

mastopexy is challenging, I believe that “mischief and major disasters” can be significantly reduced by adherence to the following guidelines:

1. *Place implants in a submuscular position.* This reduces the risk of implant exposure, devascularization of the overlying breast tissue (with consequent nipple or skin flap loss), and excessive postoperative implant descent.
2. *Perform augmentation before mastopexy.* Preoperative mastopexy markings are simply educated guesses, as the precise amount of skin excess is unknown until after the implants are placed. Tailor-tacking (and any necessary adjustment) of the preoperative markings should be performed with the patient in a semi-upright position on the operating table *after* implant placement. This prevents underresection of skin with consequent persistent nipple and/or breast ptosis. More importantly, it prevents overresection of skin with consequent excessive tension on the closure, which leads to widespread scars and skin flap loss.
3. *Do not perform augmentation with a Wise-pattern mastopexy.* Periareolar and vertical augmentation/mastopexy patterns generally can be performed without excessive skin flap tension and tissue devascularization. However, an inverted-T closure paired with an augmentation requires significant undermining, which often leads to an unacceptably high rate of complications. In the very small subset of patients requiring a Wise pattern, consideration should be given to staging of the procedures.
4. *Do not be afraid to resect breast tissue.* Although the surgery is meant to enlarge the breasts, a small amount of breast tissue may need to be excised to facilitate closure without excessive tension. In particular, resection of parenchyma superior to the nipple-areola complex may be required to enable significant tension-free nipple-areola elevation. Similarly, a vertical mastopexy pattern requires vertical wedge excision of lower-pole parenchyma. This reduces closure tension, the need for undermining, and the risk of persistent lower-pole ptosis.

Women who request elevation and enlargement of the breasts are interested in achieving both goals with a single procedure. I believe that, judiciously performed, augmentation and mastopexy can be combined with a very acceptable complication rate.

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AESTHETIC RECONSTRUCTION OF THE TUBEROUS BREAST DEFORMITY

Sir:

We would like to respond to Drs. Spear and Ganz's discussion of our article, “Aesthetic Reconstruction of the Tuberous Breast Deformity.”^{1,2} We are grateful to Drs. Spear and Ganz for taking the time to read our article and for their comments. In response, however, we would like to clarify some misunderstandings. Our aim in publishing this article was to try to share our understanding of the etiology behind the deformity and the steps we believe are necessary to correct it.

We are not sure whether Drs. Spear and Ganz believe that the development of a constricting ring is the cause of the deformity, as do we and others.³⁻⁶ We consider that the only way to correct the deformity is to deal with this constricting ring, and that simply increasing the base diameter of the breast with an implant and reducing the size of the areola with a circumareolar mastopexy, as Drs. Spear and Ganz suggest, in fact accentuate the deformity, and many authors agree with us.⁷⁻¹¹ However, at no point in the article do we claim that our technique does away with implants. We believe that in most cases a breast implant is necessary, and we invariably place the implant in a subglandular position.

By transecting the lower pole of the breast, we are dividing this constricting ring, freeing up the breast, and creating two pillars. The tuberous breast is long, with a narrow base, and by transecting the lower pole, we are able to spread the breast tissue, thus achieving a breast shape that is flatter and wider, usually at the expense of projection. This is possible because breast tissue is not rigid and allows some molding. To avoid leaving a gap at the 6 o'clock margin, where the transection takes place, we try to bring the pillars *loosely* together with sutures. We are not repairing the transected breast, and we are certainly not reconstituting the constricting ring. If the pillars are very long, as in the case depicted in Figure 2 of our article, then we can use them to create some added volume at the lower part of the breast, exactly where it was lacking initially, by redraping the loose distal part of the pillars in a double-breasted fashion.

We apologize for not showing a frontal view of the patient in Figure 5 (case 5). This was not done to hide something, as Drs. Spear and Ganz imply, but simply because the image was unavailable. This was one of the few cases in which no implant was used, and we wished to show what was possible with our technique. We do not think that frontal views would have added much additional information in this case.

We hope that we have been able to clarify any misunderstandings, and we would welcome other authors' comments on the subject.

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LASER TREATMENT OF CONGENITAL MELANOCYTIC NEVI

Sir:

The article by Reynolds, Kenealy, and Mercer describing their experience with the carbon dioxide laser in giant congenital melanocytic nevi¹ raises a number of concerns. The authors are no strangers to hyperbole, and following their previous case report, Arons from New Haven, Connecticut, cautioned them on the use of the word "successful."² While the authors again claim significant cosmetic improvement without providing evidence to support this, it is the term "giant" that is misleading in this series.

The primary concern about congenital melanocytic nevi is their malignant potential. The available evidence is difficult to interpret. Congenital melanocytic nevi form a heterogeneous spectrum.² A critical question that needs to be addressed is whether the malignant potential of a lesion is dependent on absolute or relative cell number. The clinical correlate of this is whether you categorize lesions by absolute or relative size. Quaba and Wallace³ elected to use relative size, defining a "large" congenital melanocytic nevus as being greater than 2 percent of body surface area. Arbitrary absolute size categorization of congenital melanocytic nevi is generally regarded as follows: small, less than 1.5 cm in largest diameter; intermediate, greater than 1.5 but less than 20 cm in largest diameter; and giant, largest diameter greater than 20 cm.⁴ The study by Reynolds et al.¹ gives no dimensions for the lesions in the nine patients treated, although of the two patients shown, few clinicians would regard the patient shown in Figure 2 (case 4) as having a giant congenital melanocytic nevus.

These concerns, however, are eclipsed by concerns about the safety of the procedure. In the previously reported single case,⁵ the patient became unwell after the treatment, which is not unexpected. Subjecting a neonate to an extensive "therapeutic" burn is a very risky procedure, but what about the oncological risk that has been dismissed by these authors?

In the early 1990s, when I first became involved in exploring the role of laser treatment for melanocytic nevi, the clinical focus was on dysplastic or atypical nevi. After extensive discussion with the late David Kenealy, former director of Laser Services at Frenchay Hospital, Bristol, United Kingdom, we decided to investigate laser melanocyte interactions in vitro. Using the Q-switched 532-nm neodymium:yttrium-alu-

minum-garnet laser, melanoma cells in culture were exposed to sublethal laser energy. Frankly, the results were alarming. The cell line used, Sk-mel-23, is a transformed line and proliferation was not an issue. After exposure to the sublethal energy, there was upregulation of focal adhesion kinase proteins and $\alpha 3$ and $\alpha 4$ integrin subunits and proteolysis of integrin $\beta 1$ subunit.⁶ This suggested that the cell was becoming more malignant (i.e., developing metastatic potential). Further studies, as yet unpublished, have demonstrated that the exposed cells become more resistant to apoptosis inducers, another indicator of increased malignancy. This concern is reinforced by the work from the Hong Kong University group led by Dr. Henry Chan.⁷ Using the Q-switched 755-nm laser, three melanoma cell lines were treated, HTB66, SK-mel-24, and G361. Of these three cell lines, the level of p1614a protein in cell line HTB66 was significantly increased after laser irradiation. The *p16* gene has been proposed as a candidate gene for melanoma.

The Frenchay authors also question the role of ionizing radiation in the development of melanoma. It is the *nonionizing* part of the electromagnetic spectrum that is generally implicated in the development of malignant melanoma, principally ultraviolet radiation. The long-term effect of the carbon dioxide laser operating with a wavelength of 10,600 nm is unknown.⁸

The authors have made a bold move in their clinical experimentation, but some concurrent in vitro studies would be advisable. What is the effect of temperature on malignant transformation in melanocytes? Christophers⁹ believes the epidemiological evidence supports a hypothesis that melanoma incidence increases with increases in skin temperature. Perhaps the Frenchay group could continue their laboratory research to explore this novel perspective with regard to laser-induced thermal injury and cultured melanoma cells. DOI: 10.1097/01.PRS.0000123624.85119.ED

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