Transconjunctival Septal Suture Repair for Lower Lid Blepharoplasty

Sir:

Lower lid blepharoplasty has evolved drastically over the last few decades. As rightly mentioned by Dr. Sadove,¹ many complications have been described following external subciliary skin incision. The need for safer procedures, stressing the importance of orbicularis muscle and orbital fat preservation, has long been recognized. The technique originally described² and now revived by Sadove definitely preserves both structures; however, is this what is really required to correct the stigma of lower lid aging?

Orbital septum tightening would correct the bulging lower eyelid, but it does not rejuvenate the lower lid. Facial aging is a summation of both hard- and soft-tissue changes.³ Optimal rejuvenation of the lower eyelid should correct not only prolapse of orbital fat but also descent of the cheek tissues, accentuation of the orbital rim, and the tear trough groove.⁴ To obtain a truly youthful eyelid-cheek complex, simple tightening of the orbital septum to place the protruding fat back into position cannot be enough. It is essential to restore midface volumes, reposition shifting tissues, and create a narrower, shallower orbit with a shorter lower lid.⁵ In fact, lower lid blepharoplasty should be considered a complex reconstructive procedure, part of an overall holistic approach to facial surgery aimed at restoring youthful volumes by redistributing and shifting local tissues, and whenever indicated by the addition of soft tissues or other substitutes.

The described technique does not address all the changes of aging and definitely does not result in optimal rejuvenation. As rightly mentioned by Dr. Sadove, it is not a universal technique.⁶ It is not an acceptable method when cheek sagging with elongation of the lower lid vertical length is present. The patient shown in Sadove’s Figure 13 clearly illustrates this point. Shortening lower lid vertical length by elevating the lid-cheek skin junction and camouflaging the inferior orbital rim is essential.⁷ It seems that risking muscle weakness of a small pretarsal strip, if any, is a small price to pay to achieve optimal lower lid and periorbital rejuvenation, provided lower lid sagging, rounding of the lateral corners of the palpebral aperture, and widening of the palpebral aperture are avoided by proper orbicularis sling suspension, canthoplasty, and lower lid tightening. It is not true, also as claimed, that preservation of the original palpebral aperture is desirable. On the contrary, elevation of the lower lid margin with a simultaneous decrease in the palpebral aperture may be highly warranted in most older patients.

Dr. Sadove is to be commended on his meticulous presentation and his honesty in reporting his complications. We are surprised, though, that he did not encounter more lower lid tethering, a dreadful complication that has detracted many from orbital septum tightening. This is definitely an indication of his excellent surgical skills. As a general principle and irrespective of the surgical technique, local assessment, careful preoperative planning, and conservative tissue resections can help minimize complications and optimize results.⁸ Dr. Sadove’s technique is certainly a valuable addition to our surgical armamentarium.

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REFERENCES


**Reply**

**Sir:**

I thank Drs. Atiyeh and Hayek for their kind comments. They correctly express surprise with the take-home point: septal suture can be performed with preservation of lid height and no changes in aperture shape. Mendelsohn and Muhlbauer showed us the same in their experience, via the subciliary approach.

Why weaken the muscle if this is not necessary? Preservation of orbicularis muscle integrity/inervation reduces risks in the treatment of the lower eyelid bulge.

Tightening the septum clearly rejuvenates the lower eyelid. In some patients, it is all that is required. In other patients, much more is needed. Transconjunctival septal suture is clearly not a panacea for all aging manifestations of the eyelid and/or face.

We all have seen too many patients in our offices with lax lower lids after undergoing some unknown subciliary approach many years previously. Even without a blepharoplasty, aging patients may have a loss of aperture shape. The corner of the eye becomes more rounded and open in a vertical dimension. It behooves us to offer procedures to our patients that do not create or hasten this deformity.

Drs. Atiyeh and Hayek avoid or enhance aperture change with their orbicularis sling and canthoplasty in the treatment of bulging fat. These interventions can be obviated with transconjunctival septal suture. If we do not break it, we do not have to fix it.

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**Blepharoplasty, Laser In Situ Keratomileusis, and the Corneal Reflex Arc**

**Sir:**

We applaud Korn et al.1 for their article in the July 2007 issue of the *Journal*. Prior studies in the literature have reported dry eye syndrome as a complication of either blepharoplasty or laser in situ keratomileusis (LASIK). The current study by Korn et al. provides insight into the combined effect of blepharoplasty and LASIK on the incidence of dry eye.

In their retrospective study, the authors examined six patients who underwent both blepharoplasty and LASIK. Of particular interest are the two patients who underwent monocular LASIK and subsequent bilateral upper and lower blepharoplasty. After blepharoplasty, these patients manifested the symptoms of dry eye syndrome. However, the symptoms were confined only to the LASIK-treated eye. The authors hypothesize that the dry eye symptoms (in the LASIK eye) were the result of a blunted corneal reflex arc on that side (secondary to LASIK)1 and that the lack of dry eye in the opposite eye (with blepharoplasty alone) was due to the preservation of the blink response on that side. To examine the validity of this hypothesis, we wish to elaborate on the neurophysiology of the corneal reflex.

It is known that blunting of the corneal reflex arc does in fact occur when raising a corneal flap and transecting the afferent nerves in the cornea during a LASIK procedure.2–4 However, the authors should note that the corneal reflex arc comprises an afferent pathway from each eye and a consensual efferent response that is bilateral.5 In the case of the two patients treated with LASIK in only one eye, the intact afferent arc in the opposite eye (with the blepharoplasty only) would preserve the blink response bilaterally, thus preventing a possible dry eye in the LASIK-treated eye. Taken together, the eye with blepharoplasty alone will have an uninterrupted afferent reflex arc and both eyes will have fully intact efferent motor nerves, resulting in a preserved blink response. Barring disruption of lid mechanics, the impulse for normal bilateral closure remains unaffected; thus, sweeping and lubrication of the corneal surfaces should theoretically be unchanged. In this regard, we ask the authors to reconsider their hypothesis in these patients.

We appreciate the reporting of an association between LASIK and blepharoplasty in inducing dry eye syndrome. Further studies should aim to examine the incidence of dry eye syndrome in each eye after monocular LASIK and subsequent bilateral upper and lower blepharoplasty in a larger patient population. If dry eye symptoms consistently occur only in the LASIK-treated eye, causes other than the corneal reflex arc will need to be explored and elucidated.

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**DISCLOSURE**

Neither author has any financial interest in any of the products, drugs, or devices described in this communication.
REFERENCES


Reply

Sir:

We thank Drs. Afrooz and Gorantla for their interest in our recent article describing dry eye syndrome in patients who have undergone both blepharoplasty and laser in situ keratomileusis (LASIK). Under discussion is the possible mechanism for the exacerbation of dry eye symptoms in patients who had monocular LASIK and subsequent blepharoplasty. We hypothesized that the dry eye symptoms occurred in the LASIK-treated eye secondary to a blunted blink reflex. Afrooz and Gorantla raise the point that the efferent response of the corneal blink reflex is bilateral and not unilateral, and hence this cannot explain the dry eye symptoms in our study patients. We are reassured that we are all wholeheartedly in agreement with this well-established neuroanatomical pathway. However, what is lost in the understanding of this pathway is the source of the afferent input. Recall that in the case of the corneal blink reflex, afferent signals are carried by the nasociliary branch of the ophthalmic nerve (V1). The system requires only stimulation of either the right or left corneal nerves to generate a bilateral blink reflex. However, if there is now disruption of the afferent signal in one eye (i.e., by monocular LASIK), the blink reflex only responds to afferent stimuli from the nondisrupted cornea. Hence, in the monocular eye treated with LASIK, there is blunted corneal sensitivity, and hence the blink reflex is impaired when the LASIK eye is stimulated. The fellow eye, untreated by LASIK, still contains intact corneal fibers and can provide a bilateral blink reflex in response to noxious stimuli.

Afrooz and Gorantla state, “In the case of the two patients treated with LASIK in only one eye, the intact afferent arc in the opposite eye (with the blepharoplasty only) would preserve the blink response bilaterally, thus preventing a possible dry eye in the LASIK-treated eye.” The assumption that Afrooz and Gorantla make is that both corneas receive equal afferent input and that the non-LASIK eye will respond with the bilateral efferent response to protect the LASIK-treated eye. This is clearly not the case. First, the monocular eye treated with LASIK may have preexistent keratitis, which is not clinically manifest. However, after bilateral blepharoplasty and subsequent exposure keratopathy, the previously stable keratitis now worsens. But there is no significant afferent input to stimulate the blink reflex from the LASIK-treated eye. The fellow eye does not receive the same afferent signal from the LASIK-treated eye, and hence there is no added stimulus to blink from the LASIK-treated eye. In this manner, the fellow eye (untreated with LASIK) is in essence “blind” to afferent stimuli in the LASIK-treated eye. In addition, the LASIK-treated eye does not respond as well to inadvertent foreign body stimuli that may irritate the ocular surface, further worsening the keratopathy.

We appreciate the discussion and awareness raised by Afrooz and Gorantla of our article. Indeed, we agree that further studies are needed to explain this new syndrome.

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DISCLOSURE

None of the authors has a financial interest in any of the drugs, devices or products mentioned in this communication.

REFERENCE


An Analysis of the Olivari Decompression Technique

Sir:

The recent publication by Richter et al. in this Journal updates the experience of their group with the Olivari technique of decompression in Graves ophthalmopathy. While a superficial reading of their article seems to indicate that this publication is a continuation of all their previous works, a closer perusal reveals some important questions that the authors should answer.

The authors base their indications for fat orbital decompression on the modified Werner classification. Of their cases, 94 percent were based primarily on grade III (protrusion), with some (20 percent) also having grade IV
and V involvement. If reduction of fat protrusion ameliorates extraocular muscle or corneal factors, it is by virtue of the fat realignment. Were the problem purely an extraocular muscle or a corneal problem, other types of surgery (e.g., extraocular muscle resections and tarsorrhaphy) would be in order.

The surgical procedure is an upper and lower lid blepharoplasty with excision of extraconal and intraconal fat. This is a significant change from the previous surgical descriptions of their technique, where no intracanal excision of fat is described. In one of their publications they state, “The intracanal fat is luxated,” indicating change of position, not excision. It is even described in their textbook contribution as an “en bloc” dissection in each quadrant. Is this an extraconal and intracanal bloc? In another article, they describe fat removal from the “retrobulbar fat including that of the apical area.” As mentioned below, invasive apical orbital surgery is fraught with high morbidity and possible vision loss such that orbitologists have proscribed apical surgery.

I have followed Olivari’s work for many years. In my experience of orbital decompression surgery (85 to 100 cases), I have performed the Olivari technique (in about 20 cases) and found that when intracanal fat is excised, there is a 40 to 50 percent morbidity rate, with the main problem being postoperative motility and pupillary defects. Some of these untoward effects have been permanent. I found that the upper limit for excising intracanal fat is a minimal amount (0.25 cc) in any quadrant (for a total of 1 cc) before postoperative morbidity is noted. I am aware of optic neuropathy and anterior segment ischemic syndromes with excision of significant (>2 cc) intracanal fat. Presumably this is due to the fine arborization of blood vessels and nerve fibers within the intracanal fat. So it behooves the authors to inform us of how much intracanal fat they excised and how this change affected their results.

In distinction to intracanal fat, I have never had any such problems with the Olivari technique when only extraconal fat was excised. The problem with taking only extraconal fat is that the amounts excised rarely exceed more than 4 cc, especially if the deep retrobulbar area fat is not excised. This goes along with a generally accepted dictum that surgeons should avoid the orbital apex, especially the deeper 15-mm area. But such anterior excisions have been inadequate.

Also, if 6 cc of extraconal fat is taken periocularly, there would be inadequate cushioning for the globe, as the authors state in their surgical procedure section. Presumably this would require the prolapsing of intracanal fat to form some of the cushion effect in addition to excision of this fat. Does this result in morbidity postoperatively?

Further, another subgroup would be those patients with grade III and VI involvement (sight loss). How many of those patients had adequate resection with extraconal as opposed to extraconal and intracanal excision? Of my own patients, at least 50 percent are in this grade category. I have many patients with relatively pure grade VI-type changes with only visual field loss and very little protrusion. What is their procedure for these patients?

Another issue they need to clarify is their separation of diplopia from strabismus cases and results. What is the meaning of having a class of diplopia cases and a class of strabismus cases? On what basis do they distinguish diplopia from strabismus? Are these individuals who have subjective complaints of diplopia with no objective motility disorder? Are the authors then stating that these cases can be alleviated by fat decompression alone?

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REFERENCES


Hypercoagulability Due to Homocystinuria in a Case of Head and Neck Reconstruction Resolved with Combined Systemic Therapy

Sir:

W e read with great interest an article recently published in Plastic and Reconstructive Surgery about a case of free flap failure in a patient with homocystinuria. The authors faced multiple arterial thrombosis during toe-to-hand transfer in their young patient. They tried thrombectomy and multiple local infusions of streptokinase and tissue plasminogen activator, but eventually they could not save the flap.

We experienced a similar case in a patient operated on for a squamous cell carcinoma of the floor of the mouth. After resection, he was reconstructed with a free anterolateral thigh musculocutaneous flap. Unfortunately, this flap failed due to venous and arterial thromboses. We tried to overcome the problem with arterial infusion of 1000 IU of streptokinase with opened veins and 4000 IU of subcutaneous heparin postoperatively, but we had no success and explained this failure as a technical error.
We then decided to perform another anterolateral thigh musculocutaneous flap from the contralateral side. At this stage, multiple arterial thromboses after every anastomosis with the facial, lingual, and superior thyroid arteries presented. Sometimes thrombosis was evident even before anastomosis. Intraoperative Doppler ultrasound of the carotid artery showed normal flow.

Due to this unknown hypercoagulable state, we started with empirical systemic therapy: 50 mg of tissue plasminogen activator intravenously, 0.1 μg/kg per minute continuous intravenous infusion of tirofiban (inhibits platelet aggregation) for 12 hours, and 8000 IU/day of continuous intravenous infusion of heparin. Arterial thrombosis resolved in about 10 minutes, and we then successfully performed vessel repair.

From the twelfth postoperative hour until the seventh postoperative day, prophylactic intravenous heparin was administered. From the seventh to the fourteenth postoperative day, 2000 IU of subcutaneous heparin was given three times a day.

The postoperative course was uneventful, and after 25 days the patient was discharged. A coagulation work-up was positive for elevated serum homocysteine, consistent with moderate homocystinuria. We agree with the authors that hypercoagulability syndromes should be included in the differential diagnosis of flap loss, and we think that when faced with this problem only an associated systemic therapy with thrombolytic, antiaggregant, and heparin can resolve the problem. Bleeding could be the main disadvantage, so the whole history, examination, and risk-benefit ratio should be taken into consideration.

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REFERENCES

Reply
Sir:

We thank Drs. Spanio di Spilimbergo et al. for their comments and for reporting their experience with free flap loss and salvage in a patient with homocystinuria. Although individual enzymatic defects in the folate pathway may be rare, when all potential causes are included, homocystinuria affects as much as 10 percent of the population. Therefore, it is likely that many more flap failures have occurred as a result of this entity but have gone unrecognized. In fact, the effects of homocystinuria on plastic surgical patients extend beyond microsurgery. For example, homocystinuria is a risk factor of deep venous thrombosis, and thus we advocate measuring homocysteine levels as part of the hypercoaguable evaluation for young, healthy patients who develop a deep venous thrombosis or pulmonary embolus. The treatment of homocystinuria is straightforward and significantly reduces the morbidity of affected individuals. In the case reported by Spanio di Spilimbergo et al., an anterolateral thigh flap was used to reconstruct a floor-of-the-mouth defect after tumor resection. As in our case, the first free flap was lost despite attempts at regional thrombolytic therapy. A second anterolateral thigh flap was attempted and salvaged using aggressive systemic anticoagulation: tissue plasminogen activator, tirofiban, and intravenous heparin. Systemic heparin was continued for 2 weeks after the procedure, and the patient was discharged uneventfully after a 25-day hospitalization. Spanio di Spilimbergo et al. should be commended for their technical expertise and salvage of the second anterolateral thigh flap. Moreover, they should be congratulated for thoroughly investigating the cause of their first flap’s failure and identifying the source of their patient’s hypercoagulability. Unfortunately, homocystinuria is likely to be diagnosed only after a failed free tissue transfer, which then stimulates a hypercoagulability work-up. In the patient with elevated homocysteine levels, our experience as well as Spanio di Spilimbergo et al.’s suggests that regional thrombolytic therapy is ineffective and aggressive multimodal systemic anticoagulation may be required for flap salvage. However, the morbidity and mortality risks associated with multimodal systemic anticoagulation in any postoperative patient, especially those with fresh anastomoses in the neck, are considerable.

Presently, it is unknown whether treatment of homocystinuria can abrogate the risks of microvascular thrombosis. We would predict that the observations of decreased stroke, thrombosis, and myocardial infarction seen in homocystinuria patients treated with vitamin B and folate would also translate into a reduced
risk of microvascular thrombosis. However, in the rare microsurgical patient with known, controlled homocystinuria, the surgeon should have a low threshold for the use of systemic anticoagulation. Currently, we do not recommend homocystinuria testing in healthy patients before a microsurgical procedure. Instead, during our preoperative screening we obtain a hypercoagulability profile in patients with a history of thrombosis at a young age, thrombosis without a predisposing cause, or resistance to anticoagulation.

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REFERENCES

Reply

Sir:

I am not surprised by the experience of Dr. Spanio di Spilimbergo and colleagues with a failed free flap. It has been my contention from day one that the majority of microsurgical failures are caused primarily by technical errors in either donor or recipient vessel regions. Most commonly, patient disease and other comorbidities are blamed. However, when the flawless technical execution of a case is followed by overt failure and thrombosis of all vessels, one must start looking for a cause. In this case, they found one. Cardiologists are very much aware of this problem, which is a common cause of coronary and cerebrovascular thrombosis. It is hoped that microsurgeons will become more cognizant.

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The Cutaneous Arteries of the Anterior Abdominal Wall: A Three-Dimensional Study

Sir:

We congratulate Tregaskiss et al. for their study utilizing computed tomography angiography in the evaluation of the abdominal wall vasculature. As mentioned in their article, computed tomography angiography has only recently been suggested for preoperative imaging before use of superficial inferior epigastric artery (SIEA) and deep inferior epigastric artery (DIEA) perforator flaps, but it has yet to be adequately evaluated for this purpose. Their article, “The Cutaneous Arteries of the Anterior Abdominal Wall: A Three-Dimensional Study,” certainly contributes to the literature on this topic. We would like to share our work on this topic, which we believe may shed more light on this rapidly advancing field of anatomical research, in light of their article.

We have undertaken a study of 10 cadaveric hemia- bdominal walls, which is currently in the submission process for this Journal, in which we performed isolated DIEA contrast injection in each case. Our injection technique and harvesting methods were done in a fashion similar to that of Tregaskiss et al., and our specimens underwent computed tomography scanning in a similar way. Our computed tomography scanner differed in that it was a 64 multidetector row scanner, which permits a greater number of slices and greater resolution of images. Our reformattting also differed, in that we created maximum intensity projection images in addition to three-dimensional volume-rendered technique images.

In general, our findings confirm the findings of Tregaskiss et al., in that computed tomography was able to effectively demonstrate the SIEA system, the cutaneous branches of the SIEA system, the musculocutaneous perforators, and the DIEA system. Of note, we were able to identify both SIEAs in 80 percent of patients, compared with 40 percent in the study by Tregaskiss et al. These data, however, do not achieve a formal evaluation of the use of computed tomography angiography. In an attempt to achieve this, we performed post–computed tomography dissection of all the musculocutaneous perforators in each specimen (total number on computed tomography angiography = 154 perforators), which enabled us to evaluate the use of computed tomography angiography for perforator mapping, as both a tool for demonstrating cadaveric anatomy and a potential tool for preoperative imaging before use of DIEA perforator flaps.

We found that the DIEA system demonstrated on computed tomography angiography, including the course of the DIEA and its branching pattern, was 100 percent concordant with dissection findings in all cases (Fig. 1). Similarly, the mapping of musculocutaneous...
perforators was highly accurate, with only eight false-positive results and six false-negative results out of 154 perforators seen on computed tomography angiography, revealing a sensitivity of 96 percent and a specificity of 95 percent for the mapping of musculocutaneous perforators of the DIEA (Fig. 2).

Our study demonstrates that computed tomography angiography can be used with high accuracy in anatomical studies such as that of Tregaskiss et al., and suggests that this may be an ongoing modality for imaging vasculature in both anatomical studies and preoperative imaging. This type of anatomic cadaveric study does not necessarily reflect the accuracy of computed tomography angiography in the clinical setting, where it is used preoperatively to localize DIEA perforators before DIEA perforator flap surgery. Further
work in this area is required to establish the role and accuracy of computed tomography angiography in DIEA perforator flap planning.

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DISCLOSURE

The authors declare that there is no source of financial or other support or any financial or professional relationships that may pose a competing interest.

REFERENCES


Reply

Sir:

I thank Rozen et al. for providing readers with an insight into their experience with the use of computed tomography angiography for cadaveric anatomic research. As was correctly pointed out, my article (Plast. Reconstr. Surg. 120: 442, 2007) did not seek to formally evaluate the use of computed tomography angiography in this setting. My group did, however, undertake extensive preliminary work with this modality to establish some of its limitations and to determine the accuracy of software measurement tools that can be used for perforator mapping in this setting. With our particular configuration of equipment and settings, my coauthors and I established that computed tomography angiography does not consistently demonstrate vessels less than 0.4 mm in diameter on processed images. It would be interesting to know whether the false-negative results encountered by Rozen et al. were similarly the result of difficulties in trying to image these very small perforators. Despite this minor limitation, I would agree with Rozen et al. that concordance between computed tomography angiography and dissection findings is extremely high.

The use of computed tomography angiography in a clinical setting for preoperative deep inferior epigastric perforator flap planning has previously been described and is clearly of great potential. My only reservation concerns the significant radiation dose incurred by the patient when an abdominal computed tomography scan is performed. This is an issue that needs more robust discussion in the future, and I look forward to the forthcoming articles by Rozen et al., which I hope take the opportunity to do so.

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REFERENCES

Postoperative Radiation Therapy for Keloid

Sir:

We would like to make critical comments on an article by van de Kar and colleagues entitled “The Results of Surgical Excision and Adjuvant Irradiation for Therapy-Resistant Keloids” (Plast. Reconstr. Surg. 119: 2248, 2007).

First, they used “Gy” as the scale of radiation absorption dose in the abstract, body, and Table 4 of their article. This is a clear mistake. They must have meant “cGy” (centigray). If they meant 1200 cGy, this means 12 Gy. Second, they indicated that “superficial 250-kV electron beam irradiation was used.” If 250 kV were correct, an electron beam would be impossible. They must have used x-rays. From these basic errors, we consider that no competent radiation oncologist participated in the radiation therapies. Moreover, it is doubtful that appropriate irradiation was performed on the appropriate area.

Third, they cited our article1 and commented that “some authors did not even report their minimum follow-up period.” However, in the article they cited, we indicated that “only cases that were followed for more than 18 months were selected for this study.” In fact, the median follow-up period was 24 months (range, 18 to 128 months) in our article.1

Fourth, they claimed that “some authors did not describe whether or not they performed histologic tissue analysis.” However, we clearly stated that “pathological discrimination of hypertrophic scars from keloids is difficult except for typical keloids.”1 In our experience, the history of the keloid and the clinical findings are much more important factors when it comes to distinguishing keloids from hypertrophic scars. If they insist on the importance of a histological examination, they must cite some articles2,3 that describe the pathological differences between keloid and hypertrophic scars.

Fifth, 18 of 21 patients (85.7 percent) in their study were of the black race. It is considered that keloid prevalence is five to 15 times higher in African-Americans than in Caucasians.1 Logically, therefore, their conclusions should be limited to people of the black race, admitting that appropriate radiation therapy was administered. However, they did not even describe the differences in prevalence or recurrence rates by race. Moreover, the number of patients by keloid site was too small. Recurrence rate by keloid site is a very useful indicator, as we have indicated previously.1 In our facility, keloids of Asian people are treated with dose protocols that are customized for each site: (1) 20 Gy in four fractions over 4 days for the anterior chest wall, scapular region, and supraventricular region, (2) 10 Gy in two fractions over 2 days for the earlobes, and (3) 15 Gy in three fractions over 3 days for other sites.

In conclusion, they should reconsider the cause of the abnormally high recurrence rates in their results. In addition, they should review and discuss whether or not their prescription and delineation of the target volume were appropriate. We think they should cast an eye on our facility, where we have tried to calculate the absorbed dose, even in irregular and complicated irradiation fields, on a case-by-case basis. Moreover, we have used a self-management program for postoperative patient care.

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REFERENCES


The Butterfly Design as an Alternative to the “Double-A” Bilateral Flaps for the Treatment of Large Sacral Defects

Sir:

We enjoyed reading the article entitled “A New Technique of ‘Double-A’ Bilateral Flaps Based on Perforators for the Treatment of Sacral Defects,” by Prado et al.1 The double-A bilateral flap is presented as a combination of bilateral perforator fasciocutaneous and myocutaneous flaps. Segmental cutaneous innervation can be preserved, and protective sensibility is likely to be provided. The authors write that the double-A flap is “the ideal solution for neurologically intact patients.” The flap was used successfully to treat large sacral ulcers in 50 patients. A 90 percent follow-up at 1.5 years showed no recurrence. It appears that the authors have come up with a very reliable method for treating large sacral ulcers.

They are to be congratulated for having managed to add a new and reliable surgical procedure to the treatment of large sacral defects. There is no doubt that treatment of nonambulatory as well as ambulatory patients with large sacral defects is a challenge to the reconstructive surgeon. In 1956, Conway and Griffith...
reported some fundamental principles for the management of pressure sores. The flap should have a reliable circulation. Scars should not be placed over the site of the original ulcer, and the scar should not be subjected to repeated trauma. The flap should be made as large as possible, so that if recurrences happen, the same flap can be used again without interference from the scar. We believe that these principles should still be applied. One may add that, whenever possible, protective sensibility should be provided as well.

Double-A bilateral flaps are large flaps with reliable circulation and may provide protective sensibility in the reconstructed area. As mentioned by the authors, one of the drawbacks of this technique is that the scars are positioned over bony prominences, the ischial tuberosities. The ischial tuberosity is a site of predilection for pressure sores. In ambulatory as well as nonambulatory patients, one would be reluctant to place scars in this area. We also agree with the authors that using the double-A flap interferes with future use of gluteal flaps in cases of recurrence.

Even though the double-A flap appears to be a good option for the treatment of large sacral defects, we would like to draw the authors’ attention toward an alternative, the butterfly design. This design is based on the use of two lumbar artery perforator flaps placed in the configuration of a butterfly (Fig. 1). The cutaneous nerves emerging together with the perforating arteries can be included in the flap. The butterfly design provides a large volume, may provide protective sensibility in the reconstructed area, and causes minimal donor-site morbidity. These lumbar artery perforator flaps are harvested from the “love handle” areas, no scars are made over the ischial tuberosities, and the gluteal area is still available as a donor site for future reconstructions. Unlike the double-A flap, which is composed of myocutaneous as well as fasciocutaneous flaps, the butterfly design is based solely on perforator flaps. Recent research has indicated that the capacity to heal wounds is similar for myocutaneous and perforator flaps.

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Reply
SIR:
I truly appreciate Weum and de Weerd’s communication. It is interesting that the authors add to Conway and Griffith’s 1956 principles for the management of
pressure sores, that “whenever possible, protective sensibility should be provided as well”; this statement should be attributed to Kroll and Rosenfield for their article “perforator flap sensibility concept.”

The butterfly design based on two lumbar perforator arteries was published in 2002 and is unique. The authors should then have at least a 5-year follow-up, a number of cases, and a rate of complications that we should be aware of before attempting this surgery, as I believe that this design should be used in selected cases (also shared with the double-A flap concept) with advance knowledge of the failure rates of a single perforator living flap.

The blood supply reliability of the double-A flaps is based on evidence, as each gluteal area originating from intact superior and inferior gluteal arteries will have 20 to 25 perforators for each side, as described by Koshima et al.

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Autologous Gluteal Augmentation after Massive Weight Loss

Sir:

W

e read with interest the article by Amy S. Colwell and Loren J. Borud entitled “Autologous Gluteal Augmentation after Massive Weight Loss: Aesthetic Analysis and Role of the Superior Gluteal Artery Perforator Flap” (Plast. Reconstr. Surg. 119: 345, 2007). The authors describe their technique of gluteal autologous augmentation with “rotation of the flap inferomedially,” and compare their flap to Pascal and Le Louarn’s island flap (Fig. 1) as they write, “tissue flaps were designed within the lower body lift incisions but not mobilized; rather, the inferior gluteal tissue is mobilized and brought over the stationary flaps.”

We want to point out that in our article we wrote, “This requires undermining a third of the external flap to turn it downward and inward,” and “as the flap consists of very mobile tissues . . . often it is possible for the flap to extend to the groove below the buttocks.” The figure associated with this description, similar to Figure 1, demonstrates with an arrow the inferomedial rotation of the flap. Consequently, we never described a “stationary” flap but its inferomedial mobilization.

Our flap is a superior gluteal artery perforator flap. Fujino was the first to introduce gluteal tissues as a free flap, thanks to gluteal artery perforators, for breast reconstruction, 20 years ago. Colwell and Borud state, “the locations of the two key superior gluteal perforators, at approximately 7 and 9 cm from the midline, were reliable and reproducible” to specifically nourish their flap. Therefore, they write, “the flaps were dissected laterally to medially starting at the midaxillary line until the lateral perforator was encountered approximately 9 cm from the midline.” However, converging anatomical studies demonstrate that, statistically, perforators are located at a middle distance between the posterior superior iliac spine and the greater trochanter (Fig. 2), which is more lateral (10 to 12 cm) than what the authors describe and could explain their case of unilateral major fat necrosis in 18 patients. Since 1999, we have operated on 225 patients using this island flap with no major fat necrosis, because only the lateral third of the flap is released and not the lateral half, to ensure the preservation of most of the perforators.

It is well known that Kankaya et al. have written that the superior gluteal zone combines 48.5 percent of perforators, whereas the central gluteal zone is the most poorly vascularized region. Nevertheless, Sozer et al. affirmed, “The base of the flap should originate more inferiorly than the surface,” which is obviously not
Concerning the extension of the gluteal pocket, Colwell and Borud are a bit confusing because they write, “the pocket . . . extending down to the inferior gluteal crease” and “[a] gluteal pocket . . . extending to within 5 cm of the inferior gluteal crease” and “[t]he gluteal pocket should extend only over the medial half of the buttock.”

The precise answer, determined after one case of buttock numbness, is that we need to stop the downward dissection above the inferior border of the gluteus maximus muscle, because at the inferior border of the muscle emerge two sensitive nerves, the clunium medii and the cutaneous femoris posterior, that innervate the buttock skin. To avoid definitive buttock numbness, dissection has to stop above the inferior border of the gluteus maximus muscle, and therefore well above the inferior gluteal crease in obese patients.

Finally, the four drains, removed by the authors after 15 days, can be eliminated in that type of surgery, thanks to well-localized traction sutures closing all the dead spaces. Patient mobilization and scar nursing become easier. Also, use of these traction sutures in the trochanteric area would have helped the authors to avoid the persistent saddlebag deformity shown in Figures 4, 6, and 7. We named this lateral traction “the high lateral tension body lift.”

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Reply
Sir:

We thank Drs. Le Louarn and Pascal for their comments regarding our article, “Autologous Gluteal Augmentation after Massive Weight Loss: Aesthetic Analysis and Role of the Superior Gluteal Artery Perforator...”

Fig. 2. (Above) Perforator locations. The black line is drawn from the posterior superior iliac spine to the greater trochanter. (Reprinted from Nojima, K., Brown, S. A., Acikel, C., et al. Defining vascular supply and territory of thinned perforator flaps: Part II. Superior gluteal artery perforator flap. Plast. Reconstr. Surg. 118: 1338, 2006.) (Below) The perforator location is drawn too medially (two red spots), and lateral dissection (oblique blue lines) goes too far medially. Flap vascularization can be damaged. The same black line is drawn from the posterior superior iliac spine to the greater trochanter. (Reprinted from Colwell, A. S., and Borud, L. J. Autologous gluteal augmentation after massive weight loss: Aesthetic analysis and role of the superior gluteal artery perforator flap. Plast. Reconstr. Surg. 119: 345, 2007.)

the superior gluteal zone. Besides, Sozer et al. report only two cases of partial fat necrosis in 20 cases.

To conclude about vascularization, we can assert that the gluteal island flap is very safe thanks to its numerous perforators. Therefore, various types of undermining are possible, if not exceeding a third of the flap surface, to achieve a nice inferomedial mobilization.
article contributed significantly to our field by introducing the concept of combined resection and augmentation in gluteal contouring. However, when we attempted to use their technique, our flaps were always too superior and often resulted in a “double-bubble” where the inferior ptotic gluteal tissue sagged beneath the projecting superior portion. The Pascal-LeLouarn flap simply does not allow the surgeon to position the flap in the ideal location to fill the inferior medial quadrant of the buttocks, which is the most important area of volume loss in the massive weight loss patient. The purpose of our article was to use knowledge of vascular anatomy to improve and extend the design of autologous gluteal augmentation flaps and optimize gluteal contour by medializing the rotation point. In addition, we sought to include standard key information on our patient group, which was lacking in the Le Louarn and Pascal article, including maximum and current body mass index, age, smoking status, combined procedures, detailed accounting of complications, aesthetic analysis, and satisfaction with the procedure.

To design our flap, we reviewed the literature and found several cadaver dissections of the gluteal artery perforator blood supply to the buttocks. The perforator supply to the gluteal region is robust, with 13 to 20 vessels per gluteal region (Fig. 1). Therefore, flaps based on medial perforators are very reliable and have been used extensively to cover sacral pressure sores and lumbosacral defects. As an additional safeguard in our early experience, we used Doppler ultrasound to confirm the location of medial perforators, to optimize our ability to undermine and thus mobilize our flaps. If there is any factual question of whether the key superior gluteal artery perforators are present at approximately 7 cm and 9 cm, we invite anyone to simply confirm this with their own Doppler examination based on the landmarks outlined in our article. We do deliberately sacrifice lateral perforators of the superior gluteal artery during this process; however, they are not necessary for flap viability, and if preserved, they prevent adequate mobilization of the flap into the inferomedial quadrant of the buttocks. We did have one case of significant fat necrosis in one of our early patients. This was attributable to a technical error and not inadequacy of the concept for the flap, as dozens of flaps have been used since the publication of our article and no patient has suffered this complication. We no longer routinely use Doppler ultrasound to locate the perforator blood supply because the two key perforators are so reliably found between 5 and 10 cm from the midline on each side.

The authors seem to be confused regarding the undermining of the inferior gluteal pocket. To clarify, we undermine tissue in the medial quadrant of the buttck in a plane just superficial to the gluteal muscle from the lower body lift line inferiorly extending to within 5 cm of the inferior gluteal crease. It is interesting to note that in their communication, Le Louarn and Pascal have modified their original illustration to show more inferior undermining. We have not experienced any cases of buttck numbness after the procedure and the cutaneous sensory nerves have not been visualized in our dissections, as these nerves are located closer to the inferior gluteal crease than in our dissection. We do typically switch from electrocautery to a combination of blunt and scissor dissection in the most inferior portion of the pocket, and this may help avoid any thermal injury to nerves in the vicinity.

In our experience, the basis of the superior gluteal artery perforator flap in sound anatomy offers superior versatility in the design of autologous augmentation flaps to optimize gluteal aesthetics in patients following massive weight loss (Fig. 2). We look forward to seeing a peer-reviewed journal article of Le Louarn and Pascal’s experience of gluteal autoaugmentation in 255 patients managed without the use of drains and with no cases of fat necrosis. If this builds on their initial experience of 41 patients with no complications, this will truly be an incredible result for any operation in plastic
Emergency Room Coverage Follow-Up

Sir:

In 2004, I published an article entitled “Emergency Room Coverage: An Evolving Crisis.” This communication is a 3-year follow-up to update the readership.

In that article, I evaluated the emergency room remuneration at three different hospitals over a 30-month period: an inner city tertiary care center, an urban university hospital, and a suburban tertiary care hospital. There were unexpected findings. Although the ratio of uninsured was 66 percent in the inner city tertiary care center, 50 percent in the suburban hospital, and 22 percent in the university hospital, the lowest reimbursement was at the suburban tertiary care hospital (14.48 percent). The Inova Fairfax Hospital is in one of the wealthiest counties in the country. The patients in this hospital who required the greatest amount of services and who constituted the highest risk for untoward outcome, complications, and lawsuits were not insured.

The conclusions of the article drew much interest. I have received more correspondence on this article than on any other publication. I surmised that if a per diem or contracted rate for emergency room plastic surgery services was not covered, this luxury in the emergency room may cease to exist in the future. So what has transpired in three years?

1. The hospital has added two hospital-based trauma surgeons to cover the increase in trauma volume.
2. The hospital has had to hire four hospital-based ortho-trauma surgeons to replace the ortho-trauma call schedule.
3. Ophthalmology calls coverage, in essence, ceased to exist.
4. Otolaryngology and urology have both revolted; urology as a department dropped privileges secondary to the burden of uninsured care. They later recanted after negotiations.
5. The hospital has now agreed to pay uninsured care at a rate of 85 percent of Medicare from a hospital-funded financial pool.

In 3 short years, this growing institution has had to replace several key services with hospital employees and now pays outside physicians at a contracted rate through a hospital-funded private pool. This is done to avoid violating Emergency Medical Treatment and Active Labor Act rules on specialty coverage for the emergency room.

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Surgical Strategies for Brachial Plexus Polio-Like Paralysis: An Addendum

In “Surgical Strategies for Brachial Plexus Polio-Like Paralysis” (Plast. Reconstr. Surg. 120: 482, 2007), from Chang Gung Memorial Hospital, the affiliations note at the beginning and the author signoff at the end should have included “Medical College, Chang Gung University.”