Commentary on: Iatrogenic Symmastia: Causes and Suggested Repair Technique

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In this article, the author reports the results of a single-surgeon retrospective chart review regarding the treatment of symmastia after breast augmentation in 23 patients. Conclusions based on this review are reinforced by data obtained from a concurrent 6-question survey of 181 ASAPS members regarding the etiology of symmastia. Previous anatomic study by the author is also referenced, all to make the argument that attenuation of the medial origins of the pectoralis major muscle, or outright overrelease of the muscle, leads to disruption of the soft tissue attachments of the sternum due to inferomedial pressure applied by the remnant of the pectoralis major muscle. By inference, the implant is therefore malpositioned medially, leading to symmastia. Therefore, the author concludes that the most common risk factor for iatrogenic symmastia is subpectoral breast augmentation. Additionally, based on these findings, a sequential repair protocol is proposed that includes anterior capsulectomy, thermal “popcorn” capsulodesis, soft tissue repair along the medial and inferior aspect of the pocket, reattachment of the pectoralis major, subglandular implant placement, postoperative garment support, and 6 weeks of immobility of the arm to protect the repair of the muscle. Based on this summary, further discussion is warranted.

The intraoperative findings noted in the patient cohort studied in this article noted that either dehiscence of the medial margins of the pectoralis major muscle or outright surgical disruption weakened the medial attachments of the breast, allowing the presternal “zone of attachment” to become exposed. The author then proposes that forces directed downward and inward by the overly released muscle caused the implant to migrate medially creating the symmastia deformity. Although the general idea of overrelease of the medial soft tissue attachments of the breast is undoubtedly responsible for the symmastia deformity, the sequence of events leading to medial implant malposition hypothesized by the author seems overanalyzed. The suggestion that progressive pressure downward and inward by the pectoralis major muscle remnant is what leads to implant malposition or, even further, that such forces can lead to outright disruption of a weakened muscle, contradicts observations I have made in subpectoral implant placement. It is a common misconception that inferior bottoming out of the breast after breast augmentation is caused by contraction of the pectoralis major muscle pushing the implant inferiorly. This is a general assumption that must be reconsidered because what in fact happens in almost every circumstance is that the breast implant is drawn up and out by the forces applied to the implant by the muscle as it contracts. The exact vector of pull varies depending on how much of the muscle origin is released, but the pull is always up and out toward the axilla. Even in cases where the muscle is completely overreleased, the attachments of the muscle to the overlying breast pull the breast up and out. Any bottoming out that might occur is in fact mostly due to direct surgical disruption of the inferior “zone of adherence” along the

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inframammary fold that exposes the weakened inferior breast attachments along the inframammary fold to forces from above, namely the weight of the breast implant. Along this line of reasoning then, it is not some postoperative change that causes the deformity; rather, it is a direct result of the surgical intervention with overdissection of the pocket that leads to the deformity. The same concept therefore applies to symmastia. It is not that the implant is placed either over or above the muscle; it is the fact that the soft tissues are overreleased at the time of pocket development that leads to implant malposition, no matter what pocket is chosen. Forces applied by the pectoralis major muscle play only a minor role, if any, in determining the final deformity. These differing interpretations of etiology do not simply represent an issue of semantics, as the author hypothesizes that muscle repair is an integral part of operative correction, and further, arm immobilization for 6 weeks then becomes necessary to protect the muscle repair. As well, according to the author, implant placement in the subglandular plane is required to then avoid the detrimental forces applied by the muscle. An alternative interpretation is likely to provide a more useful global approach to symmastia repair and, importantly, can avoid the need for potentially debilitating prolonged arm immobilization leading to possible contracture and loss of range of motion. Although most of the operative steps outlined by the author are well thought out and effective either separately or together, including soft tissue repair, removal of tethering scar, and “popcorn” capsulodesis of the capsule to cause it to contract and thicken to make suturing more effective, the recommendations regarding muscle management should be viewed as a preference rather than a principle. In many patients, one can easily accomplish a reliable soft tissue repair medially under the muscle and continue to use a subpectoral pocket if that is deemed the best pocket for an individual patient. Reinforcement of these types of repairs with mesh or acellular dermal matrix materials can add additional security. The main operative goal no matter what method is chosen is to accurately position the medial contour of the breast and provide a reliable durable repair.

Beyond these points, several generalizations noted by the author as presumptive evidence supporting the inferomedial muscle vector argument deserve challenge. Prior to the silicone moratorium, the idea that most augmentations were performed in the subglandular plane is an overstatement. Then, to suggest that a subsequent move to the subpectoral or partial subpectoral pocket was due to a switch to saline implants after the moratorium is likewise simply unsupported speculation. Further, the assertion that most dual plane approaches become subglandular over time is simply incorrect. All these assertions fall short of furthering the muscle-induced implant displacement argument.

To summarize, the author has correctly stated that overrelease of the medial attachments of the breast can lead to medial implant malposition, and repair of these attachments can be performed with good results. Furthermore, switching to a subglandular plane can facilitate the repair. However, the assertion that inferomedial force vectors developing secondary to release or even overrelease of the pectoralis major muscle can lead to muscle attenuation and/or outright disruption with symmastia remains an unsupported hypothesis based on the findings reported in this article. Further, the proposed surgical correction of muscle reattachment with prolonged shoulder immobilization and switch to a subglandular plane cannot be supported as a required corrective surgical strategy. It remains for the surgeon to evaluate each case individually to determine issues related to soft tissue repair, pocket plane choice, and implant size and type to provide the best long-term result.

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