phenotype.

Biological Mechanisms of Structural and Volumetric Aging

Although surgeons typically perform esthetic facial analysis using two-dimensional photographs, the face is a three-dimensional, multiplanar structure. Each layer undergoes its own aging process, and deeper layers will have an effect on those more superficial. The fact that the facial skeleton continues to change throughout adulthood^{10,11} is therefore fundamental to understanding and rejuvenating the aging face. In a study of facial bone resorption and deposition, Enlow reported that the frontal bone, nasal bone, ascending maxilla, and body of the mandible grew with age.¹² Conversely, the body of the maxilla, zygoma, coronoid process, and lingual surface of the mandible resorbed with age. These changes parallel findings in the aging facial skeleton discussed later.

Muscle action also contributes to facial skeleton remodeling. Moss described the concept of the “functional matrix”

and how surrounding tissues affect skeletal growth.¹³ The growing brain contributes to secondary growth of the calvaria at suture lines.¹⁴ The muscles of mastication are responsible for coronoid process and mandibular angle size and shape.¹⁵ Moss later revised his theory, including updates in research regarding mechanotransduction, the transformation of mechanical energy into an electrical or chemical signal to cells or tissues.¹⁶ The mechanical forces effected by muscle on bone can cause changes in the ionic milieu or molecular signaling pathways, altering osteocyte and osteoclast activity. In support of mechanotransduction, Staley et al reported alterations in facial growth of patients with myotonic dystrophy.¹⁷ Patients with myotonic dystrophy had longer faces than controls, suggesting that alterations in mechanotransduction of attached muscles affected facial skeletal growth patterns. Houston et al reported similar results in children with spinal muscular atrophy.¹⁸ Animal studies have confirmed that myectomy, neurectomy, or neuromodulation of mimetic or masticatory muscles affect facial bone growth.^{19,20} Chemical neuromodulation of facial muscles can affect not only static rhytids but also structural changes in the facial skeleton over time. Therefore it should be examined further to determine the role each mimetic and masticatory muscle group plays in mechanotransduction.

Data on the cellular and molecular mechanisms of senile lipotrophy is lacking. Recent studies of mesenchymal stem cells and aging may elucidate this process. Adipose tissue and bone marrow contain a small population of stromal pericytes that are capable of expansion, differentiation, and maintenance of a rest of cells called mesenchymal stem cells or mesenchymal stromal cells (MSCs).²¹⁻²³ These cells can differentiate into osteocytes, chondrocytes, myoblasts, and adipocytes. Studies have demonstrated that MSC function decreases with age.²⁴⁻²⁸ Although differentiation capacity remains, replication potential declines. These age-related alterations may contribute to facial volume loss. Recent studies have shown that fibrous septa separate facial fat into several compartments which change with age independent to each other.^{29,30} Mechanotransduction may also play a role in aging of facial fat components.³¹ Further studies are warranted.

Although understanding of the underlying mechanisms of facial volume and structural aging is valuable, knowledge of how those changes affect each facial anatomic region will give the surgeon a framework in which to consider each patient's rejuvenation.

Brow, Upper Eyelid, and Temple

Changes in soft tissue volume have been implicated in age-related changes in brow position. Some surgeons report that adding volume to brow tissue obviates the need for most brow lifting procedures.^{8,32,33} To investigate changes in brow soft tissue volume with respect to age, Papageorgiou et al measured brow soft tissue volumes in 52 computed tomographic (CT) scans of men and women.³⁴ They found that galeal brow fat increased with age for women but not in men. Muscle volumes tended to decrease with age for women, but

not in men. Overall, total volumes did not vary significantly for women with increasing age, but men showed a slight increase. Camp et al did demonstrate age-related volume decrease.³⁵ After registering three-dimensional images of mothers' faces to that of daughters', they found decreased volumes in the medial upper lid in the images of the mothers.

Age-related changes in the superior orbital rim (SOR) may contribute to apparent volume loss and hollowing of the brow and upper lid. Pessa and Chen reported orbital rim distortions associated with aging.³⁶ In 30 male European skulls, they identified superomedial curve distortion in the older specimens. Shaw and Kahn demonstrated decrease in the glabellar angle with age.³⁷ These authors also reported age-related superomedial remodeling of the SOR.³⁸ Changes were greater in the female cohort. Weaver et al confirmed increase in SOR height with age.³⁹ Shaw et al demonstrated similar findings in a larger cohort.⁴⁰ These studies suggest that the SOR undergoes superomedial widening with age. This change could be responsible for the "A-frame" deformity.⁴¹

Little data are available on aging in the temporal region. In a cephalometric study of 600 male and female patients of various ages, Farkas et al demonstrated decreased facial width at the level of the forehead with age.⁴² Skin surface measurements decreased commensurate with bony surface changes, suggesting skeletal framework remodeling was responsible for loss of facial width in the upper third of the face. In a cross-sectional study, Wysong et al examined magnetic resonance imaging (MRIs) of patients in various age groups and measured soft tissue thickness superficial to the temporalis at the level of the midorbit.^{43,44} They identified age-associated soft tissue thinning in both men and women.

Lower Eyelid and Lid-cheek Junction

The lower periorbital region is complex. Multiple tissue types and anatomic structures pose a challenge to appropriate treatment. Surgeons must consider the lower lid fat pad, upper cheek fat, orbicularis oculi, and underlying bony framework. Traditionally, surgeons believe weakening of the orbital septum and orbicularis to be the underlying pathology responsible for lower lid fat herniation.^{45,46} Pessa et al studied age-related changes in inferior orbital fat, rim, and cheek projection in males and females.⁴⁷ The older age group showed retrusion of the inferior rim and the cheek projection relative to the cornea, that is, increased "negative vector" with age. The lower lid fat pad projection did not change significantly. Age-related changes were greater in males. In a CT study of stereolithographs, inferior orbital rim (IOR) appeared to be recessed relative to the SOR with age as well.⁴⁸ Pessa and Chen identified inferolateral displacement of the IOR with age.³⁶ However, Mendelson et al did not find any age-related differences in the length of the superior or inferior orbital floor.⁴⁹ Chen et al also examined lower lid fat herniation relative to globe and orbit position on CT scans.⁵⁰ Measuring the curvature of lower lid fat on axial view, they found a small but significant increase in fat herniation with age. They also found an increase in fat herniation relative to the inferior orbital rim on sagittal

view, but this did not reach significance. No changes were identified in the position of the globe relative to the superior and inferior orbital rims. Darcy et al measured intraorbital soft tissue volumes with MRI to determine changes associated with age.⁵¹ They found that inferior orbital fat anterior to the globe axis and inferior orbital rim increased with age. Total orbital fat increased with age as well. No changes in globe position were identified relative to the SOR and IOR. Richard et al also found anterior migration of the inferior orbital fat pad.⁵² Lee et al reported similar findings in an Asian population.⁵³ Orbital fat volumes declined after 60 years of age, but not fat anterior to the IOR. Okuda et al found a positive correlation with age, fat herniation, and decreasing thickness of the orbicularis oculi.⁵⁴ Somewhat counter to the above studies, Ahmadi et al identified decreased ocular protrusion with age, with respect to the lateral orbital rim.⁵⁵ They did not measure IOR distance relative to the cornea.

Kahn and Shaw reported alterations in the inferior orbital aperture with age.³⁸ Women had greater change in the inferolateral portion, whereas changes were evident across the entire IOR length in men. They also identified increases in orbital width and aperture area with age for both sexes. Shaw et al. confirmed these findings in a larger study.⁴⁰

Authors have implicated weakening of the orbital septum with age or trauma.^{38,45,46} However, Camirand et al observed that patients with orbital septum tears do not develop lower eyelid fat pad herniation.⁵⁶ This suggests that the orbital septum does not play a role in the aged phenotype of the lower eyelid.

Traditionally, the cheek–lid junction was thought to descend with ptotic cheek fat with time.⁶ Lambros demonstrated that the lid–cheek junction was anatomically stable in 80 of 83 patients.⁴ Camp et al demonstrated volume loss in the nasojugal groove and lid–cheek junction when comparing mothers to daughters.³⁵ Wysong et al also identified decreased infraorbital soft tissue thickness in both men and women with aging.^{43,44}

The aforementioned studies suggest that posterior, inferior, and lateral remodeling of the inferior orbital rim influences the topography of overlying soft tissues and orbital fat, contributing to the aged phenotype. Increased orbital fat volume and thinning of the orbicularis oculi may contribute to herniation. Upper cheek fat appears to atrophy rather than descend. Further studies measuring the position of orbital structures are warranted to further elucidate changes in orbital fat and globe position over time.

Nasolabial Fold and Cheek

Traditional teachings posit that descent of midface soft tissues is responsible for the prominence of the nasolabial fold and hollowing of the malar mound.^{57,58} Recent investigations have challenged this theory. Lambros demonstrated stability of moles, wrinkles, and other skin marks on the midface in time lapse animation.⁴ Researchers have subsequently performed anatomic investigations comparing young and old patients to ascertain the process of midface soft tissue aging. Gosain et al examined cheek projection in young and old

patients at the level of the nasolabial fold and midcheek.⁵⁹ They found age-related increases in medial and lateral cheek fat pad superficial to the levator labii superioris without a decrease in the upper portion of the fat pad. The MRI volumetric analysis confirmed hypertrophy of the medial fat pad with age.⁶⁰ Of note, there was no difference in mimetic muscle thickness or length with age. On the basis of these findings, the authors suggested ptosis was a greater contributor to midface aging than volume loss. Conversely, Wysong et al identified thinning of the soft tissues superficial to the levator labii superioris and superficial to the zygomaticus major in both men and women.^{43,44} Gosain's studies placed each patient in prone position, whereas Wysong studied MRIs of patients in supine position. These differences in study design may contribute to the opposing findings. Comparing MRI scans of subjects of various ages, Le Louarn et al demonstrated loss of fat deep to the mimetic muscles and thickening of superficial fat over time.³¹ The authors reasoned that the chronic action of the mimetic muscles gradually expelled deep fat into the superficial plane, causing loss of convexity and thus contributing to the aged phenotype. Rohrich et al identified the deep medial fat compartment, just superficial to the periosteum of the maxilla.⁶¹ As the authors were able to restore the youthful appearance of the cheek by augmenting deep medial fat pad compartment in an aged cadaver, they too hypothesized that volume loss in the deep fat pad contributed to the aging midface. Corey et al found no malar fat pad volume change in young versus old patients in an MRI study of 11 patients.⁶² However, the authors did not describe boundaries used for soft tissue measurement. Gierloff et al examined midface volume by separate fat compartments and compared 12 cadaver heads of various ages.²⁹ Each fat compartment was injected with contrast material and measured on CT. Their findings suggested descent of the nasolabial fat, medial cheek fat, and deep medial cheek fat with age. They also reported age-related change in contour of nasolabial fat. The projection was decreased in the upper third and increased in the lower third portion of the nasolabial fat pad. Medial cheek fat hypertrophied with age, but the changes were greater in the lower portion. Deep medial cheek fat volume decreased with age.

Because of the different imaging modalities and measurement parameters, it is difficult to reconcile the results of these studies. Midface skin appears to remain in a stable position over time. Superficial nasolabial fat may hypertrophy with inferior redistribution. The mimetic muscles do not appear to lose tension, and may even contribute to changes in fat distribution. Deeper fat compartments do appear to lose volume.

Skeletal remodeling may also contribute to midface volume loss and soft tissue descent. Pessa et al described decrease in maxillary height relative to orbital height with age.⁶³ As the maxilla shortens, soft tissue excess forms. The authors referred to this accordion-like repositioning of the soft tissues as the “concertina effect.” Bartlett et al described shortening of anterior–posterior skull dimensions for both men and women with age.⁶⁴ Pessa et al also investigated changes in bony midface projection with age.⁶⁵ They found that maxillary

projection receded relative to the upper face with age, both in men and women. Lambros suggested that midface aging could be conceptualized as clockwise rotation of the midface relative to the skull base (with the face facing right in profile view).⁴⁸ Pessa found that the pyriform and maxillary angles decreased with age, corroborating Lambros' hypothesis.⁴⁸ Shaw and Kahn also demonstrated age-related decreases in the pyriform and maxillary angle.^{37,66} Mendelson et al reported similar findings,

as did Richard et al.^{49,52} Levine et al argued that midface bony changes are not absolute.⁶⁷ In their study, they measured cephalometric values in a cross-sectional cohort of an unknown number of subjects. They found that the distance from the sphenoid to the anterior maxillary wall increased with age, which contradicted Bartlett's study. Such discrepancy may be because of variation in measurement or small sample size. Discrepancies may also be because of paradoxes which may

Table 1 Review of structural and volumetric changes by subsite

| Region | Structure | Change with age |
|------------------|-----------------------------|---|
| Temple | Superficial fat | Thinning in men and women ^{43,44} |
| | Bone | Decreased bitemporal width ⁴² |
| Brow | Fat | Galeal fat increases in women, not in men ³⁴ |
| | Muscle | Loss in women, not in men ³⁴ |
| | Total soft tissue | Slight increase in men, not in women ³⁴ |
| | Bone (superior orbital rim) | Superomedial aperture increase, greater in females ^{36,38-40} No change in orbital roof length relative to apex ⁴⁹ Decrease in glabellar angle ³⁷ |
| Upper eyelid | Total soft tissue | Volume loss ³⁵ |
| Orbit | Globe | No change in position relative to SOR and IOR ^{50,51} |
| | Orbital aperture | Increase in orbital width and aperture area ^{38,40} |
| Lower Eyelid | Orbicularis oculi | Decreased thickness ⁵⁴ |
| | Septum | No contribution to herniation ⁵⁶ |
| | Lower lid fat | No change relative to cornea ⁴⁷ Increased herniation relative to zygoma and medial orbital wall ⁵⁰ Increased herniation relative to IOR and globe axis ⁵¹⁻⁵⁴ Increased orbital fat volume ⁵¹ |
| | Bone (inferior orbital rim) | Retrusion relative to cornea, greater in males ^{47,48} No change in orbital floor length relative to orbital apex ⁴⁹ Inferolateral aperture increase ^{36,38,40} |
| Nasojugal groove | Skin | Stable vertical position with age ⁴ |
| | Soft tissue | Volume loss, decreased thickness ^{35,43,44} |
| Nasolabial fold | Skin | Stable vertical position with age ⁴ |
| | Superficial fat | Hypertrophy ³¹ with inferior redistribution ^{29,59,60} Thinning ^{43,44} |
| | Mimetic muscles | No change in thickness or length ⁶⁰ |
| | Deep fat | Thinning ^{29,31} Descent ²⁹ |
| Malar | Fat | No volume change ⁶² |
| | Bone (maxilla) | Regression/clockwise rotation ^{37,40,48,52,64,65} Expansion from the sphenoid ⁶⁷ Shortening ⁴⁷ |
| Upper lip | Skin | Thinning ⁷¹ |
| | Total soft tissue | Thinning at vermilion border and mid cutaneous lip ^{70,74} No change in total cross sectional area (thinning + lengthening) ⁷⁰ |
| | Fat | Increased thickness ^{71,72} |
| | Orbicularis oris | Thinning and flattening (J to L) ^{71,72} |
| Lower lip | Total soft tissue | Volume loss ⁷⁴ |
| Jawline | Bone (mandible) | Decrease body height, ramus height, body length ^{40,64,75} Increased mandibular angle ^{40,64,76} |

Abbreviations: IOR, inferior orbital rim; SOR, superior orbital rim.

arise when using “fixed” cephalometric points.¹² As each facial bone has its own deposition/resorption pattern, registering images to standard cephalometric landmarks may not depict actual changes in each facial bone. Enlow recommended registering structures relative to known deposition and resorption patterns for each facial region, then creating a composite of skeletal changes to understand growth pattern appropriately.¹²

Despite the contradictory findings of Levine, the body of the literature weighs in favor of maxillary regression with age. These changes may cause loss of adequate support for the soft tissue envelope of the midface. The soft tissue itself appears to undergo deflation in the deep compartments and inferior redistribution with hypertrophy of the superficial compartments.

Perioral Region

Several authors have postulated that volume loss contributes to perioral aging.^{68,69} However, empirical data have been lacking until recently. Iblher et al performed photomorphometric and MRI evaluation of 172 male and female patients of various ages.⁷⁰ They found that mucosal upper lip height decreased with age, suggesting volume loss. Lip thickness at the vermilion border and cutaneous mid upper lip also decreased with age. Upper lip height increased with age, but the total cross-sectional area of the upper lip did not change. These findings suggest that inferior redistribution of upper lip volume contributes to lip thinning. Penna et al performed histologic analysis on 20 female lip biopsies to determine correlations to the aforementioned MRI data.⁷¹ They found that skin thickness decreased with age, whereas subcutaneous fat increased in the upper lip. Orbicularis oris showed signs of decrease in thickness and flattening of cross-sectional conformation, which contributed to a flattening of the upper lip complex with age. The authors noted that patients of the same age had different aging changes to varying degrees, suggesting the need for individualized approaches to rejuvenation.⁷² In their longitudinal cephalometric study of 39 men and women aged 17 to 61 years, Pecora et al also identified lengthening and thinning of the upper and thinning of lower lip soft tissues.⁷³ Using three-dimensional digital analysis of perioral regions of 918 men and women of various ages, Sforza and Ferrario found that upper and lower lip volume decreased with age, as did vermilion thickness.⁷⁴ No further studies on lower perioral volume changes have been performed.

Neck/Jawline

Shaw et al evaluated CT scans of 120 mandibles of men and women of various ages.^{40,75} They reported age-related decreases in body height, ramus height, and body length. Mandibular angle increased with age. These findings confirm data reported by Bartlett et al.⁶⁴ Pecora et al reported lengthening of the mandible and midface, and increase in soft tissue at the menton and pogonion with age.⁷³ The early age of the T1 cohort (mean age 17 years) in the Pecora study

may be responsible for increased mandible measurements, as the facial skeleton has yet to mature. They reported no statistically significant changes in the mandible from T2 (mean age 46 years) to T3 (mean age 57 years). Radiologic studies of soft tissue volume changes in the prejowl sulcus and jowl area are lacking. However, it would seem reasonable that as the mandible retracts, skin remains tethered at the mandibular ligament, creating soft tissue excess.

Conclusion

The process of facial aging is complex and involves changes in each tissue type. A summary of referenced findings are included in **Table 1**. Contradictory reports are included for completion. Skeletal alterations affect soft tissue support. Chronic muscle tension may contribute to skeletal changes through mechanotransduction. Meanwhile, fat compartments may atrophy, hypertrophy, or migrate. This migration may be secondary to weakening of the aponeurotic attachments, differential volume changes, or the loss of skeletal support. Taking these facial aging patterns and mechanisms into account, surgeons should nevertheless strive to individualize treatment plans for each patient. The ultimate goal is to restore each patient's own youthful appearance. To understand how faces age on an individual basis, more longitudinal studies are needed. Anatomic analyses should follow Enlow's suggestion that each region be analyzed independently and then aggregated to create a complete facial aging analysis. Comparison of studies requires greater consistency in age spans and age groupings. Regardless of the outcome of future studies, it is clear that volumetric and structural changes are major components of facial aging and must be considered in facial rejuvenation.

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