Facial Rejuvenation in the Aging Population

Jeffrey A. Fialkov, MD, MSc, FRCSC, Assistant Professor, Division of Plastic Surgery, Department of Surgery, University of Toronto; Staff Plastic Surgeon, Sunnybrook Health Sciences Centre, Toronto, ON.

This article reviews surgical and nonsurgical rejuvenation techniques as they relate to the anatomic changes that occur with facial aging. An understanding of the changes that occur to the facial soft tissues and their support structures over time and with exposure to the elements facilitates individualized treatment optimization for older adults seeking facial rejuvenation. In addition, treatment optimization must take into account the patient's underlying medical status and personal psychosocial concerns.

Key words: *facial rejuvenation, cosmetic surgery, facial aging, noninvasive rejuvenation, photoaging*

Introduction

Cosmetic procedures and treatment regimens to reverse and reduce the stigmata of aging have burgeoned in the last few decades. The availability of an increasing number of nonsurgical procedures, offering quicker recovery times, less physical sequela, and less morbidity than surgery, has created a confusing consumer environment for the prospective patient (Table 1). Often touted as replacement treatments for more invasive surgery, the latter are geared primarily towards the repositioning of ptotic soft tissues, these less invasive procedures should, rather, be considered primary treatment options to address specific changes in the inherent quality of aging skin, fat, and muscle: inherent changes that surgery may have a limited effect in addressing. An understanding of the anatomic changes that occur with aging, the factors that affect the rate of these changes and ultimately, the role and risks of surgical and nonsurgical treatment regimens in addressing specific changes is critical to ensure that individuals seeking rejuvenation therapies receive optimal treatment.

For the purposes of this paper, a surgical procedure will be defined as any treatment that involves incising the skin. Nonsurgical procedures will be defined as any treatment that does not involve incising the skin. The distinction is an important one, in that incising the skin, depending on the size of incision and any subsequent tissue dissection, carries with it a significantly higher risk of complications due to surgical trauma (infection, bleeding, nerve and muscle injury) as well as risks of anesthetic administration, particularly general anesthetic. In contrast, injection, although still carrying with it inherent risks of bleeding, infection, and even injury to underlying tissues, is significantly less likely to be complicated by these risks.

Inherent Soft Tissue Changes versus Ptosis in Facial Aging

The stigmata of facial aging (Figure 1) may be attributed to inherent changes in the skin, fat, and muscle (i.e., soft tissue) of the face, in addition to a loss of ligamentous support suspending those same soft tissues to the underlying facial skeleton (Figure 2). Inherent soft tissue changes relate to the quality of the skin, volume of fat, and muscle tone (Table 2) whereas changes due to aging in the structural support of these soft tissues result primarily in a caudal displacement of these soft tissues, i.e., ptosis (an abnormal lowering or drooping). Although both inherent soft tissue and structural support changes arise as a result of the aging process itself (intrinsic factors), exposure to ultraviolet light, smoking, heredity, and mimetic muscular activity (extrinsic factors) contribute significantly to the rate and extent of these changes.[1] Chief among these factors affecting the skin is exposure to ultraviolet light (photoaging), whereas gravity is the primary force resulting in the gradual attenuation of the suspensory support structures and consequent soft tissue ptosis. Both categories of change can produce an aged look, and the degree to which each type of change contributes to this look will vary depending on the patient's age at presentation, environmental exposure, and heredity.[2]

In general, loss of structural support (i.e., ptosis) is best addressed with surgical resuspension procedures. These include brow lifting for brow ptosis, upper and lower blepharoplasty, which addresses the loss of eyelid structural support (levator aponeurosis and septum orbitale) as well as the eyelid skin changes due to photoaging, and rhytidectomy (face and neck lifting). Inherent soft tissue changes are generally best addressed with nonsurgical procedures. This rule-of-thumb is not without exception. Patients' tolerance of more invasive procedures, both in terms of medical risk as well as psychosocial preference, must be of paramount consideration when counselling patients about rejuvenation procedures. In instances when more invasive procedures are less desirable or medically contraindicated, nonsurgical rejuvenation procedures may partially mask some of the structural support-related stigmata of facial aging, otherwise best addressed surgically.[3]

Inherent Soft Tissue Changes and Photoaging

Photoaging results in solar elastosis and collagen degeneration. Histologically, this manifests as an accumulation of elastotic material and a disruption of the normal

Table 1: Nonsurgical Rejuvenation Procedures

Treatment	Example	Addresses	Advantages	Disadvantages
Injectable fillers (temporary)	Hyaluronic acid collagen	Static rhytids that result from photoaging or repeated mimetic muscular activity	Office procedure, minimal risk or morbidity	Temporary, injector dependent
Botulinum toxin	Botox®	Dynamic rhytids that result from mimetic muscular contracture	Office procedure, minimal risk; if started early may reduce progression of dynamic to static rhytids	Temporary, does not address static rhytids
Injectable fillers (permanent synthetic)	Artecoll	Volume deficiencies, static rhytids	Office procedure, permanent	Irregularities and malposition has to be addressed with surgical removal
Laser resurfacing	YAG, erbium, CO_2	Fine static rhytids and other skin changes due to photoaging	Effective, minimally invasive	Recovery time

collagenous architecture seen in youthful skin. Grossly photoaged skin is inelastic (lax), discoloured, and wrinkled. The wrinkles (rhytids) that are primarily a consequence of photoaging alone (static rhytids) may, unlike those rhytids that result from repeated mimetic muscular activity in elastotic skin, known as dynamic rhytids, arise in areas with minimal adherence of overlying skin to underlying musculature such as the mid cheek skin overlying the masseter.[4]

Nonsurgical resurfacing procedures (chemical peels, laser resurfacing) can address these changes of photoaging by changing the cutaneous architecture on a microscopic level, restoring some of the architecture with collagen deposition, and masking the more macroscopic effects of solar elastosis. Injectable fillers achieve this result by filling the creases of static rhytids.[3,5] In areas where mimetic musculature inserts into the overlying skin, dynamic rhytids are a consequence of repeated contracture of the adherent elastotic skin (the elastic quality of skin to an extent withstands the effect of this tissue fatigue phenomenon). Although these creases can be filled with injectable fillers, the effect is not dynamic. A more effective means of addressing these rhytids is chemodenervation of the underlying musculature with Botox®. Deeper residual creases that have become static as a consequence of the breakdown in dermal architecture due to tissue fatigue and elastosis can concurrently be treated with injectable filling. A typical example of such an area is in the glabella, where some of the depth of a given rhytid is attributable to the underlying corrugator tone, while some of the depth is attributable to the static changes in the cutaneous architecture; a result of repeated contracture, exacerbated by solar elastosis and intrinsic aging (Figure 3).[6]

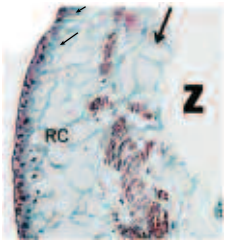
Known consequences intrinsic to the aging process, loss of muscle tone and fat atrophy occur in the face as they do generally throughout the aging body.[7] As an example; the seemingly cadaveric look of the aged periorbital region is in part due to loss of fat volume and orbicularis muscle tone.[8,9]

Figure 1: Stigmata of Facial Aging

White lines indicate stigmata primarily due to inherent soft-tissue changes. Black lines depict stigmata primarily due to ptotic changes that result from loss of ligamentous suspensory soft-tissue support. The grey lines depict changes that are due to a combination of both processes.

Source: Courtesy Dr. Jeffrey Fialkov.

Figure 2: Histological Section through Prezygomatic Soft Tissues showing Arborized Ligamentous Support System of Facial Soft Tissues.



Z: zygoma. Thick Arrow: suspensory ligaments from periosteum to superficial musculo-aponeurotic system (SMAS). Thin Arrow and RC: suspensory fibres extending from SMAS and inserting into dermis.

Source: Courtesy Dr. Jeffrey Fialkov.

Certain synthetic volumetric fillers, such as hyaluronic acid, in addition to autogenous fat (lipoinfiltration) offer a means of restoring this volume depletion. It is important to note that in the case of volume restoration as opposed to rhytid filling, the filler chosen can significantly affect the quality of results. This is because in the case of volume restoration the filler is injected into the relatively loose subcutaneous tissue plane whereas in the treatment of rhytids the filler is injected into, or just deep to, the very dense dermis. The effectiveness and persistence of the injectable material is consequently dependent on molecule size and viscosity.[5]

Ptosis and Loss of Ligamentous Support

The fat, skin, and mimetic musculature of the face are supported in position relative to the underlying facial skeleton by an arborized system of fibrous ligaments that originate from the periosteum, insert into the superficial musculo-aponeurotic system (SMAS), extend through the subcutaneous fat in an arborized pattern, and finally insert into the dermis (Figure 2). It is this relative position, in addition to the soft tissue volume that imparts a youthful appearance to the human face.

Intrinsic effects of aging, gravitational pull, and repeated loading by mimetic musculature all act to attenuate this ligamentous system and displace the facial soft tissues caudally. The result is the formation of ptotic stigmata of facial aging: brow ptosis, nasojugal (tear) trough, nasolabial fold, nasolabial crease, marionette lines and jowls, and obtuse cervico-mental angle. Exemplifying this process is the malar fat pad, a mound of subcutaneous fat that in youth rests over the malar prominence of the zygoma, and is supported in this position by the ligamentous network depicted in Figure 2. Over time this fat pad not only loses volume but also descends to the mid cheek, contributing to the formation of a cephalad hollow and a caudal fullness (infraorbital flattening, the nasolabial fold, and its consequential crease).[8]

A critical factor in the rate and extent of the descent of the facial soft tissues are the dimensions of the underlying facial skeleton, particularly facial skeletal width and projection. The bones that affect

Figure 3

a

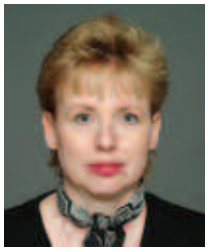
b

a) A patient in her late 4th decade with significant photoaging, dynamic rhytids and loss of eyelid structural support.

b) The same patient following upper and lower eyelid blepharoplasty and chemodenervation with Botox®. Note the residual static rhytids in the glabellar and frontal regions and the residual naso-jugal trough. These stigmata may be further addressed with injectable fillers.

Source: Courtesy Dr. Jeffrey Fialkov.

Figure 4



a

b

a) Type IIa patient (see Table 3) in her 4th decade with significant soft tissue ptosis due to mandibular hypoplasia before surgery

b) The same patient following chin augmentation and rhinoplasty. Note the correction of jowls, marionette lines and cervico-mental angle. No other procedures (specifically rejuvenation procedures) were performed.

Source: Courtesy Dr. Jeffrey Fialkov.

Figure 5

a

b

a) Type IIb patient (see Table 3) in her 5th decade, with adequate skeletal dimension, stigmata of ptosis, and photoaging.

b) The same patient following rhytidectomy to address ptotic changes. Residual static and dynamic rhytids can be addressed with injectable fillers and chemodenervation.

Source: Courtesy Dr. Jeffrey Fialkov.

Table 2: Inherent Soft Tissue Changes versus Ptosis in Facial Aging

Inherent Soft Tissue Changes (Photoaging and Atrophy)	Changes Due to Loss of Ligamentous Support (Ptosis)
Rhytids (wrinkles)	Nasolabial folds
Fat and muscle atrophy (hollowing)	Marionette lines
	Jowls
Actinic skin lesions and discolouration	Laxity in neck skin and muscle

these dimensions most significantly are the zygomas and the mandible, which define facial width and projection for the upper face and lower face, respectively. The greater the width and projection of the underlying facial skeletal dimensions, the greater the distance of soft tissue descent needed to form ptotic stigmata, and consequently the slower their development. In this regard, a useful analogy for patients is a blazer hanging on a coat hanger. The fuller the structure of the coat hanger, the longer and better preserved the shape of the blazer. Hanging the blazer on a wire hanger will result in a more rapid loss of shape due to gravity.

Thus, given an equal degree of photoaging, a patient who presents with proportionately small zygomas and/or a relatively small mandible will likely have a greater apparent age than a patient with a more proportionate facial skeleton. Furthermore, given an equal rate of photoaging, the relative contribution of ptosis versus inherent soft tissue changes to the development of the stigmata of facial aging will vary depending on underlying facial skeletal structure. This can be seen in some apparent premature aging of individuals in their third and fourth decade. Persons in this age range may have minimal photoaging but present with ptotic stigmata such as jowling due to mandibular hypoplasia or prominent nasolabial folds due to zygomatic hypoplasia (Figure 4).[9,10]

Surgical resuspension, by means of face, neck, and brow lifting as well as eyelid surgery, is designed to address the results of age-related attenuation of the soft tissue structural support system by repositioning ptotic structures (skin, fat, and muscle) to their preptotic position. These procedures are optimal for individuals with adequate underlying skeletal width and projection, and significant ptosis. These patients generally present in their fifth and sixth decades (Figure 5). Inherent soft tissue changes in these patients may be treated with concurrent or subsequent nonsurgical procedures.

Bony skeletal augmentation, such as porous polyethelene implant fixation to

Figure 6

a

b

a) Type III patient in her 5th decade with minimal photoaging but significant ptotic stigmata of aging before surgery.

b) The same patient following zygomatic (malar) augmentation, mandibular (chin) augmentation, and rhytidectomy.

Source: Courtesy Dr. Jeffrey Fialkov.

Table 3: Facial Aging Treatment

Type	Skeletal dimension	Photoaging	Ptosis	General Age at Presentation	Primary Rejuvenation Technique
I	Adequate	Minimal-moderate	Minimal	4th decade	Resurfacing Chemodenervation Fillers
IIa	Deficient	Minimal	Minimal to moderate	3rd and 4th decade	Skeletal augmentation
IIb	Adequate	Moderate-severe	Moderate-severe	5th–6th decade	Rhytidectomy + Adjuvant resurfacing, fillers, chemodenervation
III	Deficient	Moderate-severe	Moderate-severe	5th–6th decade	Skeletal augmentation, rhytidectomy + Adjuvant fillers, chemodenervation and resurfacing

the zygomas (malars) or mandible (chin) is a surgical technique that may adequately reverse the ptosis in those younger patients with minimal ligamentous attenuation of aging, or contribute to the longevity of resuspension procedures in those patients with skeletal deficiency and age related soft tissue ptosis (Figure 6). The desired result of this treatment regimen is the delay of ptosis redevelopment with a view to precluding or minimizing the need for further surgery in later years.

In patients with adequate skeletal dimension, or in those who have had skeletal augmentation, the duration of soft tissue resuspension procedures is maximized. Therefore, in later years (seventh and eighth decade) when the likelihood of these patients developing underlying medical conditions that will increase the risk of a general anesthetic or lengthy surgical procedures, is greater, lower risk nonsurgical treatments may provide adequate rejuvenation by addressing the stigmata of photoaging and fat atrophy or masking more minimal ptotic changes.

Conclusion

The stigmata of facial aging arise as a consequence of changes in the inherent microscopic structure of skin, volume of fat, and tone of mimetic musculature of the face, as well as loss of soft tissue suspensory support relative to the underlying facial skeleton. These changes are a result of the intrinsic aging process as well as extrinsic environmental factors, chiefly UV exposure, mimetic muscular activity and gravity.

The wide variety of surgical and nonsurgical rejuvenation procedures available offer specific means of addressing these changes (Table 3). In general, surgical resuspension procedures address ptosis of the facial soft tissues due to intrinsic suspensory ligamentous attenuation and gravity while nonsurgical resurfacing, soft tissue volume augmentation (filling), and chemo- denervation (Botox®) address the inherent soft tissue changes of photoaging, solar elastosis, and fat atrophy. Nonsurgical rejuvenation techniques may, however, offer an alternative to more invasive surgery, for patients in which prolonged operative time and general anesthetic pose a significant risk, or in those patients who, for psychosocial reasons prefer not to undergo surgery.

In addition, facial skeletal augmentation is a means of more effectively addressing the ptotic changes due to aging and prolonging the results of surgical resuspension. It may be the optimal procedure in younger patients (third and fourth decades) with less extrinsic soft tissue changes in contradistinction to a patient presenting in later life (fifth and sixth decades) who may benefit from both skeletal augmentation and resuspension. The latter group of patients will likely develop stigmata of facial aging in subsequent decades (seventh and eighth) that are predominantly due to photoaging and inherent soft tissue changes, making them more amenable to nonsurgical rejuvenation techniques at a stage in life in which they are more likely to develop medical conditions which put them at a higher surgical and anesthetic risk category.

No competing financial interest declared.

References

1. Kadunce DP, Burr R, Gress R, et al. Cigarette smoking: risk factor for premature facial wrinkling. Ann Intern Med 1991;114:840–4.
2. Lambros V. Observations on periorbital and midface aging. Plast Reconstr Surg 2007;120:1367–76.
3. Carruthers A, Carruthers J. Non-animal-based hyaluronic acid fillers: Scientific and technical considerations. Plastic Reconstr Surg 2007;120:33S–40S.
4. Mendelson BC, Freeman ME, Wu W, et al. Surgical anatomy of the lower face: the pre-masseter space, the jowl, and the labiomandibular fold. Aesthetic Plastic Surgery 2008;32:185–95
5. Fagien S, Klein AW, A brief overview and history of temporary fillers: evolution, advantages, limitations. Plastic Reconstr Surg 2007;120;6s–16s.
6. Zimbler M, Nassif P. Adjunctive applications for botulinum toxin in facial aesthetic surgery. Plastic Surg Clin North Am 2003;11:477–82.
7. Faulkner JA, Brooks SV, Zerba E. Muscle atrophy and weakness with aging: contraction-induced injury as an underlying mechanism. J Gerontol A Biol Sci Med Sci 1995;50:124–9.
8. Rohrich, RJ. Multiple, combined plications of the SMAS-platysma complex: breaking down the aging-face vectors. Plast Reconstr Surg 1999;104:1101–2.
9. Owsley JQ, Roberts CL. Some anatomical observations on midface aging and long term results of surgical treatment. Plast Reconstr Surg 2008;121:258–68.
10. Pessa JE, Peterson ML, Thompson JW, et al. Pyriform augmentation as an ancillary procedure in facial rejuvenation surgery. Plast Reconstr Surg 1999;103:683–6.
11. Friedman C, Constantino P. Alloplastic materials for facial skeletal augmentation. Plastic Surg Clin North Am 2002;10:325–33.