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Who was the first in history to treat radial nerve palsy by tendon transfer?

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Letters

Allograft Skin as a Permanent Tissue Repair: To Mesh or Not?

Sir:

We read with great interest the recent report by Dr. Pomahac et al.¹ We fully agree that skin allograft is a valid option in very selected cases.

Immunosuppression has been suggested to play a role in skin allograft rejection since 1942, when two plastic surgeons, Dr. Brown and Dr. McDowell, performed allotransplantation of skin in a burn patient with subsequent skin allograft rejection 3 weeks later.² They stated that "If a second crop of homografts from the same donor were applied to the patient at this time, one would expect almost complete failure to take." Transplantation medicine has since adopted this theory.

To date, no controlled or randomized clinical trials have been published regarding the role of systemic immunosuppression on skin allograft healing. Level IV case studies suggested that for cyclosporine, at least during and shortly after treatment, skin allograft rejection is diminished. Allograft skin appears less im-

munogenic and might serve as a matrix for autogenous cells to remigrate into the skin allograft.³

The depth of débridement of a given burn injury has not been addressed in the published case series addressing allograft skin transplantation. Partial-thickness burns have been reported to be treated successfully with viable human allografts with no prior surgical excision.⁴ Whether a tangential débridement of a second-degree burn injury and an epifascial necrosectomy in a third-degree burn injury responds the same to skin allograft transplantation has not been addressed to the best of our knowledge in a systematic way. Although a more sufficient healing of a skin allograft might be expected after tangential débridement rather than epifascial débridement, systemic factors or systemic circulating stem cells might play a role if skin allografts reintegrate as skin on the fascia.

In addition, the treatment of the skin allograft with or without a mesh has not been evaluated systematically either. A case series with three patients receiving cyclosporine A and skin allografts led to skin rejection in two cases with unmeshed allograft skin 2 weeks after cyclosporine A cessation, whereas the meshed allograft healed well without and visible evidence of rejection.⁵ The mesh might facilitate migration of recipient skin cells way better than unmeshed allograft skin, which has to be determined.

Furthermore, it has to be considered to what degree a systemic burn injury exerts immunosuppressive action when interpreting the clinical cases with immunosuppression by means of low-dose steroids, as in the case series by Pomahac et al., or systemic profound immunosuppression after organ transplantation. In conclusion, meshed skin allografting during immunosuppression appears to be a valid consideration in selective patients, which might be studied in a more systematic way in the future.

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Focused Ultrasound for Noninvasive Body Contouring in Cellulite

Sir:

We read with great interest the recent report regarding the technical and preclinical assessment of nonthermal focused ultrasound for body contouring.¹ Nonoperative alternatives to liposuction or surgical resection for body contouring appear attractive to the majority of patients. Thus, clear-cut prospective studies with appropriate power and outcome measures are mandatory in this field of body contouring, and we appreciate the comprehensive preclinical workup by the aforementioned group regarding nonthermal focused ultrasound. Based on their experimental results in a porcine model, they suggest that a cavitation-like effect is generated by each shock, with a treatment focus in the subcutaneous and not in the dermis, without any evident temperature change.

Extracorporeal shock wave therapy has been suggested as another ultrasound therapy in the treatment of lipedema and cellulite. One of the first reports of extracorporeal shock wave therapy among 26 women showed improved skin properties with mitigation of oxidative stress.² Another uncontrolled cohort study with 21 women using the low-energy ActiVitor-Derma (SwiTech Medical AG, Kreuzlingen, Switzerland) focused shock wave device with 0.018 mJ/mm² at 5- to 25-mm depth two times per week over 6 weeks (96,000 shots per person) suggested a remodeling of the collagen of the dermis.³ Outcome was measured by high-frequency ultrasound.

Recently, 59 women were analyzed in a nonrandomized clinical trial with two different dosages of focused extracorporeal shock wave therapy.⁴ Outcome was determined by using the DermaScan C (Cortex Technology, Hadsund, Denmark) ultrasound system, skin elasticity measurement using the DermaLab System (Cortex Technology), and photographs. Over 4 weeks, skin elasticity increased by 73 percent, with a further improvement to 105 percent after 6 months' follow-up. A stimulation of microcirculation and altered cell permeability was hypothesized. However, to date, there has been no randomized controlled trial regarding the effectiveness of extracorporeal shock wave therapy for the treatment of cellulite.

As far as the Contour I (UltraShape Ltd., Tel Aviv, Israel) is concerned, the authors cite two clinical studies suggesting that the Contour I focused ultrasound device is a safe and effective technology for body contouring. The multicenter trial regarding the Contour I device was a nonrandomized, controlled clinical study

with 164 subjects, with only 27 in the control group.⁵ Notably, a reduction of approximately 2 cm in the treatment area was achieved within 2 weeks after a single treatment, which lasted for 12 weeks.

Given the aforementioned attractiveness of a noninvasive measure to improve body contour, further prospective, randomized, controlled trials are mandatory to elucidate the role of these novel applications. It would be interesting to learn more about the similarities and differences between extracorporeal shock wave therapy and the Contour I focused ultrasound device. Furthermore, we wonder whether given differences in both device technologies might lead to a differential clinical outcome in noninvasive body contouring.

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Vascularized Groin Lymph Node Transfer Using the Wrist as a Recipient Site for Management of Postmastectomy Upper Extremity Lymphedema

Sir:

We read with interest the article by Lin et al.¹ on surgical treatment of postmastectomy lymphedema. We would like to commend them on reporting an exciting and novel technique with good reported results and a low complication rate.

We would like to raise a few points regarding their study. The specialist lymphedema team within our unit reported a reduction in arm circumference as high as 50 percent within the first 8 weeks of nonsurgical treatment in the majority of their patients. This gives comparable results to this study without the need for lengthy and complex surgery with its inherent risks. We feel it would be useful to know the extent of lymphedema in this group of patients, using one of the recognized grading systems, as it is has been reported that more severe disease responds less favorably to treatment.² In addition, we were hoping the authors could provide more details regarding the extent and success of nonoperative management options used in their patients before surgery and whether such treatment was continued in the postoperative period.

Our main reservation concerning the study is the lack of information regarding donor-site morbidity. Although the authors state that the superficial inguinal lymph nodes that were transferred mainly drain the abdominal wall, there is presumably a chance that removing them may adversely affect lymphatic drainage of the abdominal wall or even the lower limb. It would clearly be very distressing for the patient to gain improvement in the symptoms of their upper limb at the expense of lymphedema at another site. Despite the ample length of follow-up of the patients in the study, this does not seem to have been noted.

Finally, we feel that the results could have been recorded and described in a clearer and more meaningful manner. The main outcome measure was arm circumference, which was reported as a change in circumference relative to the difference between the affected and unaffected arms. We found this to be a confusing method of reporting the results and suggest that absolute changes in arm circumference would be a clearer, simpler method of describing the excellent results that appear to have been achieved by the authors. Frequency of cellulitis was described as an intended outcome measure but was only subjectively reported by the patients, as was hand function. Both of these parameters could have easily been measured objectively during the routine follow-up period. Similarly, lymphoscintigraphy was performed twice in the postoperative period and was described as "improved" but without any real quantitative data to back this up or any comment on whether the transplanted lymph node demonstrated any uptake of the technetium-labeled radiotracer.

We would like to congratulate the authors on their description of this novel technique that we hope will prove useful in the management of this troublesome condition. We look forward to seeing their long-term results and hope they may consider some of our suggestions for making the results easier to understand and more applicable to a wider surgical audience.

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Reply

Sir:

We would like to thank Elizabeth Chipp et al. for the helpful comments in response to our article on vascularized groin lymph node transfer.¹ In response to their questions, we would like to make the following comments. First, they mentioned a 50 percent reduction in arm circumference within the first 8 weeks with nonsurgical treatment. We are of the opinion that this is a relatively short follow-up and that the lymphedema will recur when the nonsurgical treatment is discontinued. In our study, we had an average follow-up of 56.3 months following surgical treatment, with no recurrence.

All 13 of our patients previously responded poorly to conservative treatment. If they had benefited from conservative treatment, they would not have been offered the surgery. In addition, if the lymphoscintigraphy did not demonstrate any drainage from the axilla, the vascularized groin lymph node transfer was suggested. In terms of severity, three of our patients had symptoms of lymphedema for less than 1 year (stage 1, early edema); and the remaining 10 patients had suffered from lymphedema for more than 1 year (stage 2, older edema) according to the classification of Becker et al. We are also in agreement that patients with "more severe disease responded less favorably to treatment," which was mentioned in the discussion section on page 1274, paragraph 4. Postoperatively, the entire patient group did not require any routine physiotherapy, because of the improvement from surgery. We do not recommend routine daily physiotherapy, as this brings much inconvenience to the patients.

The donor-site morbidity is usually a source of concern for patients when using the groin lymph node as a donor site. None of our patients experienced any problems in their donor sites; in particular, there were no findings of lymphedema in their abdomen or lower limbs (p, 1270, left column, first paragraph, lines 4 and 5). There are two groups of lymph nodes in the

groin: the superficial and deep groups. All of the lymph nodes were harvested from the superficial group. Therefore, the theoretical possibility of developing secondary lymphedema in the abdomen and lower extremity is extremely small (p 1274, paragraph 3).

Although we had collected the data for absolute decrease in arm circumference for each patient, we disagree that this is the best way of measuring the surgical outcome. Lymphedema may be confined to one limb, but the factors that influence its severity are often systemic, and the circumference of both limbs may be affected by body weight increase or decrease, hydration, dehydration with diuretics, and episodes of cellulitis. The absolute changes are easy to read, but we do not feel they appropriately represent the symptoms of lymphedema.

In our study, we did not provide quantitative data for the degree of cellulitis or hand function. We feel that the absence or presence of cellulitis is a valid result in itself. With regard to hand function, objective measurements were not carried out, as the preoperative disability was completely attributable to the presence of swelling and edema and was not a motor or sensory deficit. Such improvement, after the removal of swelling, is best assessed subjectively. We agree, however, that more quantitative and objective measurements can be obtained from future studies, and we are in the process of carrying out further studies for this purpose. DOI: 10.1097/PRS.0b013e3181c87b7e

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Chemoprotection/Bioprotection Achieved with Botulinum Toxin Type A Injections in Tendon Surgery

Sir:

We read with great interest the recent report by Dr. De Aguiar et al. regarding the use of botulinum toxin in flexor tendon repairs.¹ The authors used electromyography-guided injections of 25 U per injured flexor muscle belly of botulinum toxin type A (Botox; Allergan, Inc., Irvine, Calif.) following zone 2 tendon ruptures. We would like to comment on some issues raised by the authors. Because a nonrandomized controlled study was performed, costs of treatment should be highlighted, given the current limitations in the health care system.

As far as the reported results are concerned, the authors state that they had significantly more excellent results (94 percent compared with 81 percent; $p = 0.002$). The authors explain that in both groups, 6 percent achieved good results ($n = 8$) and state a value of $p = 0.058$ using the Fisher's exact test for this comparison. At least for us, it is hard to understand and follow how the calculations of identical percentages might lead to a value of $p = 0.058$. We would appreciate it if the authors could clarify this issue to us.

The authors describe the term "chemoprotection" for the mechanism of action of Botox in the rehabilitation of tendon surgery. In 2007, a North Carolina group suggested a "bioprotection" of tendon repair using botulinum toxin type A.² They blocked the gastrocnemius muscle in Achilles tendon rupture with 6 U/kg body weight botulinum toxin type A versus saline injections in rats. The muscle was incapable of actively producing enough force to rupture the repaired Achilles tendon.

Notably, botulinum toxin type A up-regulates the expression of substance P and calcitonin gene-related peptide, neuroproteins essential in tendon healing.³ Currently, we cannot exclude that chemoprotection or bioprotection of a sutured tendon is supported or influenced in any way by such effects on the neuropeptides. Recovery following intramuscular injection of botulinum toxin type A involves a sequence of cellular events, where the insulin-like growth factor 1 pathway plays a crucial role.⁴ Thus, mechanisms currently under investigation might be responsible for the favorable outcome following Botox chemoprotection or bioprotection of sutured tendons.

From a clinical point of view, injections of botulinum toxin type A have been suggested to be beneficial in treating lateral epicondylitis in a randomized, double-blind, placebo-controlled, multicenter study.⁵ We be-

lieve that future prospective, randomized, controlled trials are warranted to elucidate the role of Botox injection in the rehabilitation of tendon injuries and would like to congratulate the authors for their inspiring contributions.

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Reply

Sir:

I thank Dr. Karsten Knobloch and colleagues for the pertinent comments regarding the publication “Chemoprotection of Flexor Tendon Repairs Using Botulinum Toxin.”

In the United States, the approximate cost per vial of 100 U of botulinum toxin type A (Botox; Allergan, Inc., Irvine, Calif.) is approximately \$600. In Europe, the cost is approximately €230 (plus value-added tax). These costs vary from state to state in the United States and from country to country in the European Economic Community (figures were provided by Genop Health Care, Halfway House, Johannesburg, South Africa, as of August of 2009).

The results of outcomes of flexor tendon injuries treated with Botox or without (control) Botox were shown at 18 months postoperatively in Tables 2 and 3, respectively. Total active range of motion was measured using the Kleinert and Verdan¹ scoring system as ex-

cellent (total active range of motion >230), good (total active range of motion, 189 to 229), fair (total active range of motion <189), or poor (contractures/adhesions). Tables 2 and 3 show that the Botox group achieved a statistically significant 94 percent excellent results versus 81 percent in the control group. There were no fair or poor results in the Botox group, which is highly significant against the control group, with 8 percent and 6 percent, respectively. As shown in Tables 2 and 3 both groups shared a 6 percent incidence of good results, clearly without any difference between the groups. Table 4 has a typographical error regarding the *p* value of the good results between the two groups. Using the one-tailed Fisher’s exact test, the probability should read 0.63, which as expected shows no difference between these groups.

Focal, intramuscular injection of Botox or botulinum neurotoxin type A effectively denervates a muscle by inhibiting the release of acetylcholine at the neuromuscular junction.² It decreases a spastic muscle’s ability to generate forceful contractions, thus decreasing the strength of the contractions.³ It has therefore been used widely and successfully for adjunctive treatment of conditions characterized by muscle overactivity, including strabismus,^{4,5} blepharospasm, dysphonias, hemifacial spasm, spasmodic torticollis, other dystonias,^{6,7} and cerebral palsy,⁸ and for the treatment dynamic facial lines in cosmetic surgery.^{9–12} Recently, techniques have used the ability of botulinum neurotoxin type A to chemically denervate, reversibly weaken, and thus modify muscle forces during tendon healing.^{13–15}

Ma and colleagues¹⁵ performed a study in which juvenile rat Achilles tendons were bisected and then repaired, followed by intramuscular injections of botulinum neurotoxin type A into the gastrocnemius muscles. This was compared with a suitable saline-treated control group. The spontaneous Achilles tendon rupture rate of repaired tendons in the botulinum neurotoxin type A group was three times lower than in the saline-treated group at 1 week. The force required to rupture the repaired tendons was significantly higher in the botulinum neurotoxin type A group between 1 and 3 weeks after repair ($p < 0.007$). After this, no significant difference was noted. Intramuscular botulinum neurotoxin type A produced a significant, reversible reduction in active muscle force that could be generated ($p < 0.007$).

In the botulinum neurotoxin type A group, injections resulted in a 75 percent decrease in the force of twitch contractions after nerve-muscle stimulations at 1 week and a greater than 50 percent decrease at 1 month compared with the saline-treated control, and remained significantly decreased ($p < 0.007$) up to 8 weeks. By 6 months, twitch contractions normalized. Similar results were achieved with tetanic (maximal) contractions.

Despite the obvious limitations of this study (the 1-month-old rat model cannot be extrapolated directly to flexor tendon mechanics and healing in adult humans), the above study assisted in satisfying

some of the shortcomings in our article. An avenue that my colleagues and I would like to have explored in our study would have included measurements of tendon force generated in the botulinum toxin-treated tendon repairs. This was not done, as we were concerned that measurements of tendon force may indeed induce tendon gapping or rupture. The mechanical forces required to produce tendon rupture in both treated and control groups could not be measured because of obvious deleterious effects to the patient. The study by Ma et al.¹⁵ assists in providing some of these answers, albeit in an animal model.

A variety of mechanisms currently under investigation may be responsible for the favorable outcomes following botulinum chemoprotection of repaired tendons, as follows:

Pain: botulinum neurotoxin type A injections may reduce pain,¹⁶ thereby facilitating rehabilitation and the associated active, protected early processes of tendon-healing following tendon repair.¹⁵ Botulinum neurotoxin type A has been shown to inhibit the release of neurotransmitters specifically involved in pain transmission and inflammation, including glutamate, substance P, and calcitonin gene-related protein.^{17–20} Botulinum neurotoxin type A prevents the stimulated release of calcitonin gene-related protein through the cleavage of SNAP-25 protein.¹⁹

Neuropeptides: botulinum neurotoxin type A injections up-regulate the expression of neuropeptides such as substance P and calcitonin gene-related protein,^{17,21–23} which have been shown to play a substantial role in the healing process following Achilles tendon rupture.^{24,25} Other trophic neuropeptides playing a role in tendon healing include autonomic neuropeptide Y, growth-associated protein 43, protein gene product 9.5, and galanin.^{24,25}

Early postoperative mobilization: as discussed in our article, chemoprotection of tendon repairs allows for early active mobilization, with its attendant benefits that facilitate the tendon-healing process.^{26–33}

Other mechanisms: botulinum neurotoxin type A could result in increased tendon strength through mechanisms independent of muscle weakening that as yet require further elucidation.¹⁵ Restoration of function and motor end plate recovery following intramuscular botulinum neurotoxin type A involves a two-stage sequence of cellular events (aneural and neural) that eventually leads to neuromuscular junction stabilization, remodeling, and myogenesis during which insulin-like growth factor 1 signaling plays a central role.³⁴

In 1997, Morré et al.³⁵ reported promising results using botulinum toxin (type and preparation unspecified) for the treatment of chronic radial epicondylitis (tennis elbow). More recently, a prospective, controlled, double-blind, randomized trial³⁶

showed statistically significant results in radial epicondylitis treated with botulinum toxin type A. In this study, the preparation used was Dysport (Ipsen Pharma, Bobenheim, Germany) at a dosage of 60 U. Conversely, Hayton et al.³⁷ performed a double-blind, randomized, controlled study on 40 patients with tennis elbow and failed to find significant benefits using 50 U of botulinum toxin injections (Botox). This demonstrates the importance of understanding the differences between various botulinum products. Clinical trials comparing different botulinum toxin formulations [Botox, Dysport, and Myobloc/Xeomin (Merz Pharmaceuticals GmbH, Frankfurt am Main, Germany; Elan Pharmaceuticals, South San Francisco, Calif.)] show that the safety and therapeutic margin rankings are ordered largest to smallest in the sequence as above.³⁸ These studies show that results using botulinum toxin are variable and dose comparisons inconclusive with unique and incomparable dose units. Ratios of mean doses of Dysport to Botox may range from as low as 2:1 to a high of 11:1.³⁹ The effects on muscle atrophy following intramuscular injection also vary between the products. Aoki et al.⁴⁰ showed significant atrophy of nearby noninjected muscle in the mouse model with Dysport and Myobloc at doses that caused moderate weakening of the injected muscle compared with Botox. This suggests that the former two products show greater local diffusion, with higher incidences of adverse events and narrower therapeutic windows.

In August of 2009, the U.S. Food and Drug Administration announced name changes for the botulinum toxin products as follows⁴¹:

Botox: new name, onabotulinumtoxinA; old name, botulinum toxin type A.

Dysport: new name, abobotulinumtoxinA; old name, botulinum toxin type A.

Myobloc: new name, rimabotulinumtoxinB; old name, botulinum toxin type B.

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Who Was the First in History to Treat Radial Nerve Palsy by Tendon Transfer?

Sir:

The first learning objective of the well-written article by Sammer and Chung, entitled “Tendon Transfer: Part I. Principles of Transfer and Transfer

for Radial Nerve Palsy,” is “to be able to describe the history of tendon transfer procedures.”¹ The authors provide some sketchy information on the early history of tendon transfers in general and mention several important contributors to the development of this surgical principle to treat radial nerve palsy. Unfortunately, the authors omitted crediting the first surgeon in history to perform a tendon transfer for this indication. Therefore, some additional historical remarks seem necessary.

The idea of using the tendon of a dispensable healthy muscle to function in place of one that has become paralyzed was first conceived by Carl Nicoladoni (1847–1902) in the early 1880s.² Nicoladoni’s concept was further popularized in the 1890s by the Polish surgeon Tomasz Drobniak (1858–1901).²

Boyes, in his classic article entitled “Tendon Transfers for Radial Palsy,”³ mentioned Drobniak as the first in history, in 1894, to use this method to treat radial nerve palsy. Unfortunately, Boyes provides no reference to Drobniak’s operation, but the account can be found in Drobniak’s most influential article on tendon transference, entitled “Ueber die Behandlung der Kinderlähmung mit Funktionsheilung und Funktionsübertragung der Muskeln,” published in 1896.⁴ In the article, Drobniak describes a case of a 4½-year-old girl who suffered a postpolio paralysis of all extensors of the hand and wrist, except the extensor carpi radialis longus and extensor carpi radialis brevis. On February 27, 1894, Drobniak transferred the extensor carpi radialis longus to the extensor digitorum, but the result was not satisfactory. Therefore, he performed another operation in which he transferred part of the extensor carpi radialis brevis to the extensor pollicis longus. The result was acceptable to Drobniak, as the girl was able to extend her thumb fully and the index and middle finger 67 percent. The fourth and fifth digits showed only 50 percent extension.⁴

Soon after Drobniak’s report, several other surgeons published their attempts to treat radial nerve palsy with tendon transfer.³ However, if someone asks us who the first in history was to treat radial nerve palsy with tendon transfers, the answer should be Drobniak, in 1894.

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The Key Roles of the Deep Fascia of the Anterolateral Thigh Flap

Sir:

We would like to congratulate Kuo et al. on their article regarding the use of the anterolateral thigh flap and the deep fascia for complex soft-tissue reconstructions (*Plast Reconstr Surg.* 2009;124:171–180). We agree that the anterolateral thigh flap is the workhorse flap for reconstruction of several soft-tissue defects. The authors present an impressive series of 973 flap anterolateral thigh flaps over 10 years. They used the deep fascia in 36 cases (12 to the abdominal wall, 15 to the lower lip/cheek, and the remaining to tendon and ligament reconstruction of extremities), 32 as free flaps and four as pedicled flaps. Twenty-eight eight patients underwent reconstruction with fasciocutaneous anterolateral thigh flaps and eight patients underwent reconstruction with musculocutaneous anterolateral thigh flaps (vastus lateralis or rectus femoris muscles flap according to most recent classifications and depending on the muscle harvested). The authors reported that five of the 15 patients who underwent lower lip reconstruction suffered occasional mild oral incontinence. Regarding this problem, we would like to cite Yildirim et al., who did not report microstomia or oral incompetence, in either the resting condition or during speaking and eating, in 11 patients who had undergone lower lip reconstruction. The authors used fascia lata extensions divided longitudinally into two fascial strips at both margins of the flap: two strips were tunneled through the orbicularis muscle in the upper lip and sutured to each other and to the orbicularis muscle, and the remaining two strips were anchored to the zygomatic bone periosteum by permanent sutures.¹ Evidently, this clever expedient provides a long-lasting functional outcome.

Kuo et al. state that thigh thickness represents the reason why the anterolateral thigh flap is used in Asians more frequently as compared with Western patients and list intraoperative thinning, secondary debulking, and other flaps as possible alternatives. In our opinion, these solutions are less feasible for oral cavity reconstruction as compared with head and neck and extremities.^{2–5} The lateral cutaneous thigh flap definitely provides one of the largest surface areas of any skin flap available for reconstruction. The flap skin is thin, pliable, and frequently hairless,

especially in women. Fat deposits in the thigh are usually distributed more proximally than where the flap is sited. As a consequence, the proximal portion of the flap can be used to provide bulk and the thinner (distal) aspect of the thigh can be used for reconstruction of the thin oral and pharyngeal membranes.⁶ Moreover, thinned adipofascial anterolateral thigh flaps represent a valid and safe alternative when thin pliable tissue is necessary, and this is especially true for oral cavity reconstruction.^{4,5} Also, Alkureishi et al. demonstrated the inadequate arterial supply in the subdermal plexus of the skin distal to the perforator in thinned anterolateral thigh flaps used for the oral cavity.^{2,3} The reason lies in the vascular supply: the main bloody supply to the skin arises from the outer fascial layer, passing as fasciocutaneous or musculocutaneous perforators, with branches oriented perpendicular or radiating in all directions. The dominant vascularity of fascial flaps is in the prefascial plexus, which is found most prominently around the shoulders and extremities where the skin is tightly adherent to the underlying deep fascia; on the contrary, the subdermal vascular plexus tends to be dominant in loose-skinned areas such as the trunk.^{2,3,6} Thus, the thinned adipofascial anterolateral thigh flap reveals a safe procedure because the fascial plexus is preserved, which guarantees superior results in oral cavity reconstruction, providing a functional hairless wet tissue according to the principle of replacing “like with like” tissue and avoiding the nervous anastomosis necessary for sensate flaps. For these reasons, we recommend that the anterolateral

thigh flap be thinned in an adipofascial configuration either to preserve the vascular fascial network or to make maximum use of its support properties, especially in the iliotibial tract, where it is most thick (Fig. 1).

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DISCLOSURE

Neither of the authors has any commercial associations that may pose or create a conflict of interest with information presented in this communication.

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Fig. 1. Thinning an adipofascial anterolateral thigh flap is safe and useful, because the prefascial plexus is preserved and the surgeon chooses the adequate thickness for the defect. The excised skin can be grafted on thinned adipofascial flaps when used for extremity reconstructions. This adipofascial flap was overturned (deep fascia toward the oral cavity) to reconstruct the anterior floor of the mouth after a T3N2M0 tumor excision; the deep fascia was anchored to the genial apophysis (to prevent flap descent) and to the hyoid bone (hyoid bone suspension).

Current Applications and Safety of Autologous Fat Grafts: A Report of the ASPS Fat Graft Task Force

Sir:

We read with interest the article entitled “Current Applications and Safety of Autologous Fat Grafts: A Report of the ASPS Fat Graft Task Force” (*Plast Reconstr Surg.* 2009;124:272–280). We would like to congratulate Dr. Gutowski and the ASPS Fat Graft Task Force for their publication, providing a systematic, evidence-based review about fat grafting for aesthetic and reconstructive purposes. This study

offers a graded summary of the evidence about indications, techniques, patient selection, and complications.

Concerning the listed “current and potential applications” of autologous fat grafting, we would add some reconstructive indications, which we showed in recent articles (evidence level, IV to V). After the first case series published in February of 2008, we verified the safety and effectiveness of scar remodeling by fat graft in more than 250 patients, including outcomes of burn, trauma, radiation damage, chronic ulceration, and peripheral nerve entrapment.^{1–5} We found that the procedure is suitable in each body area in cicatricial areas at least 12 months after injury. We observed a significant improvement in texture, color, softness, quality of skin and subcutaneous tissue, and functional motility and pain. Histologic examination after treatment showed patterns of neoangiogenesis and regularization in collagen deposition. Patients were satisfied with the procedure and the results.

The role of lipostucture and biological mechanisms in regenerative surgery are discussed. An active role and differentiation of adult mesenchymal stem cells from the stromal fraction of transplanted fat have been suggested, but it is unclear which is the relative role of the molecular components and surrounding microenvironment in modulating cicatricial remodeling. In our experience and after this evidence,^{1–5} the application of autologous fat transplantation to mature scar treatment should be included in its current indications, providing new perspectives of reconstructive and regenerative surgery. DOI: 10.1097/PRS.0b013e3181c722cf

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Reply

Sir:

The information regarding the use of fat grafts for scar remodeling submitted by Dr. Villani and colleagues is both interesting and promising. These latest contributions were not included in the ASPS Fat Graft Task Force, as they were outside the time frame during which the task force was able to collect data before the final analysis and publication of recommendations. However, we did expect that there would be continued progress in expanding the role of fat grafts, including applications in the emerging field of reparative and regenerative medicine. We look forward to hearing more from these and other investigators who develop further innovative applications of fat grafts.

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Correction: Autologous Fat Transfer for Facial Recontouring: Is There Science behind the Art?

In the article by Kaufman et al. entitled “Autologous Fat Transfer for Facial Recontouring: Is There Science behind the Art?” published in the June 2007 issue of the *Journal* (*Plast Reconstr Surg*. 2007;119:2287–2296), the fourth author’s name was misspelled. The correct spelling is Jason Roostaeian.

This error has been noted in the online version of the article, which is available at www.PRSjournal.com. DOI: 10.1097/PRS.0b013e3181d1267f

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Correction: High-Frequency Ultrasound: A Useful Tool for Evaluating the Abdominal Wall following Free TRAM and DIEP Flap Surgery

In the article by Zhong et al. entitled “High-Frequency Ultrasound: A Useful Tool for Evaluating the Abdominal Wall following Free TRAM and DIEP Flap Surgery,” published in the April 1, 2006, issue of the *Journal* (*Plast Reconstr Surg*. 2006;117:1113–1120), the third author’s name was misspelled. The correct spelling is Melanie S. Werstine.

This error has been noted in the online version of the article, which is available at www.PRSJournal.com. DOI: 10.1097/PRS.0b013e3181d12791

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Correction: First U.S. Near-Total Human Face Transplantation: A Paradigm Shift for Massive Complex Injuries

In the article by Siemionow et al. entitled “First U.S. Near-Total Human Face Transplantation: A Paradigm Shift for Massive Complex Injuries” published in the January 2010 issue of the *Journal* (*Plast Reconstr Surg*. 2010;125:111–122), the year given in the next to last sentence of the “Transplantation Procedure” paragraph (on p. 113) is incorrect. The sentence should read, “After 22 hours, the successful operation was completed on December 10, 2008.”

This error has been noted in the online version of the article, which is available at www.PRSJournal.com. DOI: 10.1097/PRS.0b013e3181d3e4d3

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