GUIDELINES

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Reconstruction of Neck Scar Contractures Using Supraclavicular Flaps

Sir:

This communication is in regard to the article entitled “Reconstruction of Neck Scar Contractures Using Supraclavicular Flaps: Retrospective Study of 30 Cases” by Vinh et al. (Plast. Reconstr. Surg. 119: 130, 2007). I am glad to see that the supraclavicular island flap is used worldwide and features great success rates. I have some questions, however, about this specific publication. The supraclavicular island flap has been researched extensively; my coworkers and I have published articles in Plastic and Reconstructive Surgery,1–4 and there have been numerous presentations at international congresses over the last 10 years.

In this article, however, Vinh et al. seemingly present this method of neck reconstruction as their own. In the Introduction, they state that “they have added to the development of the flap through numerous clinical and anatomical studies.” No single article by the authors is cited, but two of my articles are quoted. I described the raising of the flap and its anatomical basis extensively in 1997 and 2000, and it seems that no new information is contained in this article. In addition, the anatomical drawing in the article seems to be a redrawing of my drawing in my original Plastic and Reconstructive Surgery publication.

Clinically, I have taken the use of this flap much further by pre-expansion to ultrathin flaps for facial reconstruction. This approach has also already been published in this Journal but is not mentioned in this article.

Vinh et al. also describe the use of skin grafts for donor-site morbidity in two-thirds of the cases; however, I have never needed a skin graft, even when using flaps larger than those mentioned in the article.

In conclusion, I believe that the authors do not give enough credit to the fact that the supraclavicular island flap for neck reconstruction had been described and published more than 10 years earlier and that its clinical use has been extended much further. On the other hand, I congratulate the authors on their large and successful series using this extremely useful and versatile flap.

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REFERENCES


Preoperative Osseous Dysmorphism in Unilateral Complete Cleft Lip and Palate

Sir:

I read with great interest the article by Kane et al. entitled “Preoperative Osseous Dysmorphology in Unilateral Complete Cleft Lip and Palate: A Quantitative Analysis of Computed Tomography Data,” published in the April 1, 2007, issue of the Journal (Plast. Reconstr. Surg. 119: 1295, 2007). In this study, 28 infants with unilateral cleft lip and palate underwent high-resolution computed tomography scanning before surgery. The authors concluded that the premaxilla was...
displaced toward the cleft side as a result of the cleft. In addition, they postulated that the unilateral cleft affects development of the entire face and possibly the cranial base. In 1986, Delaire and I published a study of seven infants with unilateral cleft lip and palate who also had preoperative computed tomography scans. We compared these scans to those of an age-matched group of five normal infants who had been scanned for other reasons. Kane et al. did not have age-matched controls, which they stated limited their findings. My coauthor and I found that there was asymmetry of the cranial base and, to an extent, the orbits. The cleft side was always more obtuse, with the petrous portion of the temporal bones more flared and asymmetrical. This effectively compresses the middle cranial fossa; thus, structures housed in the temporal bone, such as the eustachian tubes, are malpositioned. My coauthor and I concluded that there are effects on the cranial base and other parts of the skull in unilateral clefts, as previously suggested by Wardill.

We were also frustrated with the U.S. Food and Drug Administration’s labeling requirement, which affected saline-filled implants as well as silicone gel implants, similar to the ones commonly used in Europe. We therefore conducted an experimental in vitro study to assess the effect of povidone iodine on the physical properties of the implant shells. Solutions of povidone iodine with concentrations ranging from 0.01% to 10% (Betadine Solution neat) were used. Our study showed that there is no effect on the mechanical properties of the implant shells from the use of povidone iodine for irrigation of the pocket or the implant itself. Although our study was performed on shells from gel-filled implants, these shells are the same as those used for saline-filled implants, and our conclusions can also be applied to those.

The issue of fibroblast toxicity by povidone iodine raised by Dr. Wiener has been the object of several studies. We would like to submit that this effect could actually be beneficial in the battle against capsular contracture. Prantl et al. showed that fibroblasts were among the main cellular populations identified histologically in breast implant capsules, and this toxicity on fibroblasts could explain why povidone iodine has been found to be one of the most effective irrigants in decreasing capsular contracture. Of course, further studies are warranted to identify the quantitative effect of povidone iodine on this cellular population, but we believe that this issue should be taken into consideration when discussing capsular contracture prevention.

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REFERENCES

The Role of Betadine Irrigation in Breast Augmentation

Sir:

We read with great interest the article entitled “The Role of Betadine Irrigation in Breast Augmentation” by Dr. Wiener. We are in complete agreement with the author, and we would like to draw the readers’ attention to our own work published in the Journal a few years ago.

We compared these scans to those of an age-matched group of five normal infants who had been scanned for other reasons. Kane et al. did not have age-matched controls, which they stated limited their findings. My coauthor and I found that there was asymmetry of the cranial base and, to an extent, the orbits. The cleft side was always more obtuse, with the petrous portion of the temporal bones more flared and asymmetrical. This effectively compresses the middle cranial fossa; thus, structures housed in the temporal bone, such as the eustachian tubes, are malpositioned. My coauthor and I concluded that there are effects on the cranial base and other parts of the skull in unilateral clefts, as previously suggested by Wardill.

We were also frustrated with the U.S. Food and Drug Administration’s labeling requirement, which affected saline-filled implants as well as silicone gel implants, similar to the ones commonly used in Europe. We therefore conducted an experimental in vitro study to assess the effect of povidone iodine on the physical properties of the implant shells. Solutions of povidone iodine with concentrations ranging from 0.01% to 10% (Betadine Solution neat) were used. Our study showed that there is no effect on the mechanical properties of the implant shells from the use of povidone iodine for irrigation of the pocket or the implant itself. Although our study was performed on shells from gel-filled implants, these shells are the same as those used for saline-filled implants, and our conclusions can also be applied to those.

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REFERENCES
Reply
Sir:

I thank Drs. Zambacos, Mandrekas, and Morris for their kind comments regarding my observations on this important subject. The studies conducted by Dr. Zambacos et al. have been corroborated by other studies, as cited in the article, confirming no deleterious effect on the implant shell by povidone iodine (Betadine).

The concern regarding tissue toxicity was primarily from the standpoint of wound-healing problems. This has clearly been shown, as cited in my article, to not be an issue. My personal use of Betadine is a dilution of 50%, a concentration that is significantly higher than that used by many other authors; as discussed, this dilution has not been shown to affect wound healing at the incision. With regard to the myriad possible reasons that Betadine is effective in decreasing capsular contracture by a significant rate, I believe basic research studies would be valuable, not only to plastic surgery but also potentially to other surgical specialties, for improving outcome. The possible effect on fibroblasts is certainly an intriguing idea and a possible additional explanation for the value of Betadine.

My attempts to reverse the policy of the U.S. Food and Drug Administration, through multiple communications with them and both Allergan/Inamed and Mentor, are at a standstill. Although the consensus among plastic surgeons is clear regarding the use of Betadine with breast augmentation, this issue is being ignored at other levels, and I strongly believe that allowing plastic surgeons to treat patients with the best possible care is our duty.

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The Use of Quantitative Abductor Pollicis Brevis Strength Testing in Patients with Carpal Tunnel Syndrome

Sir:

I enjoyed the well-documented and detailed article by Liu et al. entitled “The Use of Quantitative Abductor Pollicis Brevis Strength Testing in Patients with Carpal Tunnel Syndrome.”1 There is a particular difficulty in obtaining long-term follow-up of study patients, and the efforts to document a group of them and report their results 7 years later are commendable.

Having performed abductor pollicis brevis strength assessments in some 4000 carpal tunnel patients over the past 14 years, I am confident of its validity and consistency in objectively evaluating carpal tunnel syndrome.2 There is a remarkable correlation between strength and clinical progress in both surgical and nonsurgical patients. The more symptomatic hand is invariably weaker on abductor pollicis brevis testing. Objective assessments of abductor pollicis brevis strength changes in patients with improvement or deterioration of symptoms is invaluable, whether they have undergone surgical or conservative treatment.

I also believe that correlation of electromyographic testing combined with abductor pollicis brevis strength testing is worthwhile. My colleagues and I have just completed a 50-patient study which we intend to publish shortly. We hope it will help establish recognition of the merits of abductor pollicis brevis strength testing as a simple, reliable, inexpensive, and objective examination.

The accuracy of the measuring devices for clinical use can be much less exacting than that of the laboratory-quality instruments often used in muscle measurement studies. There is such a profound loss in abductor pollicis brevis strength in the typical carpal tunnel patient (1 kg or so) that a 50-g accuracy is well within acceptable tolerances.3

The other anecdotal comment I should make is that patients with abductor pollicis brevis strength of less than 1 kg on presentation often do much less well in terms of symptom resolution. They almost invariably are relieved of their pain, but they may still be symptomatic in terms of numbness with repetitive activities or when sleeping or driving. Warning them ahead of time about this possibility avoids many prolonged discussions postoperatively.

Even when patients still have subjective symptoms, almost all will demonstrate some increase in abductor pollicis brevis strength. This may require several months to achieve.

There is a documented, objective preoperative and postoperative strength measurement. This is especially helpful in the unhappy patient with compensation and insurance claims, who complains postoperatively that he or she is really not better. The patient often implies some fault on the surgeon’s part. This measurement, which a patient cannot fool, has saved many prolonged discussions and avoided what could potentially have become a somewhat confrontational situation.

In summary, I strongly support Liu et al.’s suggestion that this test offers a valid and objective measurement of the status of a patient’s carpal tunnel syndrome. It correlates well with other clinical findings. Their article also outlines the steps we must all take to objectively determine the true role of the abductor pollicis brevis strength measurement in becoming a useful standard examination for evaluating carpal tunnel syndrome.

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REFERENCES

**Reply**

**Sir:**

We appreciate the opportunity to respond to the letter by Dr. Bell supporting the study of the use of quantitative abductor pollicis brevis testing in evaluating carpal tunnel syndrome. Despite the ubiquitous nature of the syndrome and the obvious need for objective methods of diagnosis, there is still no universally accepted, reliable, sensitive, and specific method for diagnosis and quantification. Furthermore, in reviewing the vast literature regarding carpal tunnel syndrome, it becomes clear that many support the sensory examination as being more definitive and more sensitive in the diagnosis of carpal tunnel syndrome, while there are fewer studies that support the motor and muscle strength examination as being better, or at least as good, at defining and quantifying the syndrome. Electrodiagnostic testing is still considered by many to be inferior to the clinical diagnosis, and its significance in postoperative evaluation is even less well documented.

We agree with Dr. Bell that abductor pollicis brevis strength assessments are important in the diagnosis and consequent follow-up and evaluation of these patients. We also agree that the majority of patients improve in strength after surgery (85 percent at 6 weeks and 89 percent at 7-year follow-up in our study). We do not have any definitive data on electromyographic testing, but we look forward to Dr. Bell’s study evaluating these results critically in reference to abductor pollicis brevis strength testing.

In conclusion, we thank Dr. Bell for his comments. Abductor pollicis brevis muscle testing has been undervalued and should be considered a routine part of the diagnostic workup and management of patients with carpal tunnel syndrome.

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**REFERENCES**


**Factors Impacting Thromboembolism after Bariatric Body Contouring Surgery**

**Sir:**

I am writing in reference to the article in the April 15, 2007, issue of the *Journal* entitled “Factors Impacting Thromboembolism after Bariatric Body Contouring Surgery,” by Shermak, Chang, and Heller (*Plast. Reconstr. Surg.* 119: 1590, 2007). Missing from the risk factors for venous thromboembolism listed in their Table 1 is general anesthesia greater than spinal or epidural anesthesia. The way to prevent venous thromboembolism in these patients is to avoid general, spinal, or epidural anesthesia. It is the common denominator for all deep vein thrombosis and pulmonary embolism cases reported in the literature for plastic surgery in general. The mechanism of action is quite simply that general anesthesia and spinal epidural and narcotics all depress the body’s functions to such an extent that all muscles lose their tone as a result. The blood in the deep veins within the soleus and gastrocnemius muscles coagulates from stagnation. Attempts to improve that situation by pneumatic pumping, early ambulation, and anticoagulants do not prevent deep vein thrombosis or pulmonary embolism. They merely decrease the incidence by five- or 10-fold. The way to prevent this lethal problem is quite simply to avoid general anesthesia and opiates. I use Valium and ketamine dissociative anesthesia, wherein all of the body’s reflexes remain intact and yet the patient is rendered completely comfortable, with no memory of the events. Patients are able to walk out of the operating room under their own power.

An alternative is to use propofol and ketamine, as advocated by Friedberg. Ketamine has an added advantage of decreasing platelet adhesiveness, which may contribute to its prophylactic affects in preventing deep vein thrombosis. We have performed more than 30,000 procedures without ever having a case of deep vein thromb-
bosis or pulmonary embolism. Chuck Vinnik and Tom Baker have each performed far more procedures with the same perfect prophylactic prognosis.

I write to urge the authors and all of our colleagues to consider dissociative anesthesia, either with Valium and ketamine or propofol and ketamine, to avoid the deleterious effects of general anesthesia. In 30 years, I have never had a case of deep vein thrombosis, pulmonary embolism, chipped tooth, vocal cord injury, positive pressure, pneumothorax, negative pressure, pulmonary edema, malignant hyperthermia, flash pulmonary edema, fire in the operating room, fire in an anesthesia apparatus, or fire during intubation, all of which have been reported with the use of general anesthesia.

The discussion by Downey (Plast. Reconstr. Surg. 119:1597, 2007) that follows leads us to another article that lists many ways to counteract the problem caused by general anesthesia. Those authors spend 17 pages describing the cause of venous thromboembolism and fail to notice the common denominator. They do list “general anesthesia (risk increases with each hour in surgery regardless of procedure)” as “known to be a risk factor but probably not in the top 10; do not disregard as unimportant.” Obviously, if risk increases with every hour, we should extrapolate back to zero. Eliminate general anesthesia and eliminate venous thromboembolism! Why not just avoid the cause of all those problems: general anesthesia.

Everyone involved with cosmetic surgery will benefit from reading Friedberg’s new book, Anesthesia in Cosmetic Surgery. DOI: 10.1097/01.prs.0000287445.61666.29

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REFERENCES

Perforator-Plus Flaps: A New Concept in Traditional Flap Design

Sir:

I carefully read and appreciated the article entitled “Perforator-Plus Flaps: A New Concept in Traditional Flap Design” by Sandeep Mehrrota published in the February 2007 issue of the Journal (Plast. Reconstr. Surg. 119:590, 2007). I congratulate the author on his approach to flap surgery. In the era of perforator flaps, we are convinced that the perforator concept must be applied to every flap because we have sufficient knowledge to ensure safe skin vascularization. Thus the concept of a perforator-plus flap is one that should avoid the now obsolete, as Cormack and Lambert recently stated, random flap concept.

I, however, would like to make two points about the article. First, the concept of basing a very long flap on the lower limb is not new. Although they didn’t call it a perforator flap, Fix and Vasconez1 used and reported this kind of flap as early as 1991. As Sir Sidney Sunderland stated, we should “honor those who go first, even if those who came later went further.”

Second, I have one suggestion for flap design. The author states that the flap was raised as a peninsula, instead of islanded, to improve venous drainage. Poor venous drainage, however, is not a matter of perforator insufficiency. Each perforating artery has, in fact, two venae comitantes. When venous drainage is insufficient, it is only because the rotating movement of the pedicle makes the thin-walled, low-pressure vein twist and collapse. With meticulous perforator skeletonization to free the vessels from all attachments to the surrounding tissues, no twisting will occur and an islanded, very long propeller may be raised and turned even 180 degrees with no worries about venous stasis.2

Having said that, I again congratulate the author on his approach to flap surgery.

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REFERENCES

Reply

Sir:

“An idea is not responsible for the people who believe in it.”

—Don Marquis

I thank Dr. D’Arpa for his insightful comments and the issues he raised. The concept of raising long flaps in the leg is undoubtedly not new. What is pertinent is that there is crystallization of anatomical and physiologic facts that has led to the evolution of “perforator-plus” flaps. Traditional flaps with preserved perforators...
can be raised in the lower extremity or elsewhere with reproducible safety and effectiveness. Fasciocutaneous flaps raised along with and including the subcutaneous axial vessels, such as the sural and saphenous arteries, have perforator feeders at regular intervals. These can be considered “boosters” to the flow in the main channel and have an important contribution to overall flap dimensions, particularly length. As mentioned in the article, conscious effort was made to preserve subcutaneous neurovascular bundles. Though the perforator provides the inflow, the subcutaneous plexus oriented along the neurovascular bundles may improve axially of supply and contribute to length. Though it is feasible, I have had no occasion to dissect and safeguard multiple perforators along axial arteries and create “superlong” flaps.

The cases depicted in the article indicate the efficacy of these flaps in acute severe trauma, in deeply burned skin, based distally in the lower third of leg and in the presence of infection. These adverse factors are not only unsuitable for raising traditional flaps, but some are even contraindications for the same. Perforator-plus flaps are robust and survived with no loss in all cases.

All perforators are not accompanied by paired ve- nae comitantes. These friable veins are also likely to be disrupted in the setting of trauma. My colleagues and I have successfully performed flap rotation by 180 degrees in some cases. Rotation in an islanded flap will invariably lead to twisting of the perforator, which is the flap pedicle. Minimal perforator mobilization will localize the twist to a narrow area with complete venous occlusion. Meticulous deep perforator skeletonization has the effect of distributing the twist over a long gentle spiral, possibly without significant venous flow impediment. This process is time-consuming, and the delicate veins are prone to injury. Early flap edema in islanded flaps is undoubtedly contributed to and compounded by the lack of lymphatic drainage. The additional base in perforator-plus flaps is evidently contributing to both venous and lymphatic drainage. I have observed minimal or no edema in most perforator-plus flaps I have used over the last few years.

I would like to reiterate that the concept of perforator-plus flaps is a continuum in the desire to improve flap surgery and is likely to receive more input and undergo more improvement.

“Now this is not the end. It is not even the beginning of the end. But it is, perhaps, the end of the beginning.”
—Sir Winston Churchill

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