Silicone implants have been placed in about 500 million patients worldwide for both aesthetic and reconstructive purposes and are still the most reliable technique for breast augmentation. A normal tissue response consisting of inflammatory infiltration and deposition of collagen fibers forming a collagenous capsule surrounding the foreign material is inevitable regardless of the implant type. Capsular contracture (CC) remains, however, a major yet unpredictable local potential complication. In fact, it is the most commonly reported complication following alloplastic breast augmentation or reconstruction and is one of the most difficult complications to treat. It is mostly unilateral and may be the most significant cause of patient dissatisfaction. The reported incidence of CC over time ranges from 2% to 74%. In 1978, Baker described 4 clinical stages of CC. In severe cases, CC can cause pain, rippling, hardening, and asymmetry of the breasts. Foreign body reaction is not yet fully understood and the pathogenesis of CC is still unclear, but it is believed to...

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An Innovative Procedure for the Treatment of Primary and Recurrent Capsular Contracture (CC) Following Breast Augmentation

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**Abstract**

**Background:** Capsular contracture (CC) is the most frequently reported complication following alloplastic breast augmentation. At present, none of the available preventive measures are effective, and various treatment modalities have been advocated. Reduction of the inflammatory process is critical for successful treatment. Late intracapsular glucocorticosteroid (GC) injections have been somewhat effective for the treatment, but the fine balance between the effectiveness of therapeutic GC dosages and their potential serious side effects is of utmost importance.

**Objectives:** The authors investigate whether instillation of a rapid-acting water-soluble GC in the implant pocket during the early proliferative phase of wound healing is more effective than delayed instillation during the remodeling phase.

**Methods:** Between 2003 and 2009, 33 consecutive patients presenting with CC (Baker grades III and IV) were managed by capsulectomy with implant replacement and corticosteroid therapy immediately as well as 2 to 3 days later through an indwelling catheter left in place for that period. This delayed but early administration is a novel technique for GC injection.

**Results:** Complete correction of the contracture with no recurrence was achieved in all patients with a follow-up range of 2 to 10 years.

**Conclusions:** This GC administration technique avoids the potential complications of long-term, slow corticosteroid release. It has a targeted anti-inflammatory effect, probably at a critical stage of the healing process, and could effectively prevent CC following alloplastic breast augmentation.

**Level of Evidence:** 4

**Keywords**

breast augmentation, capsular contracture, intracapsular glucocorticoid, recurrent capsular contracture

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be mostly multifactorial, influenced by patient age, type of implant (shape, surface, and material), silicone leakage, individual susceptibility and propensity for hypertrophic scarring, surgical technique, surgical incision site, and occurrence of postoperative complications such as bleeding and hematoma formation.\textsuperscript{1,4,7,11-14} Recent evidence suggests that subclinical infection and the presence of a bacterial biofilm on the surface of the implant could be a likely major contributor to CC.\textsuperscript{15-17} Interestingly, a high bacterial colonization rate of 61\% to 66.7\% has been detected in association with grade III and IV contractures, suggesting that bacterial stimuli may accelerate the process of inflammation and fibrosis.\textsuperscript{18} In addition to skin bacteria that might contact the breast implant during insertion, endogenous breast flora expressed through the nipple represent a potential source for colonization of the implant surface, producing a nidus for subclinical infection and biofilm formation.\textsuperscript{14} Suggested procedures to minimize bacterial contamination include preoperative washings with Betadine solution before hospital admission and before transfer to the operating theater and prophylactic antibiotic administration.

Early CC (within the first 2 months after implantation) is likely the result of poor hemostasis, unsterile implant insertion, or traumatic surgical technique. Late contractures that occur months to years later are most probably a result of biofilm activation, bacteremic seeding, or silicone exposure due to implant rupture.\textsuperscript{7} In both situations, and regardless of the implant type, capsule formation is part of the normal healing process and is the effect of a low-grade chronic proliferative inflammation pattern manifesting initially by the healing process and is the effect of a low-grade chronic proliferative inflammation pattern manifesting initially by the activation and production of extracellular matrix (ECM) proteins and a synovial-like metaplasia of proliferating mesenchymal cells, culminating in a layer of dense hyaline collagenous fibrous connective tissue normally not thicker than 1 mm.\textsuperscript{2,5,11,15,19-21} The degree of foreign body reaction and local inflammatory response has a positive linear correlation with the severity of CC and is independent of the implant surface.\textsuperscript{11} Capsular contracture appears to be a manifestation of abnormal scarring, with an abundance of inflammatory cells, collagen, and myofibroblasts.\textsuperscript{7} In the most contracted capsules, there is increased capsule thickness with a spindle-like, smooth muscle actin–positive layer.\textsuperscript{7} Although the role of textured-surface implants in reducing the rate of CC remains controversial,\textsuperscript{4} attention must be drawn to the relatively new phenomena of double capsule and late seroma only seen with Biocell textured-surface implants (Allergan, Inc, Irvine, California).\textsuperscript{1,22}

Reported recommendations to prevent CC include precise hemostasis, sterile surgical technique, placement of the implant beneath the pectoralis major muscle, use of saline implants, use of nipple shields, use of antibiotic-impregnated mesh, and breast massage.\textsuperscript{1,14,17} At present, none of the available preventive measures are effective.\textsuperscript{1,12} As for treatment, many modalities have been advocated. Massages or tight immobilization have been described for the treatment of CC in patients with mild symptoms.\textsuperscript{15} In an attempt to break the capsular scar tissue, closed capsulotomy by manual compression of the breast was performed in the past, but this is a traumatic maneuver associated with increased risk of hematoma, implant rupture, and implant “pseudoherniation.” It has long since been abandoned.\textsuperscript{23} Surgical open capsulotomy or capsulectomy and implant removal with replacement of a second implant in a retropectoral instead of a retroglandular pocket is indicated only in patients with symptomatic highest degrees of contracture (Baker grades III and IV). The use of textured implants rather than smooth implants has also been suggested.\textsuperscript{11-13,23-27} Since more extensive surgical dissection is required for capsulectomy and these procedures carry increased associated risks, it is generally recommended that fibrous capsules should be left in place. This issue, however, is far from being resolved and is still subject for debate.\textsuperscript{23}

It is unanimously agreed that reduction of the inflammatory process is critical for the successful treatment of CC.\textsuperscript{23} Oral administration of leukotriene receptor antagonist has been recently described to be effective in the treatment of CC by dampening inflammation, but evidence provided is very tenuous at best.\textsuperscript{1,8,23} Flector Tissugel (Laboratoires Génévrier, Sophia Antipolis, France), the only locally active nonsteroidal anti-inflammatory drug available in the form of patches, has been recently reported to downstage CC from Baker grade II or III to Baker grade I when applied no later than 3 months after CC onset.\textsuperscript{23} Late corticosteroid injections have also been demonstrated to be somewhat effective for the treatment of this condition.\textsuperscript{27,29,30} To prevent contracture recurrence, systemic or intracapsular delivery of steroids, antineoplastics, antileukotriene, anti–transforming growth factor $\alpha$, or antibiotics has been described.\textsuperscript{29,31-33}

Glucocorticoids (GC), especially methylprednisolone, are very effective anti-inflammatory drugs. Their pharmacologic effects are based on a wide range of mechanisms of action, and they are being used in numerous inflammatory conditions.\textsuperscript{34-36} At a lower concentration, the effects of GC are mediated mainly by the classic GC receptor. At higher concentrations, additional nongenomic mechanisms—such as other membrane receptors and activation of secondary messenger systems—may be implicated.\textsuperscript{35,36} Severe negative effects of GC on the synthesis of collagen fibers and growth factor, and subsequently on wound healing, have been reported.\textsuperscript{2,37-40} The fine balance between the effectiveness of therapeutic GC dosages and their potential serious side effects is of utmost importance. It depends on the route of administration, plasmatic concentration, tissue distribution, and rate of excretion.\textsuperscript{2} We describe a novel modality of GC administration for the treatment and prevention of CC recurrence.

**METHODS**

Between 2003 and 2009, 33 consecutive patients presenting with CC (Baker grades III and IV) in 1 or both breasts were managed with capsulectomy with implant replacement and corticosteroid therapy immediately as well as 2 to 3 days later through an indwelling catheter left in place for
that period. All patients were healthy, and none were diabetic, had any surgical contraindication, or had been given any medication that could potentially affect the rate of CC formation, such as zafirlukast. Details of patient age, weight, previous surgical procedures (including technique and implant details), and CC status (primary or recurrent with grade of contracture and type and size of replacement implants) are shown in Table 1. Age range of the patients was 23 to 48 years. Eighteen patients had primary contractures; 4 of those had undergone 2 previous attempts at correction to no avail. Fifteen patients had recurrent contractures. Eight patients had undergone breast augmentation 1 year earlier, 7 patients had their implants for 2 years, and 3 for 4 years. The size range of the implants was 200 to 350 cc. Two patients had saline inflatable implants. Twelve patients had smooth, silicone gel implants, and 19 had textured implants. There were 4 patients with Ceréform (Ceréplas, France) breast implants, 5 with Sebbin (Groupe Sebbin SAS, Boissy-l’Aillerie, France), 3 with Perthese (Perouse Plastie, Nanterre, France), 7 with Mentor (Santa Barbara, California), 9 with PIP (La Seyne-sur-Mer, France), and 5 with Arion (Mougins Sophia-Antipolis, France). In 26 patients, implants were originally placed in a subglandular pocket. In 7 patients, the pocket was submuscular. All patients underwent bilateral implant replacement (66 breasts) and signed the standard informed consent form provided by the French Society of Aesthetic and Plastic Surgery, to which a specific paragraph describing the procedure being proposed with potential risks of recurrent CC and complications of treatment with antibiotics and corticosteroids was added.

**Surgical Technique**

Surgical incisions were dictated by the previous surgical approach (periareolar, inframammary, or vertical) whenever initial surgery was associated with mastopexy. After removing the implant, a complete capsulectomy was usually performed unless the posterior leaflet of a subpectoral capsule was strongly adherent to the chest wall. In all cases, partial retention of the capsule in a way that supported the new implant infrafibrously and prevented its displacement, as described by some, was not judged to be indicated. Moreover, no attempt was made to create a new pocket in a different plane. The pocket was infiltrated with 40 mg of gentamycin (1 ampoule) for 20 to 30 minutes, after which saline irrigation was performed as needed. A new implant was then inserted. A suction drain and an 18-gauge catheter usually used for epidural anesthesia were placed and fixed in position, and the surgical incision was closed. Special care was taken to place the tip of the instillation catheter deeply, mostly in a retroprosthetic position. The suction drain was then clamped and a first instillation of corticosteroid (methylprednisolone hemisuccinate: Solu-Medrol; Hospira, Inc, Lake Forest, Illinois) was performed: 40 mg of Solu-Medrol (1 vial) was diluted in 10 mL of injectable water, and 5 mL was then instilled in each breast through the catheter. The suction drain was unclamped 2 to 3 hours later, after the patient was transferred from the recovery room to her room (Figures 1 and 2).

The patients were discharged home the following day, with suction drains and catheters in place. Suction drainage was continued for 2 to 3 days until cessation of serosanguineous drainage, at which time the suction drain was removed. Delayed, same-dose second corticosteroid instillation was then performed with the catheter still in place. The catheter was then removed. This procedure was simple and quick, not necessitating any special expertise. All patients received perioperative antibiotics (cefotaxime 1 g/d) for 3 days. No nipple shields were used; instead, patients were instructed to wash their breasts prior to surgery. Gentle manual breast massage was performed only after the second instillation of the long-acting steroid and removal of the catheters for better drug distribution in the pocket.

**RESULTS**

In all patients, bilateral implant replacement was performed; a different type of implant was inserted in 26 cases. Of the newly implanted devices, 29 were textured and 4 were smooth. After 2005, all replaced implants were textured. Complete correction of the contracture with no recurrence was achieved in all patients, and follow-up ranged from 2 to 10 years (Figures 3 and 4). Each patient was seen 15 days following surgery and then checked at 1 month and every 6 months thereafter. A single patient (patient 23 in Table 1) had early subcutaneous atrophy in the lower lateral breast quadrant, with bluish skin translucency. This could have been due to pooling of the injected drug with uneven distribution. Fortunately, the condition resolved spontaneously within 6 months (Figure 5). Partial retention of the capsule in some patients who underwent previous subpectoral implant placement did not seem to have affected the final result.

**DISCUSSION**

Despite extensive discussion in the literature about preventive measures, CC remains an unwelcome and sometimes late complication of alloplastic breast augmentation.9 So far, there is no general agreement about the exact pathophysiology of CC. Naturally, conflicting opinions about its prevention and treatment prevail, without any agreement on a generally accepted modality of treatment.5,9 When it occurs, surgical revision with capsulotomy or capsulectomy and replacement of the implant is still the mainstay of treatment. Unfortunately, efficacy of this therapeutic regimen is limited because of a high recurrence rate.41,42 The incidence of recurrent contracture after capsulotomy alone is 54%.27

It is evident that preventive measures to reduce bacterial contamination have a profound effect on CC incidence
Table 1. Details of Patients’ Age, Weight, Previous Surgical Techniques and Implants, CC Primary or Recurrent With Grade of Contractures, and Type and Size of Replacement Implants.

| Year | Age, y | Weight, kg | Previous Implant and Surgical Technique | CC Duration or Recurrence | Baker Grade | New Implant |
|------|--------|------------|----------------------------------------|---------------------------|-------------|-------------|
| 1    | 2003   | 45         | PIP Saline SG, IPA                     | P, 3 y                    | IV          | Mentor Smooth 250 cc |
| 2    | 33     | 57         | PIP Saline SG, IPA                     | P, 3 y                    | IV          | Mentor Smooth 220 cc |
| 3    | 23     | 59         | PIP Smooth SG, IPA                     | P, 1 y                    | III         | PIP Textured 220 cc |
| 4    | 42     | 62         | PIP Smooth SG, IPA                     | P, 1 y                    | IV          | PIP Textured 320 cc |
| 5    | 46     | 56         | Mentor Smooth SM, IPA                  | R, 2nd                    | IV          | Sebbin Textured 350 cc |
| 6    | 37     | 55         | Mentor Smooth SG, IM                   | R, 2nd                    | IV          | Arion Textured 250 cc |
| 7    | 36     | 52         | Sebbin Smooth SM, IPA                  | R, 2nd                    | IV          | Sebbin Textured 350 cc |
| 8    | 45     | 58         | PIP Smooth SG, IM                      | R, 2nd                    | IV          | Arion Textured 320 cc |
| 9    | 2004   | 48         | Sebbin Smooth SM, mastopexy            | P, 4 y                    | III         | Mentor Smooth 280 cc |
| 10   | 33     | 50         | Mentor Smooth SG, IPA                  | P, 2 y                    | III         | Mentor Smooth 300 cc |
| 11   | 31     | 53         | PIP Smooth SG, mastopexy               | P, 2 y                    | IV          | PIP Textured 250 cc |
| 12   | 46     | 52         | Mentor Smooth SG, mastopexy            | P, 4 y                    | IV          | PIP Textured 220 cc |
| 13   | 2005   | 34         | PIP Smooth SG, mastopexy               | P, 1 y                    | III         | PIP Textured 310 cc |
| 14   | 38     | 54         | Sebbin Smooth SM, IPA                  | P, 4 y                    | III         | PIP Textured 350 cc |
| 15   | 42     | 57         | Mentor Textured SG, IPA                | P, 2 y                    | III         | Mentor Textured 280 cc |
| 16   | 26     | 58         | Mentor Textured SG, IPA                | P, 1 y                    | III         | Mentor Textured 300 cc |
| 17   | 41     | 60         | Arion Textured SG, IM                  | R, 3rd                    | IV          | Mentor Textured 310 cc |
| 18   | 39     | 61         | Arion Textured SM, mastopexy           | R, 3rd                    | IV          | Sebbin Textured 280 cc |
| 19   | 2006   | 27         | Mentor Textured SG, IPA                | P, 1 y                    | III         | PIP Textured 300 cc |
| 20   | 32     | 55         | PIP Textured SG, IPA                   | P, 1 y                    | III         | PIP Textured 300 cc |
| 21   | 36     | 57         | PIP Textured SG, IPA                   | R, 3rd                    | IV          | Arion Textured 220 cc |
| 22   | 38     | 56         | Perthese Textured SG, mastopexy        | R, 3rd                    | IV          | Mentor Textured 250 cc |

(continued)
and severity. Minimizing contamination with Betadine irrigation and prophylactic antibiotics has been demonstrated to reduce CC incidence.\textsuperscript{9,43-46} Several medications have also been proposed to modulate the various stages of healing responsible for CC formation.\textsuperscript{2} Mesna,\textsuperscript{47} mitomycin C,\textsuperscript{33,48} pirfenidone,\textsuperscript{49} halofuginone,\textsuperscript{50} and zafirlukast (a leukotriene receptor antagonist)\textsuperscript{8,32,51,52} have all been shown to reduce capsule thickness, fibroblast cell proliferation, and collagen deposition.\textsuperscript{9,33} With the exception of zafirlukast, approved for the treatment of asthma, these drugs are not common in clinical practice. Anecdotal evidence about the clinical efficacy of oral zafirlukast on CC has not been confirmed. In view of its potentially serious liver toxicity that may outweigh the benefits as a treatment modality for CC, its use is still restricted to severe and recurrent contractures.\textsuperscript{3,9,23,42}

The effects of corticosteroids on all cells involved in the postinjury inflammatory processes and wound healing have been well demonstrated. Corticosteroids suppress inflammatory, endothelial, and epithelial cells as well as fibroblasts; decrease deposition of ground substance and collagen; and reduce contraction.\textsuperscript{9} However, their role in the treatment and prevention of CC is controversial and not completely understood. Reported evidence of corticosteroid effectiveness remains sparse and contradictory\textsuperscript{9} despite documented evidence that postoperative injection of corticosteroids reduces the risk and delays the onset of recurrent contracture in high-risk patients and reduces capsular thickness (and discomfort) in patients with Baker grade IV CC.\textsuperscript{27,29,30,53}

![Figure 1. Catheter set for epidural anesthesia (18-gauge).](image)
Interestingly, a reduced incidence of CC has been reported when corticosteroids are instilled in saline inflatable breast implants, without causing any wound complications attributable to the steroids. However, when long-acting corticosteroids are instilled in the pocket intraoperatively, their efficacy in preventing CC is none, as reported

Figure 2. (A) The suction drain is indicated by an arrow, and the catheter is shown in place at the end of the surgical procedure. (B) The suction drain is clamped and Solu-Medrol (Hospira, Inc, Lake Forest, Illinois) is injected (40 mg/2 mL).

Figure 3. (A) This 32-year-old woman presented with Baker grade IV capsular contracture 1 year after subglandular breast augmentation. (B, C) Two years after capsulectomy and corticosteroid treatment immediately and, through an indwelling catheter, 2 to 3 days following replacement with a 300-cc PIP (La Seyne-sur-Mer, France) implant.
by Caffee and Rotator.\textsuperscript{27,29,30} This is probably because the drug is rapidly eliminated by sponges and/or by early suction drainage, but when steroids are injected percutaneously into the capsule at 4 and 8 weeks postoperatively, they are reported to have a somewhat modest efficacy of less than 50%.

The method for intracapsular percutaneous injection of long-acting corticosteroid as described by Caffee\textsuperscript{27,30} (20 mg triamcinolone acetonide diluted in 10 mL of saline) or with ultrasound guidance (40 mg triamcinolone acetonide diluted in 10 mL of saline)\textsuperscript{3} is labor intensive and requires certain expertise. Moreover, postinjection recommendations for frequent massages (6 times per day for 2 weeks) and for the patient to hang inverted while massaging her breast to prevent drug pooling in the lower breast quadrant\textsuperscript{30} are certainly impractical. It is also not without major complications such as prosthetic puncture, bleeding, or major soft tissue atrophy due either to large corticosteroid doses or, most probably, to poor distribution in the peri-implant space. Implant distortion due to incomplete capsulotomy is also a potential complication.\textsuperscript{30}

Figure 4. (A) This 46-year-old woman presented with second recurrence of left Baker grade IV capsular contracture. (B, C) Three years after capsulectomy and corticosteroid treatment immediately and, through an indwelling catheter, 2 to 3 days following replacement with a 220-cc textured Perthese (Perouse Plastie, Nanterre, France) implant.
Unfortunately, the corticosteroid doses required for the treatment or prevention of CC have resulted in severe adverse effects. Their negative effects on collagen fiber synthesis and growth factor, and subsequently on wound healing, have impaired their current clinical use. Moreover, despite initial excellent results observed with steroid-containing inflatable prostheses, at least over a short period, significant dose-related complications—including prosthesis ptosis, atrophy, thinning of overlying breast tissue, impending extrusion, and even CC—have been observed many years after implantation. It is worth mentioning, however, that the rapid-acting, water-soluble methylprednisolone sodium succinate we have used immediately and 3 days later (instead of the previously described injection of longer-acting, practically insoluble triamcinolone acetonide 6-8 weeks after surgery) may have affected GC distribution and diffusion in the pocket. In fact, with the mostly used textured implants, strong adhesions form between the capsule and the implant membrane that make uniform distribution of the injected GC unlikely, invariably leading to GC pooling at the injection site and an increased complication rate. Interestingly, in the 1 patient who developed early subcutaneous atrophy in our clinical series, the instillation catheter apparently was misplaced superficially, which prevented proper GC distribution within the pocket.

More recently, it was suggested that glucocorticoids implanted in local liposome depots yield a 10-fold reduction in dosage with more efficient continuous release and decreased adverse systemic effects. Local deposition of liposomal prednisolone effectively decreases fibrous capsule thickness around textured silicone breast implants, with no apparent late complications like the ones observed with steroid-containing inflatable prostheses.

Poor results observed with delayed percutaneous intracapsular steroid injection are probably due to the fact that the injection is performed very late to treat an already established contracture or not early enough to modulate a certain critical phase of the wound-healing process. The technique we are describing ensures immediate effect of the long-acting corticosteroid in the fresh surgical pocket by clamping the suction drain for a few hours postoperatively, supplemented with a more sustained action by an additional injection 3 days later through an initially positioned catheter. It seems that early corticosteroid treatment is critical in reducing fibroproliferation and CC formation. As to the most appropriate formulation and dosage of corticosteroid for optimal benefit with minimal side effects, available evidence is still very limited. The selected dose of Solu-Medrol (20 mg/5 mL/breast) was arbitrarily selected based on previously published studies. It is worth mentioning, though, that methylprednisolone sodium succinate’s time to peak effect is 4 to 8 days for intramuscular injection, while triamcinolone acetonide has a sustained duration of action over a period of several weeks. The different pharmacokinetics, in addition to the different timing of injection, may explain the improved results and fewer side effects we have observed compared with what has been previously reported.

The concern that some may have about losing a lifetime warranty that few manufacturers (not all) offer in case of implant rupture, on the assumption that steroids might weaken the implant, is unjustified, because implants removed due to CC, rippling, loss of product integrity caused by cosmetic revision surgery, by open or closed capsulotomy, or as a result of complications other than deflation/rupture are usually not covered by the warranty. A treatment modality that would prevent CC would be greatly welcomed in that context.

The study is limited by being noncomparative (lacking a control group) and not being randomized. Even though it seems that steroid instillation much earlier than 4 to 8 weeks could be valuable, a key question remains: we must determine the exact stage of wound healing beyond which corticosteroids become maximally effective in preventing CC. Another issue is the determination of the minimal effective dosage.
CONCLUSIONS

From the available data, it is obvious that corticosteroids placed intraoperatively in the pocket are not effective for the prevention of CC. Before wound closure, GC may be mostly removed by sponges or eliminated early by suction drainage. Only late administration, either by ultrasound-guided injection or by slow-release liposome depots, has been demonstrated to have value. These 2 modalities, however, necessitate sophisticated resources and certainly require expertise that is not easily attainable.

The clinical efficacy of the simple and safe technique we are proposing is in line with what has already been described. Our decision to clamp the suction drain for a few hours after the first GC instillation and then perform a second delayed instillation after cessation of drainage and removal of the suction drains ensured that proper GC dosage was maintained in the implant pocket to exert its effects. Moreover, this technique avoids potential complications of long-term slow corticosteroid release by favoring a targeted effect at a critical stage of the healing process. Excellent results have been observed so far with this technique. It is currently applied in our practice prophylactically to all alloplastic breast augmentations.

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