Oculoplastic Surgery

Special Topic

Malar Mounds and Festoons: Review of Current Management

Dzifa S. Kpodzo, MD, MPH; Foad Nahai, MD; and Clinton D. McCord, MD

Abstract

Blepharoplasty, the most common aesthetic eyelid procedure, sometimes involves a challenging patient subgroup: those who present with malar edema, malar bags, and festoons. In this review article, the authors describe the relevant anatomy in festoon development, discuss the pathophysiological basis of this condition spectrum, outline clinical examination basics, summarize various surgical approaches for treatment and propose an algorithm for their application, and describe the most common postsurgical complications.

Keywords

festoons, malar mounds, malar edema, facelift, blepharoplasty, periorbital rejuvenation, oculoplastics

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Blepharoplasty is an immensely popular aesthetic procedure. A report by the American Society for Aesthetic Plastic Surgery (ASAPS) cited blepharoplasty as the fourth most frequently performed aesthetic surgical procedure, with approximately 153,000 cases in 2012.1 Periorbital rejuvenation is an important part of restoring an overall youthful appearance; the goal is to restore a smooth and harmonious contour from brow to cheek. For the lower eyelid, that means identifying and correcting any problems around the lid/cheek junction. Particularly challenging are patients presenting with chronic malar edema, malar mounds, and festoons of the lower eyelid and cheek. Goldberg et al2 examined 114 consecutive patients to analyze different anatomical contributions to aesthetic concerns of the lower eyelid. They found the cumulative contribution of malar edema and malar mounds to lower eyelid appearance in their patients was 32% (n = 38) and 13% (n = 15), respectively. These problems occur often enough that a thorough understanding of the causes and treatment is essential.

Some authors have published their approach to addressing these challenging problems, and as often occurs in our field, the techniques vary. Since Furnas’s treatise in 1978,3 few articles have outlined a comprehensive approach to treating these conditions. A major challenge in reaching standard-of-care consensus is the variable terminology describing these problems. Terms used in the literature include malar mounds, malar bags, saddle bags, eyelid bags, cheek bags, palpebral bags, fluid bags, secondary bags, malar edema, triangular cheek festoon, and festoons. Inaccurate or incorrect use of diagnostic terminology increases the possibility of incorrect treatment.4 In this

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In this article, we review the relevant anatomy, discuss new findings about possible etiology of festoons, describe the clinical examination’s salient points, and summarize current surgical approaches. We also propose an algorithm for approaching the challenge of treating festoons, thus making it easier for surgeons to select appropriately from among the many surgical techniques.

**ANATOMY**

The lower eyelid is a complex structure containing multiple layers. From anterior to posterior, those layers are the skin, subcutaneous fat, orbicularis oculi muscle, suborbicularis oculi fat (SOOF), the orbital septum that fuses with...
the tarsal plate superiorly and capsulopalpebral fascia inferiorly, and the periorbital fat pads. The tarsoligamentous sling, stretching from the medial to lateral canthus, provides support.

To better understand the development of malar edema, malar mounds, and festoons, understanding the anatomy and supporting structures of the lower eyelid and cheek is essential. Several authors have made important contributions in this regard. To understand the development of malar edema, malar mounds, and festoons, understanding the anatomy and supporting structures of the lower eyelid and cheek is essential. Several authors have made important contributions in this regard.5-9 There are 2 important anatomic areas when considering this spectrum: the lid/cheek junction and midcheek that together create the borders of the prezygomatic space.

We will first discuss the lid/cheek junction. Described by Furnas9 as the “orbit-cheek fold,” this area is at the level of the orbital rim and is consistent with the location of the orbitomalar ligament (OML). First described by Kikkawa et al,6 the OML delineates separation between the lower eyelid and malar eminence. An osteocutaneous structure originating from the orbital periosteum at the level of the arcus marginalis, the OML penetrates the orbicularis oculi muscle, terminating in multiple cutaneous insertions at the lid/cheek junction.10 Muzaffar et al7 described this structure further, naming it the orbicularis retaining ligament (ORL). The ORL provides an indirect “attachment of the orbicularis to the [orbital] rim.”9 It closely attaches the orbicularis muscle to the orbital rim medially above the levator labii superioris and laterally at the lateral orbital thickening.7 The length of the ligament is maximal centrally, measuring 10 to 14 mm (see Figure 2). The ORL/OML marks the upper border of the area where festoons develop. For consistency in nomenclature, we will refer to this structure as OML throughout this article.

The lower border of this condition is in the midcheek, where Furnas9 described the presence of a “midcheek fold.” The midcheek fold’s location is consistent with the location of Pessa and Garza’s malar septum, found 2.5 to 3 cm below the lateral canthus.5 The malar septum, a thin impermeable membrane, extends from the inferior orbital rim to the cheek skin.5 From its origin at the rim, the malar septum crosses the SOOF (creating both a superior and an inferior SOOF), penetrates the orbicularis muscle, and finally interdigitates with fibrous septa to insert into the midcheek dermis.5 Muzaffar et al7 and Mendelson et al8 defined the lower border as the zygomaticocutaneous ligament. They described the prezygomatic space bordered by the OML above, zygomaticocutaneous ligaments below, and 2 distinct layers of fat: preperiosteal fat and SOOF.7,8 Although they did not specifically identify the malar septum, they hypothesized that it “is a reflection of the membrane covering the preperiosteal fat and caudal boundary of the pre-zygomatic space.”7 The malar septum and underlying zygomaticocutaneous ligaments mark the inferior border of the area where festoons develop (Figure 3).

**PATHOPHYSIOLOGY**

Many theories abound concerning festoon etiology. Furnas11 described the festoon as a sagging hammock of orbicularis oculi muscle carrying skin with it. Furnas theorized that the combination of senility and gravity results in...
the attenuation—or degeneration—of orbicularis muscle; this in turn stretches the overlying skin, resulting in festoons. Although there is consensus with Furnas’s determination that festoons consist of attenuated orbicularis oculi muscle with overlying skin excess, causes of this attenuation continue to be debated. In a case report, Goldman described the presentation of bilateral festoons after periorbicular Botox (Allergan, Irvine, California) injections in a patient who had previously undergone quad blepharoplasty. The patient received 2 units of Botox in the lower eyelid 2 to 3 mm below the ciliary margin and 10 units of Botox to the crow’s feet. Goldman hypothesized that “the muscular pumping action of the lower eyelid [was] weakened, causing localized lymphedema and a transient ‘festoont’. This case demonstrates a clear connection between muscular attenuation and edema as causes for festooning.

Anatomical studies have yielded further insight into the pathophysiology of this morphological/aging change. In their cadaver dissections, Muzaffar et al cited OML as key to aging changes of the lower eyelid and appearance of malar mounds. Elongation of OML marks the floor for bulging eyelid fat at the lid/cheek junction and the roof of the prezygomatic space containing malar edema, bags, and festoons. As OML stretches, it causes descent of the lid/cheek junction, orbicularis attenuation, and “redundancy of the soft tissue immediately below.” However, the floor of the prezygomatic space—marked by the impermeable malar septum and zygomatic ligaments—is much stronger than OML. This results in “downward sliding of the tissues of a poorly supported roof of the prezygomatic space against the resistance of the more strongly supported lower boundary” (Figure 4).

In their anatomic study of malar mounds, Pessa and Garza proposed that festoons result from malar edema progression, causing malar mounds and eventually resulting in festoons. They hypothesized that the consistent location of malar mounds in all age groups seemingly argues against senile ptosis or gravity as a reason for malar mound development. Rather, they proposed that chronic malar edema above the impermeable malar septum may cause malar mounds, with chronic distention resulting in attenuation of orbicularis oculi muscle and overlying skin and, thus, festoons. There is still knowledge to seek and more research must be done to better understand how delayed lymphatic drainage contributes to the conditions.

We propose that festoon pathophysiology is multifactorial. The triangular shape and consistent location of malar edema, bags, and festoons (3 cm below the lateral canthus) supports the importance of the prezygomatic space and its boundaries. Orbitomalar ligament stretching can be senile or due to primary laxity, thus explaining why malar bags are sometimes seen in young people. Based on patient history and examination, we agree that lymphatic imbalance and “predisposition to prolonged post-operative swelling” are contributing factors in patients in this spectrum. Either 1 or a combination of these 2 factors results in attenuated orbicularis oculi muscle and excess skin over edematous fat.

**HISTORY AND CLINICAL EXAMINATION**

Prior to clinical examination, obtain a complete medical history from the patient; many systemic and localized illnesses cause periorbital swelling, thereby masquerading as age-related eyelid changes. We will limit this broad topic to a brief discussion, as it falls outside the scope of this review. Take careful note of patients who describe “overnight” changes to the eyelid. Changes should occur slowly over years rather than weeks or months. Unilateral changes should also raise a red flag, as these can be secondary, for example, to primary cutaneous B-cell lymphoma or peripheral T-cell lymphoma. Medications can also cause eyelid edema; common culprits are steroids, acetaminophen, nonsteroidal anti-inflammatory drugs, and an exhaustive list of other offenders. Allergies, a frequent cause of eyelid swelling, can be distinguished by the presence of vertical rather than the usual horizontal rhytids, erythema, and, in the case of urticarial reactions, thickened eyelid skin. Ocular rosacea is another cause of eyelid edema, particularly at the eyelid margin, and these patients usually have blepharitis or meibomitis and an erythematous and thickened lid margin. Additionally, inquiring about history of thyroid disease and related symptoms is essential, as thyroid eye disease is a major cause of periorcular edema. Sometimes these patients are undiagnosed and present...
first to plastic surgeons with 1 or several of the following signs of orbitopathy: dry eyes, periorbital edema, conjunctival edema, proptosis, and lid retraction. Finally, eyelid edema may be associated with whole-body edema in cardiac, renal, or hepatic disease or hypothyroidism. Before proceeding to surgery, all systemic illnesses should first be resolved or addressed.

The clinical examination begins with a standard evaluation of the eyelids, as well as 2 maneuvers specific to festooning. Furnas’s suggested the following 2 techniques for evaluating festoons.

“Squinch” Test
The patient is asked to animate the orbicularis oculi muscle by squeezing the eyelids tightly together. This effort results in maximal voluntary contraction of orbicularis muscle, thereby improving the appearance of the cascade of attenuated muscle. This maneuver gives the surgeon a sense of the degree of muscle involvement responsible for the appearance of the festoon. If orbital fat is suspended within the festoon, it will not be effaced but rather change position with the muscle contraction. The fat, displaced closer to the ciliary margin during muscle contraction, returns to its original position as the muscle relaxes. This indicates to the surgeon that fat excision must be part of the surgical plan.

Pinch Test
Moving systematically, the surgeon pinches the skin of the festoon at different sites while the patient makes expressive movements in tandem. Tissue resistance may provide additional information about the degree of orbicularis oculi bulk contributing to the festoon.

SURGICAL APPROACHES

Microsuction
Some authors have proposed microsuction in the subcutaneous plane as an important tool for addressing malar edema and malar bags. Some have postulated that malar edema mostly exists in the subcutaneous plane superficial to orbicularis oculi muscle. In this plane, the cutaneous insertions of OML serve to separate the malar mound from the eyelid. According to Rosenberg, the malar mound is caused by expansion of fat in the subcutaneous plane. He postulated that left long enough, expanding subcutaneous tissues stretch the overlying skin, causing festooning. Excess fat in the subdermal plane can be suctioned using a small-caliber liposuction cannula (2.3 mm in Rosenberg’s technique paper). The suctioning is halted once the “bulge has been removed, and the fat is no longer palpable.” A mild compressive dressing is applied, allowing the excess skin to redrape on its own. Rosenberg reported no skin excision with this technique but recommends that, if necessary, it be completed after the initial procedure.

Liapakis and Paschalidis proposed a combined approach involving microsuction with lateral suspension of the orbicularis oculi muscle to the temporalis muscle. Their article described a 45-year-old female patient with persistent malar edema that developed into malar bags 2 years after blepharoplasty. The patient’s subcutaneous malar area was treated with tumescent liposuction using a 3-mm blunt-tipped liposuction cannula. Then, via a temporal incision and a small lateral canthal incision, the lateral orbit, inferior orbital rim, and entire midface were extensively mobilized in the subperiosteal plane, followed by lateral orbicularis oculi suspension to temporal fascia. As previously discussed, it is important to understand that this extensive subperiosteal dissection in the midface releases the periosteal attachments of the OML as well as the malar septum. The impermeable barrier to malar edema drainage is addressed through this release. After this extensive midface dissection, the lateral suspension suture to the temporalis fascia provides additional eyelid support. Both the above-discussed approaches are effective only for patients with malar edema or bags with limited skin excess; they are not options for patients with festoons.

Skin-Muscle Flap
Several authors have advocated the skin-muscle flap as a means of addressing festooning. Most approaches involve some type of differential skin and muscle treatment, whether by degree of dissection, excision, or suspension. In Furnas’s 1978 article, he advocated making a subciliary incision and dissecting a skin-muscle flap down to the orbital rim. This provided the needed separation between flap and rim to accomplish adequate upward mobilization. Here again, separating flap from orbital rim results in OML division, thereby releasing the roof of the malar mound or festoon. The flap is then advanced with the degree of upward and lateral vector needed to adequately efface the festoons. The excess tissue is excised, beveling the scissors such that more muscle is removed than skin. The muscle is then suspended to the periosteum, followed by muscle-to-muscle sutures and, finally, closure of the skin.

Furnas also described an alternative muscle-plication technique where a skin-only flap is developed: the muscle is plicated laterally to itself and the periosteum, followed by skin excision and closure. Rees and Tabbal described a similar skin-muscle flap technique but added that patients with severe festoons may require dissection beyond the “infraorbital rim so that the soft tissues are
freed extensively over the anterior surface of the maxilla.” An extensive dissection over the anterior maxilla may likely release the malar septum and zygomaticocutaneous ligaments responsible for creating the prezygomatic space’s floor. Finally, the skin-muscle flap technique may require complete undermining of the skin from muscle and separate redraping to adequately address anatomical problems.

**Midface Lift**

We firmly agree with Pessa and Garza’s anatomical analysis5 of malar mounds and festoons, as well as the importance of releasing the OML and malar septum to successfully treat these conditions. Previous authors agree that release and resuspension of OML, followed by a variable amount of skin excision, is a highly effective approach to correcting malar mounds.19,20 The article by Hoenig et al19 described their technique of the video-assisted endoscopic subperiosteal vertical upper-midface lift (SUM-lift). A subperiosteal dissection of the malar region was completed using a temporal and a buccal sulcus incision; the midface then was mobilized to the desired position, and sutures were placed to secure it to the deep temporal fascia. The technique by Hoenig et al addressed the lower eyelid through a subciliary incision and suspension of the orbicularis oculi muscle to the lateral orbital rim. Le Louarn20 described a “concentric malar lift” in an effort to correct natural vectors of the aging process around the eyelid. Following a subperiosteal midface dissection, 3 drill holes were placed: the first just above the lateral canthus for vertical orbicularis suspension, the second at the intersection of the lateral and inferior orbital rims for vertical malar suspension, and the third at “the junction of the middle third and the lateral third of the inferior orbital rim . . . for the nasolabial suspension.”

This review’s senior authors (CDM and FN) prefer the midface lift to address malar mounds and festoons and employ a similar operative technique presented next. (Important differences are noted.)

**Senior Authors’ Surgical Technique**

The lower eyelid and midface are infiltrated with 0.5% lidocaine with epinephrine solution. A subciliary incision is made through the skin and orbicularis muscle; the total length is from 10 mm lateral to the lateral canthus to within 10 to 15 mm of the medial canthus. The incision is started with the scalpel and completed by angling scissors, such that a stairstep-type incision is made through the skin and orbicularis oculi muscle, leaving behind 2 to 3 mm of skin and a 5-mm cuff of pretarsal orbicularis. Electrocautery raises a skin-muscle flap to the orbital rim and completely releases the arcus marginals with attached OML. A periosteal elevator completes total subperiosteal dissection of the midface. One author (FN) prefers to preserve the zygomatico-facial nerve during this dissection; the other author (CDM) prefers to transect the nerve completely to avoid any complaints of dysesthesia or paresthesia. Patients with this level of midface undermining routinely receive some sort of lid-anchoring procedure. The orbicularis oculi is redraped with an oblique vector, the excess is marked, and a skin flap is raised as much as needed to allow the muscle to be trimmed. The orbicularis oculi flap is secured to the temporal fascia or the confluence of the lateral orbital thickening and the temporal fascia using 4-0 PDS (Ethicon, Inc, Somerville, New Jersey). To efface the festoon effectively, the skin is undermined as extensively as needed to eliminate excess. A triangular skin excision is completed, and the skin is gently redraped with a vertical vector. With this type of extensive undermining, care should be taken to avoid overresecting the skin and to anchor the lid with canthopexy or canthoplasty to prevent lid retraction. The skin is closed with 6-0 Prolene (Ethicon, Inc) interrupted sutures lateral to the lateral canthus and in a subcuticular fashion medial to the lateral canthus. This technique addresses a broad range of conditions from mild malar mounds to severe festooning (Figures 5-8).

**Direct Excision**

Direct excision presents another treatment option of festoons in a select patient group. This technique permits direct excision of a significant amount of skin in patients with the classic description of cascading skin hammocks. Some argue that, similar to the skin-pincher technique, direct excision may avoid certain risks, including ectropion or lagophthalmos, that more commonly occur with some of the other techniques.13 Bellinvia et al21 described their success with this procedure in 55 patients using a skin-pincher technique to determine the amount of tissue excision. To avoid excessive skin excision, patients were asked to look upward during preoperative marking “to account for the progression of lid laxity that might limit the excision.” Skin alone was removed, and the incision was sutured closed. For patients requiring fat excision, this was accomplished by spreading between fibers of orbicularis oculi to retrieve fat.

As previously stated, appropriate patient selection and education is imperative with this procedure. Excision, while a good operative technique, is not ideal for patients desiring purely cosmetic improvement. Patients must be informed about location and visibility of postoperative scars. Older patients with deep wrinkles and crepe-like skin are the best candidates for this technique, as the scar will be least conspicuous in this group.22 Several steps should be taken to optimize the scar’s appearance. First, the incision should remain within the thin eyelid skin, avoiding thicker skin over the malar region. Second, the edges should be meticulously reapprorimated. Finally, patients with darker pigmentation over the lower eyelid...
should be excluded.\textsuperscript{13,21} We also advocate considering a lid-anchoring procedure with direct excision. Patients may have preexisting lid laxity that could lead to ectropion, even with this skin-only procedure. These techniques can result in successful direct excision of festoons with acceptable scarring.

Figure 5. (A, C) This 64-year-old woman presented with an aging appearance of face and eyelids, including moderate malar mounds and prominent eyes, with Hertel measurement of 18 mm. (B, D) Forty-nine months after lower blepharoplasty with fat excision and redistribution, primary spacer graft and canthopexy, upper blepharoplasty with fat redistribution, and lower face and necklift with submental platysmal plication. (E) Split frontal image comparing preoperative and postoperative views.
Figure 6. (A, C) This 54-year-old woman presented with an aging appearance of her upper and lower eyelids, including severe malar mounds, with Hertel measurement of 16 mm. (B, D) Thirteen months after lower blepharoplasty with fat excision and canthoplasty, transpalpebral midface lift, upper blepharoplasty with fat excision and internal browpexy, and endoscopic browlift.
Treatment Algorithm

Again, determining the best surgical approach for treating this morphological/aging change depends on its presenting severity. Treatment is complex, but aesthetic improvement is possible using the discussed surgical procedures. We propose an algorithm for application of these surgical techniques to the spectrum from malar edema to festoons (see Figure 9 and Table 1).

Placement of fillers alone or in combination with other procedures is becoming an increasingly important approach to facial rejuvenation. In the case of mild malar mounds, fillers for camouflage may provide a future, nonsurgical means of addressing this problem. However, the current literature lacks sufficient evidence to evaluate this approach’s effectiveness.

Complications

Much of the literature regarding this condition spectrum provides limited information on complications, revision rate, and length of follow-up. The only reported complication of the microsuction technique is skin perforation from the liposuction cannula. This complication can be avoided easily with careful surgical technique and by always orienting the cannula parallel to the skin. Although unmentioned in the literature, we caution against overaggressive suctioning, which can create contour defects. For the skin-muscle flap technique, Klatsky and Manson reported in 532 cases a 4% rate of mild ectropion not requiring surgical correction and a 3% hematoma rate with a minimum of 3 months follow-up. With the transtemporal midface lift, Hoenig et al reported 3 of 12 patients (25%) with an intraoral wound dehiscence of 3 to 5 mm, which healed by secondary intention; the median length of follow-up was 12 months. Le Louarn, in his review of 67 cases involving concentric malar lifts, reported no eyelid malposition. In their 55-patient series of direct excision, Bellinvia et al reported no instances of lid malposition or any other complication. Our technique, the transpalpebral midface lift with routine canthal anchoring for malar mounds and festoons, necessitated revision in none of our patients.

Figure 7. (A) This 63-year-old woman presented with an aging appearance of her upper and lower eyelids, including moderate festoons, with Hertel measurement of 16 mm. (B) Twelve months after lower blepharoplasty with fat excision and canthoplasty, transpalpebral midface lift, upper blepharoplasty with fat excision, and internal browpexy.
A retrospective review of 291 patients undergoing primary blepharoplasty with the full open approach of OML release and routine canthal anchoring, lid malposition rate was 5.5% (n = 16; unpublished data). All patients had a minimum follow-up period of 6 months. We have encountered no other complications or limitations to our technique beyond those typically found with lower lid blepharoplasty. Complications in reference to the above-described procedures should be reviewed.

Chemosis—conjunctival response to numerous causes of eye or eyelid inflammation—is a common complication after lower eyelid surgery. Therefore, it may be expected that greater surgical manipulation of the eyelid will result in a higher incidence of chemosis. Indeed, some studies of lower eyelid blepharoplasty with lateral canthal anchoring demonstrate a 11.5% to 12.1% incidence of chemosis. Treatment methods for chemosis vary with the severity of the occurrence. Mild instances can be treated with steroid drops or ointment, decreasing inflammation and lubricating the eye. For moderate cases, one may want to also consider patching the eye for 24 to 48 hours to completely eliminate any external exposure. Patients with billowing conjunctiva may require a conjunctivotomy to release the fluid, followed by application of antibiotic ointment. Refractory chemosis usually signals a problem with the eyelid-closing mechanism, requiring identification and surgical resolution.

Lid malposition—lid retraction or ectropion—is a common and sometimes very challenging complication following lower lid blepharoplasty. Even in routine canthal anchoring and orbicularis suspension in 757 patients, Hester et al reported a 6% (n = 45) revision rate for moderate lid malposition and a 3% (n = 23) rate of major revision for recalcitrant lid malposition. With a similar surgical approach, Codner et al reported a 3.5% (n = 264) rate of lid malposition requiring revision and a 6.1% rate of mild lid retraction that improved with massage. The causes for eyelid malposition may be senile, cicatricial, or paralytic, the latter 2 being the most pertinent here. In terms of paralytic ectropion, previous studies have demonstrated that the inner canthal orbicularis is responsible for blinking and maintaining eyelid position and tone. The
inner canthal portion of the orbicularis is innervated by the facial nerve’s buccal branch after it “passes through a triangular window formed by the inferior edge of the orbicularis muscle, the zygomaticus minor muscle, and the alaeque nasi muscle and then . . . over (or under) the levator labii muscle” to reach its target.27 As long as inner canthal orbicularis and buccal branches are not injured, paralytic causes for lid malposition can be avoided.

Overly aggressive skin and muscle removal can contribute to cicatricial causes for postblepharoplasty lower eyelid malposition. Scarring in the middle and posterior lamella caused during dissection or with improper tissue suspension is an additional culprit. Unbalanced biomechanics caused by failure to surgically anchor the eyelid can also be an important factor. Depending on the degree of surgical intervention and baseline preoperative laxity, the unanchored eyelid may not be able to withstand postoperative cicatrical forces. In the senior authors’ (CDM and FN) midface lift technique, canthal anchoring (canthopexy or canthoplasty) is routine—the key factor in reducing risk of lid malposition. Failure of canthal anchoring sutures may cause early lid malposition and must be addressed surgically with repeat canthopexy. In secondary or recalcitrant cases of lid retraction, where the surrounding tissues are inadequate to hold canthal anchoring, suture drill holes are placed instead. In some cases, lid malposition occurs because of failure to recognize the need for horizontal lid shortening during the initial procedure. For patients who have greater than a 6-mm lid distraction, a canthotomy and canthopexy with horizontal lid shortening is necessary at the time of revision. Scarring in the anterior lamella requires remobilization, advancement, and redraping of skin and muscle as needed. Posterol lamella scarring may require release of the lower lid retractors and placement of a spacer graft.

Recurrence of malar mounds and festoons is an important concern. In his 1993 article, Furnas9 raised this issue, stating “malar mounds have a reputation for persistence despite surgical efforts at correction[;] residual malar mounds have been our most common complication,” usually recurring after 12 months. With our technique, we have not yet encountered any recurrences. Figure 10 presents additional long-term follow-up with a postoperative result at 46 months. As previously discussed, the literature contains limited long-term follow-up for this condition spectrum; as such, assessing long-term stability of the postoperative results is difficult and presents a limitation to our review.

Periorbital lymphatics play a role in the development of malar edema, malar mounds, and festoons, but many details are still missing. Happily, our understanding of periorbital lymphatics continues to evolve: recent articles have identified periorbital drainage patterns differing from the classic understanding of the eyelid’s upper and lateral portions draining to the preauricular basin, and the lower and medial ones to the submandibular basin. Using lymphoscintigraphy, research now suggests that preauricular nodes

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**Figure 9.** Algorithmic approach to the treatment of malar edema, malar mounds, and festoons. OML, orbitomalar ligament.

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**Table 1.** Treatment of Malar Edema, Malar Mounds, and Festoons

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The number of pluses indicates the degree of effectiveness for each procedure. OML, orbitomalar ligament.
are sentinel lymph nodes. However, questions remain—for instance, determining the cause of edema and whether fluid accumulates exclusively deep or superficial to orbicularis oculi muscle or both. Eyelid lymphatics “can be divided into a superficial (pretarsal) plexus and a deep (posttarsal) plexus [that] although previously . . . thought to be freely interconnected . . . may be separate.” A histological study of dermatochalasis in 15 blepharoplasty specimens, compared with 10 controls, demonstrated “an increased number of lymphatic vessels, enlarged lymphatics, a larger collagen stromal bed diameter, and widely spaced collagen fibers strongly suggest[ive of]
lymphedema” in the blepharoplasty group.31 Nagi et al31 also describe “a markedly decreased density of elastic fibers in patients with dermatochalasis, indicating the loss of an elastic network that may disrupt lymphatic drainage, resulting in lymphedema.” Although their study provides a few answers as to causes of malar edema and mounds and festoons, it only assesses the superficial plexus.

CONCLUSIONS

The spectrum from malar edema to festoons represents a challenge for plastic surgeons who strive for periorbital rejuvenation. Thoroughly understanding the anatomical basis of this condition is essential to learning how to address it surgically. Thanks to the contributions of Furnas and other authors, we now recognize attenuation of the orbicularis oculi muscle as an important factor in festoon development. Later identification of OML ligament and malar septum, as well as improved understanding of their contribution to these conditions, advanced our comprehension of their anatomical and physiological basis. In this article, we described the existing literature on treatment of malar bags, malar edema, and festoons, and we provided a treatment algorithm based on the senior authors’ clinical experience. As our understanding improves, we can equally expect continued refinements in surgical treatment.

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