Commentary

Commentary on: New Insights Into Physical Findings Associated With Postblepharoplasty Lower Eyelid Retraction

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An analysis of the clinical findings in patients referred to a single surgeon with the problem of lower lid retraction following blepharoplasty is welcomed information. The reported series includes 46 patients over almost a 2-year recent period and should be of keen interest to surgeons who perform lower lid blepharoplasty.[1] The series reported includes almost 2 patients per month, which is a good sampling of patients with this problem.

Commonly recognized problems were present in the series: skin shortage (79%), unaddressed lower lid laxity (62%), and middle lamellae scarring (17%), as well as some not so commonly recognized. A preexisting negative vector (in many cases unrecognized global globe prominence) (65%) is a known pitfall when performing lower lid blepharoplasty. Not so common factors recognized by the author were inferior orbit/lower lid volume deficits (70%) and orbicularis weakness (87%). As indicated in the article, as from my own personal experience, many of these occur in combination, which makes surgical efforts at repair a difficult situation.

The author includes descriptions of his methods of quantitation or certification of each problem, which is a worthwhile checklist for any surgeon facing restoration of these patients. They include the following:

- Middle lamellar scarring—"a forced traction test," resistance to upward pulling on the lower lid
- Anterior lamellae shortage—increasing retraction on looking up and/or mouth opening
- Orbit/lower lid volume deficiency—subjective measurement
- Negative vector—observation that the cornea projects more anteriorly than the midface
- Lower lid laxity—"snapback test," distraction test
- Orbicularis weakness—examiner's ability to pry the patient's eyelid open during forceful eyelid closure

A personal observation in these patients with a "negative vector" is that many times, it is extremely difficult to reposition the lower lid with "posterior lamellar stenting with graft" alone in that these patients also require augmentation of their malar area with either autogenous (dermis fat) or alloplastic material.

A "preliminary review" of the data showed only a 40% satisfaction rate following reparative surgery in the hands of the author (a skilled surgeon), despite procedures such as a combination of midface lifting, lateral cancel suspension, posterior lamellar stenting with spacer graft, and presumably other "touchup" procedures. The author is simply relating what is known to most surgeons who inherit problem patients following blepharoplasty and have been faced with their repair. At this stage, many of these patients are "unreconstructable" with regard to expectations, anatomic restoration, or both. I would commiserate and simply mention that as a retired oculoplastic surgeon, I personally have felt the disappointment of this conundrum.

I take issue with the concept of intrinsic orbicularis weakness or loss of strength cited in the majority of these patients (87%), as well as disallowing the method of "quantitation" used by the author. Prying the lids open in a patient who is voluntarily squeezing his or her eyelids closed, in my opinion, is hardly adequate as a measurement. It is a doubly subjective maneuver of the patient and the examiner. The author also indicates that the maneuver used in this series was reduced from a graded scale of 1 to 4 quantitation to "binary," in which prying open the eyelids was either "weak or not." It should be pointed out that other factors produce the variation in force needed to open squeezing eyelids with prying fingers. Loss of mechanical advantage of the orbicularis for eyelid closure from canthal dehiscence is one.

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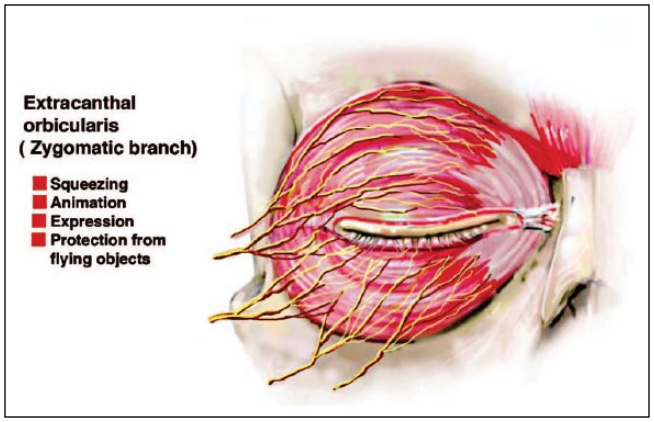

Figure 1. Recruitment of the extra canthal orbicularis from branches of the zygomatic branch of the facial nerve. This portion of the orbicularis is recruited in the active squinting or squeezing of the eyelids. From McCord and Miotto.[3] Reprinted with permission from Sage Publications.

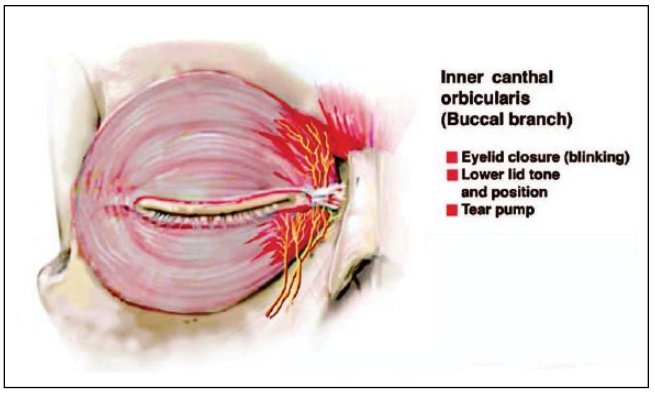

Figure 2. Recruitment of the inner canthal orbicularis from buccal branches of the facial nerve. This portion of the orbicularis is recruited in the act of blinking and provides tone to the lower lid. From McCord and Miotto.[3] Reprinted with permission from Sage Publications.

The author mentions that "open canthal anchoring" was needed in many patients, and it is well known that loss of canthal integrity of the upper/lower lid produces weakened eyelid closure. This naturally requires less force in prying the eyelids open. On the basis that blepharoplasty surgery has produced orbicularis weakness, the author recommends "minimizing orbicularis trauma" during surgery (presumably incisional, excisional, or manipulative) to avoid this problem. Unfortunately, the orbicularis is commonly redundant, and its manipulation is required for the best results in many lower lid and midface procedures (ie, malar festoon correction).

Further explanation: The orbicularis muscle is positioned concentrically around the eye and requires a counterfixation and pull at the lateral canthus to convert the sphincter-like action of orbicularis contraction to a vertical vector in the central portion of the eyelids, which forces the upper and lower lids together (see Figures 1 and 2). This concentric-like force, whether it be produced by the canthal orbicularis (recruited by the buccal branch of the facial nerve to cause a "blink") or the extra canthal orbicularis (recruited by the zygomatic branch of the facial nerve to produce a "squeeze"), requires firm lateral canthal stability and fixation to cause a vertical vector of closure to occur (see Figure 3). It is very common in postblepharoplasty patients who have weakness

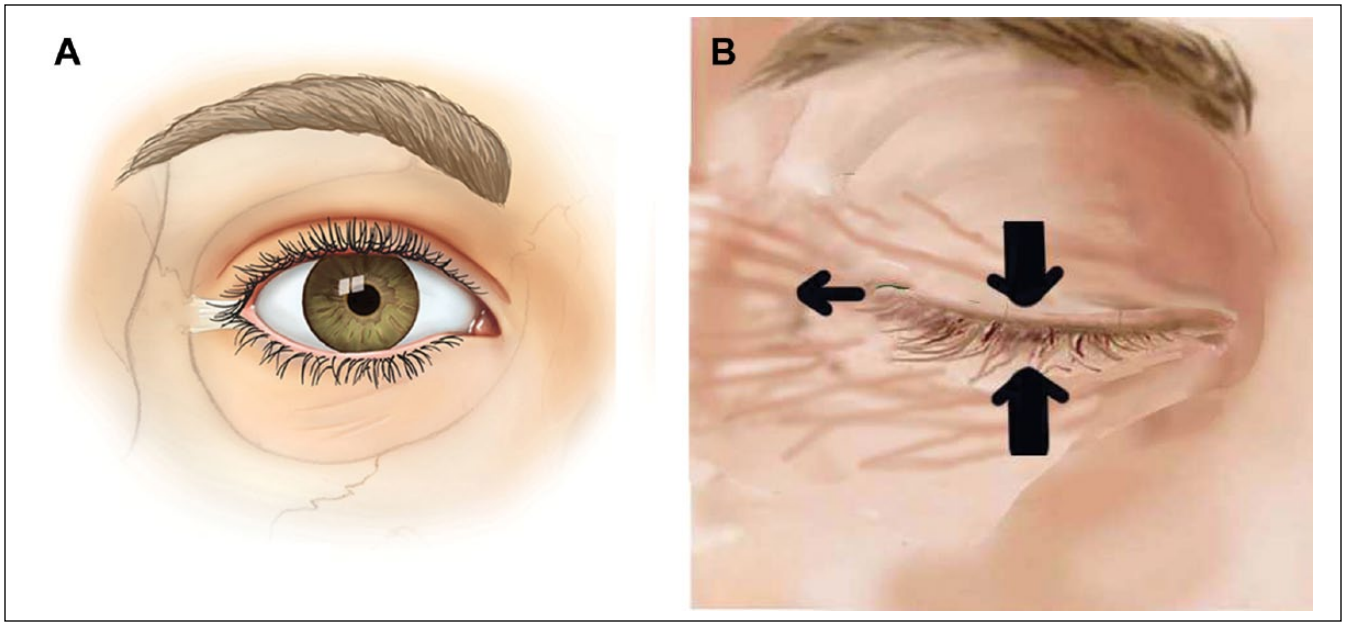

Figure 3. (A) Normal eyelids open with secure lateral canthal attachment. (B) Normal eyelids closed with squeezing after recruitment of the inner and extra canthal orbicularis via the buccal and zygomatic branch of the facial nerve with a strong vector of closure with secure lateral canthal attachment. The relative thickness of the arrows depicts amount of force. From McCord and Miotto.[3] Reprinted with permission from Sage Publications.

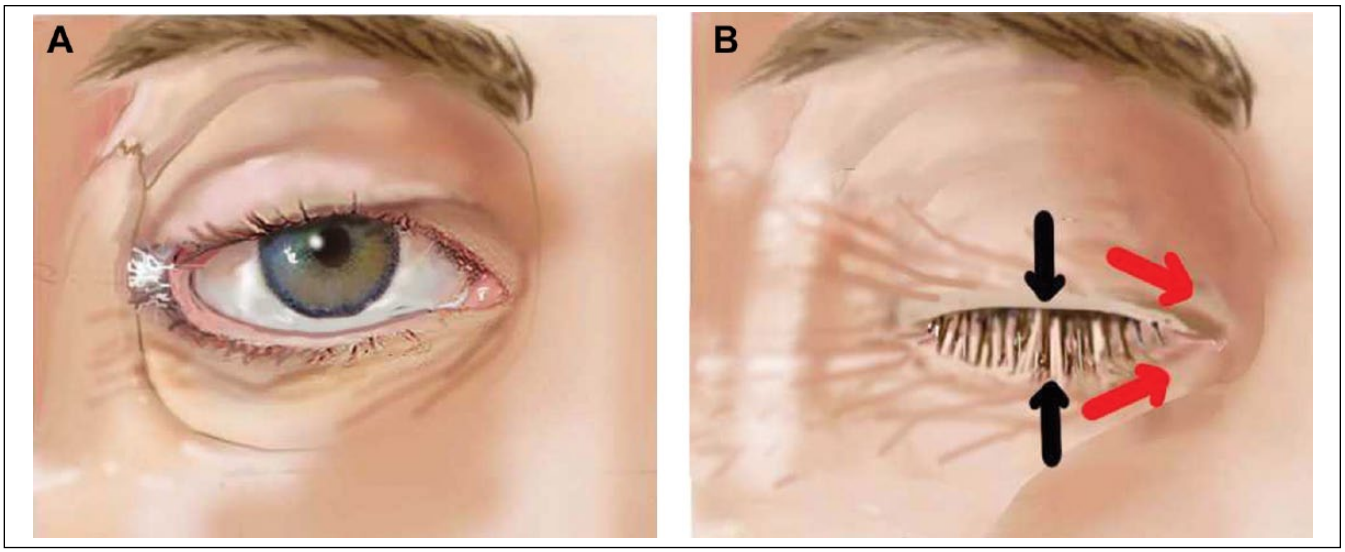

Figure 4. (A) Lower lid malposition with scleral show and lateral canthal dehiscence. (B) With squeezing of the eyelids. The strength of vertical vector closure has weakened because of the loss of mechanical advantage caused by lateral canthal weakness. The relative thickness of the arrows depicts amount of force. From McCord and Miotto.[3] Reprinted with permission from Sage Publications.

of eyelid closure to be misinterpreted as having intrinsic weakness of the orbicularis, whereas it is on the basis of faulty lateral canthal attachments (see Figure 4). The resulting "fishmouthing" movement on eyelid closure in these patients is one of the most overlooked complications in postblepharoplasty patients and was the subject of 2 reports in this journal recently.[2,3] It is also possible that other anatomic mechanical anomalies in the eyelid and periorbital area caused by scar tissue following blepharoplasty can play a role in the difficulty of prying the eyelids open.

I am grateful that the author did not mention the word *denervation* with regard to his observed "orbicularis weakness" in these patients since its existence has been disproven with very precise studies.[4] In the absence of exceptional things such as unlikely postblepharoplasty intrinsic orbicularis myopathy causing the weakness, the observed eyelid closure or "prying open" anomaly can be explained by the loss of mechanical advantage from canthal deficiency or other mechanical factors.

Despite my above concern, this article is extremely well received by me because of its attempt to make sense of the reasons for postblepharoplasty problems, which will benefit the surgeons who perform the surgery by forewarning them. I happily listen to the frustrations of the surgeons who have to deal with these patients since I have been in that boat.

Disclosures

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