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REVIEW

Infections After Plastic Procedures: Incidences, Etiologies, Risk Factors, and Antibiotic Prophylaxis

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Abstract
Background Through a review of the English literature, this study aimed to assess the incidence, etiology, risk factors, and preventive measures for postoperative infections occurring after plastic surgery operations.
Methods All studies describing the occurrence of infections after plastic surgery procedures including case reports, prospective trials, and retrospective series were selected.
Results The 85 articles analyzed showed that incidences differ greatly among procedures and seem to be influenced by different and specific risk factors for each operation. Etiologic agents are primarily bacteria, although mycobacteria, virus, and fungi also have been described. No agreement exists on the use of antibiotic prophylaxis, except for abdominoplasties, because few specific prospective trials are present in the literature.
Conclusions Infections remain an important problem in plastic surgery with different points that still need to be clarified. Hopefully, in the future prospective randomized trials will definitively address this issue in order to provide plastic surgeons with clear and unbiased guidelines on its prevention and management.

Keywords Abdominoplasty · Complications · Infections · Liposuction · Mammaplasty · Plastic surgery

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Postoperative infections are rare after plastic surgery procedures, but when present, seriously endanger the aesthetic outcome. Currently, no specific guidelines for perioperative prophylaxis exist, and according to the definition of "clean operations," this would not be necessary. However, most surgeons use antibiotics, based on personal convictions or experience, and the lack of specific prospective studies brings an additional degree of uncertainty. Furthermore, important risk factors have been investigated recently (i.e., smoking, obesity, the duration of surgery, and the amount of fat removed), and all could be used to stratify patients according to their risk for infections so that appropriate preventive measures could be dedicated only to those at high risk.

This study aimed to assess the incidence, etiology, risk factors, and preventive measures of postoperative infections occurring after plastic surgery operations through a review of the English literature.

Breast Augmentation

Infections are present in 0.001% to 7% of breast augmentations, with most series showing an incidence of less than 1% in aesthetic surgery [1–4]. Two-thirds of these infections develop within 1 month after the operation, but in the series of Brand [2], 13.3% of the patients manifested the infection 3 months after surgery, 8.3% after more than 6 months, and anecdotal cases even years or decades after surgery [3]. The occurrence of these infections is extremely rare (1:10,000 implants), but incidences can be even higher due to the length of the intervening time. Patients with late infections may not be seen by the surgeons who performed the implantation. The timing has not been influenced by the type of surgery, the implants adopted, or the route of

insertion [2], but some late infections have occurred after hematogenous metastatic spreads from other sites [2].

Bacteria are the most common etiologic agents of postoperative infections. Among them, *Staphylococcus aureus, S. epidermidis*, streptococci A and B, *Klebsiella pneumoniae, Bacillus*, and *Propionibacterium* are those more frequently isolated, whereas *Corynebacterium* species, *Propionibacterium acnes, Pseudomonas aeruginosa, Escherichia coli*, and *Enterobacteriacee* are less present [2,5–8]. Late infections have been caused by *S. epidermidis*, *S. aureus*, *Pseudomonas aeruginosa*, and *Enterobacteriacae* [2]. Sporadic cases of mycobacteria have been described (*Mycobacterium tubercolosis, M. fortuitum, M. chelonae*, and *M. thermoresistible*), and although rare, they should be suspected when common techniques fail to isolate a cause [2,8–12].

In the study of Cregg et al. [12], six surgeons reported six infections with *M. fortuitum*, and two surgeons reported infections with *M. tubercolosis*. However, the occurrence of mycobacteria infections could be even higher because another eight surgeons described 12 patients whose manifestations were similar to those with mycobacteria, but appropriate cultures were not prepared. Still another seven surgeons received cultures with positive results for "diphteroids" [12].

Finally, fungine infections attributable to *Candida albicans*, *Curvularia*, and *Aspergillus niger* have been reported. In these cases, normal intact silicone membranes are impermeable to fungi. Small punctured ports allow them to translocate into implants and reproduce in the saline environment [13–17].

Risk factors have not been carefully assessed in prospective studies (almost all are retrospective or case series), and available trials do not allow definitive assessment and quantification. Women with breast reconstruction have a 10-fold greater probability for the development of infection (24–53% in some series) than aesthetic patients [3]. This depends on the degree of preexisting tissue scarring and skin atrophy resulting from cancer surgery, radiation therapy, or chemotherapy, which causes postoperative tissue ischemia and delayed wound healing [2,3]. Furthermore, nodal dissection and immediate reconstruction increases the spillage of bacteria and augments the possibility of infections [3].

In aesthetic augmentations, smoking, obesity, and diabetes did not significantly increase the occurrence of infections, whereas corticosteroids, hematomas, pregnancy, preceding lactation, vigorous exercising, massages, and postsurgical traumas were significantly associated [2]. Additional simultaneous surgery was a consistent risk factor for postoperative infections.

In the study of Spears et al. [18], the complication rates were 17% for primary and 23% for secondary augmentation/mastopexy patients, and the respective revision rates were 8.7% and 16.6%. For reference, primary augmentations and secondary augmentations had respective complication rates of 1.7% and 21.6%, and a 1.7% revision rate, compared with 18.7% for secondary augmentations. Furthermore, in many cases the position of the implant can be inappropriate. As a result, the increased difficulty and occasionally the implant impart excessive tension, which can cause skin necrosis, nipple-areolar necrosis, or glandular necrosis with infections. In such cases, infection is secondary to local dermal ischemia [19].

Sporadic cases of implant infections after systemic spread from other organs have been reported (i.e., after bilateral pneumonia, hemorrhagic cystitis, severe stomatitis, dental surgery, sty) [2]. Implant texture type (smooth, textured, or polyurethane-coated) was associated with similar infection rates (respectively, 0.06%, 0.16%, and 0.12% for aesthetic augmentations and 0.6%, 0.4%, and 0.3% for reconstructions and expansions). Similar results also were found for implant types (saline vs silicone) [5,11]. The route of prosthesis insertion (areolar, transaxillary, or inframammary) and the type of placement (retropectoral vs subglandular) showed contrasting results, although in some studies, they did not seem to influence the occurrence of postoperative infections [2,5,11].

In the survey of Clegg et al. [12], the transaxillary approach was found to have the lowest incidence of infections (0.08%) compared with the inframammary (0.63%) or periareolar (0.67%) approach, and subglandular positioning was more risky than retropectoral positioning when the patient was taking steroid medications [12]. Furthermore, the same study showed that single-lumen prostheses had a lower incidence of infections (0.55%) than double-lumen (0.78%) or saline-filled prostheses (0.87%). Also, a significant difference was found between the procedure in which the prosthesis was rinsed or soaked in saline before insertion (0.56%) and that in which it was not (0.81%) [12].

An interesting theory links subclinical infections with capsular contractions. Cultures of periprosthetic fluids obtained from patients with early capsular contractures showed positive results for *S. epidermidis*, compared with only one in eight samples obtained from patients with minimal or no contracture ($p = 0.0006$), suggesting an influence of bacteria on the occurrence of capsular scarring and contraction. Scanning electron microscopy confirmed the presence of extensive biofilms on contracted capsules and implants [5,11].

There has been a long debate about the utility of perioperative antibiotic prophylaxis, and few studies have specifically addressed this issue. In a randomized study, the authors compared 60 patients who received a first-generation cephalosporin (short-term prophylaxis) with 132

patients who did not receive it for the occurrence of infections. All the patients had their prostheses washed with an antiseptic solution and their pockets irrigated before positioning. The authors concluded that there was no significant difference in the occurrence of postoperative infections between the two groups (0% for those who received antibiotics vs 0.7% for those who did not).

However, the study had a small sample for each group, considering the low incidences of postoperative infections reported in literature and the important differences between treatment groups. In fact, bacitracin solution (50,000/l) was used for 83% of the patients who received antibiotics and for 97.7% of those who did not. Also, in this study, 5% Betadine solution was used for 6.7% of patients who received antibiotics and for 7.5% of those who did not. Bilateral drains were placed in 25% of the patients who received antibiotics compared with 50% of the nonantibiotic group [1].

A second retrospective survey of 1,487 plastic surgeons and almost 40,000 breast augmentations found 254 infections (0.64%), providing statistically significant evidence for the value of antibiotic prophylaxis. The infection rate was 0.42% in the prophylaxis group compared with 0.87% in the group without antibiotics [12].

Once the infection is manifested, little is published about its effective management. In the literature, 8.3% of implants are salvaged [2]. A retrospective review identified seven different groups of patients based on the type of infection including mild infection, severe infection, threatened exposure without infection, threatened exposure with mild infection, threatened exposure with severe infection, actual exposure without clinical infection, and actual exposure with infection.

All the patients with mild infections were started immediately on oral antibiotics, and the patients with severe infections were started on parenteral antibiotics. Those who responded completely required no further treatment, whereas operative intervention was adopted for persistent infections or threatened or actual exposure. In these cases, implant removal, pocket curettage, partial or total capsulectomy, debridement, site change, placement of a new implant, or flap coverage was chosen according to the surgeon's experience and clinical judgment. After aggressive interventions, 77% of threatened implants with infection or threatened/actual exposure of prosthesis were salvaged, but the presence of severe infections adversely affected the salvage rate. Successful salvage was accomplished for 95% of patients without infection or with mild infection, whereas only 29% of those with severe infection were salvaged. Salvage was accomplished for 91% of devices with threatened or actual exposure but not with the complication of severe infection. No immediate salvage was attempted in five cases because of severe infection, nonresponding infection with gross purulence, marginal tissues, or lack of options for healthy tissue coverage. The author concluded that salvage attempts for periprosthetic infection and prosthesis exposure may be successful except in cases of overwhelming infection or deficient soft tissue coverage [20].

Breast Reduction

The incidence of postoperative infections for breast reductions is 1.1% to 22%, higher than for other types of breast procedures (see Breast Augmentations) [21–34]. Most of these infections appear in the early postoperative period, but a few cases of late manifestations have been described [21]. Cultures from wound exudates have shown that the etiologic agents are both gram-positive and gram-negative bacteria (*Staphylococcus aureus*, *S. epidermidis*, *Streptococcus pyogenes*, *Bacteroides fragilis*, and *Enterobacter cloacae*) [22,23].

Obesity (body mass index [BMI], > 30), large reductions ($\geq 1{,}000$ g per breast), duration of surgery (more than 2 h), steroids, and other immunosuppressive therapies are significant risk factors for infections [22,24–27]. Serletti et al. [24] found that obese patients experienced more infections than nonobese patients (11% vs 5.5%). Obese patients who experienced infections were equally divided between those who received antibiotics and those who did not. Thus antibiotics did not seem to affect the infection rate among obese patients.

In a retrospective study, O'Grady et al. [25] analyzed the influence of the amount of tissue resected on the occurrence of infections, with a cutoff of 1,000 g of tissue removed. All patients received preoperative prophylaxis. These authors found that patients undergoing larger resections experienced a significant increase in infections (28% vs 10.6%), delayed healing (14% vs 6%), and wound dehiscence (16% vs 7%). However, the groups analyzed were not homogeneous for BMI (37 for larger resections vs 29 for. small resections). For this reason, the increase in infections cannot be ascribed with certainty to the amount of weight resected, to BMI, or both. Furthermore, Serletti et al. [24] failed to find differences in infection occurrence between patients who had more than 600 g of tissue removed and those who had less than 600 g removed (7% vs 8%), although a significant increase in delayed healings was present with larger resections (38% vs 20%) [24].

Andenaes et al. [35] found that the infection rate nearly tripled in an operation lasting more than 120 min, compared with an operation with a duration of less than 1 h. They concluded that the duration of surgery was an independent risk factor.

Similar results also were found by Kompatscher et al. [22]. However the peculiar incisions adopted for breast reductions/mastopexies, not used in other cosmetic operations, probably are an important factor influencing the occurrence of infections. Numerous authors have found an overall wound complication rate of 45% to 53% for T-shaped reductions [23,29], and infections were present in up to 22% of these patients [23,29–34].

For vertical breast reductions, a considerable number of studies adopted the Lejour technique, and apart from Lejour's personal experience, which involved very low complication rates (12%) [32], the wound complication rates are strikingly similar to those obtained with the T-shaped incision (37–40%) [36,37]. The concept of an intrinsic risk conferred by the T-inverted incision also is sustained by the occurrence of major flap necrosis in different operations that adopted the same incision [38].

A 1984 survey of prophylactic antibiotics used among plastic surgeons showed that 32% always prescribed prophylactic antibiotics for reduction mammaplasty [39]. The findings showed that 12% often used antibiotics for this operation, 17% seldom used prophylactic antibiotics, and 39% never prescribed them. The results of this survey compared with those of a similar study 10 years earlier showed a significant increase in the use of prophylactic antibiotics by plastic surgeons. In 1974, only 17% stated that they always prescribed prophylactic antibiotics for reduction mammaplasty, whereas 51% responded that they never used them for this procedure [39].

In recent years, a few studies have investigated the influence of perioperative antibiotic prophylaxis on infection occurrence, all of them retrospective and with contrasting results. Kompatscher et al. [22] analyzed their series of patients in a large retrospective study and divided those who received single-shot antibiotics prophylaxis (cefuroxime) from those that were given no antibiotics. They found no significant difference in the postoperative occurrence of infections between the groups (3.9% vs 3.7%).

A second retrospective study, conducted by Serletti et al. [24], compared 47 patients who received prophylaxis with 59 patients who did not. Prophylaxis was conducted using a first-generation cephalosporin (vancomycin if allergies were referred). The authors found that antibiotics did not reduce the incidence of infections in breast reductions (8.5% for patients with antibiotics vs 6.5% for the control group), even among high-risk patients (old age, obesity, smoking groups) [24].

Similar results were reported by Baran et al. [40] in their retrospective study, which found that infections after reduction mammaplasties were not affected by the preoperative use of antibiotics. However, these authors found that patients who received prophylaxis manifested infections later than those that did not (after 11 vs 5 days).

In contrast, Platt et al. [28] reported an odds ratio of 1.69 for infections in patients who did not receive treatment (95% confidence interval, 0.58–4.93) compared with. those who received it. Unfortunately, these authors combined herniorrhaphies, mastectomies, lumpectomies, excisional breast biopsies, reduction mammaplasties, and axillary node dissections for breast cancer, operations that have a nonhomogeneous risk for SSI. The study also combined results from a plethora of AMP agents, including clindamycin and vancocin. Of the 233 patients (7%) who underwent a breast reduction, 85% obtained antibiotic prophylaxis. In this study population, the rate of infections from breast reductions was 3 of 188 (1.6%) for the group with antibacterials and 2 of 29 (6.9%) for those with no antibacterials, figures that unfortunately do not permit a meaningful statistical analysis [28].

Finally, O'Grady et al. [25] investigated the prolonged postoperative use of antibiotics in a retrospective study. All the patients received preoperative antibiotics (cephalexin or cephalexin; clindamycin for penicillin-allergic patients) before the skin incision and postoperative antibiotics for 24 h. However, the one group received a prolonged antibiotics course (5 days after surgery), whereas the other group received antibiotics only during the first day after surgery. The rates of infection appeared to be lower among the patients who received an additional 5 days of antibiotics after surgery (7% vs 19%) [25].

Abdominoplasty

Abdominoplasty is one of the most frequently performed and standardized aesthetic procedures. However the incidence of infections reported in the literature is variable, ranging from 0.2% to 32.6% of patients in large series [38,41,42].

Smoking generally increases the risk of complications in plastic surgery, resulting in poor aesthetic outcomes [43–48]. In abdominoplasty, it increases the incidence of wound complications (47.9% of smokers vs 14.8% of nonsmokers) and nonaesthetic scars [41,43]. Numerous processes are involved in this phenomenon. The principal components of tobacco smoke (nicotine, nitric oxide, and carbon monoxide) influence wound healing in peripheral tissues [49–51]; blood flow to the flap's distal portion, which has the highest sympathetic innervation and is particularly sensitive to the vasoconstrictive effects of smoking [52,53]; and an increase in carboxyhemoglobin level and platelet adhesiveness with microthrombi formation, leading to a reduced ability to deliver oxygen [54,55]. Finally, the increased serum levels of fibrinogen and hemoglobin together with decreased fibrinolytic activity

and a direct endothelial injury in smokers [56,57] contribute a reduced local circulation.

All these effects are enhanced by the surgical technique that, creating a cutaneous and subcutaneous flap, requires perfect vascularization for the wound repair process due to the lack of perforating vessels from the abdominal muscles. Reports of major flap necrosis after abdominoplasty support this idea [38].

Sex, BMI, and percentage of body fat also are important risk factors for the occurrence of infections after abdominoplasties [41,58–63]. Van Uchelen et al. [58] found a higher prevalence of wound complications in males than in females (64.3% vs 15.3%), However, their series was not homogeneous, and the phenomenon was explained by the higher presence, in males, of other important risk factors for infections (smoke, diabetes, high BMI). A significant difference for infection occurrences was found between obese and nonobese patients (80% vs 32%) [62], and a trend of association was found between patients with high and those with low amounts of body fat content (measured with the bioelectric impedance analysis): 45.2% incidence of infection for fat mass exceeding 35% compared with 34.8% for fat mass less than 35% [63].

The utility of perioperative antibiotic prophylaxis in abdominoplasty was investigated by Sevin et al. [64]. A prospective study of 207 patients was planned, and three study groups were formed according to the administration of antibiotics: no antibiotics (group 1, n = 69), preoperative antibiotics only (group 2, n = 69), and both preoperative and postoperative antibiotics (group 3, n = 69). Bacterial growth in the intraoperative bacterial culture was shown by 20 patients, and there was a significant difference in the incidence of infection between groups 1 (9/69, 13%) and 2 (3/69, 4%), between groups 1 (9/69, 13%) and 3 (6/69, 9%), but not between groups 2 (3/69, 4%) and 3 (6/69, 9%). For this reason, the authors concluded that a single preoperative dose of intravenous antibiotic was useful and sufficient to prevent postoperative infection [64].

Liposuction

During the past decade, liposuction was the most common aesthetic procedure performed, and important technological advances from the original technique have been described [65–67]. Lázaro Cárdenas et al. [68] reported an incidence of 0.09%, with only one infection in 1,047 patients. This manifested on the 12th postoperative day, 6 days after drain removal, in a large lumbar area after flank lipoaspiration and abdominoplasty for abdominal contouring [68]. However, in 2004, a retrospective study of eight single-stage total body-lifts after postbariatric weight loss depicted an incidence of 25% [69].

Finally, a few cases of severe necrotiziing fascitis were described [70–77]. In one of these cases, 4 days after tumescent liposuction of the patient's thoracic roll/flanks/hips, abdomen, medial thighs, and knees, the woman presented with weakness, fever to 40°C, and a red, painful nodule on her left flank. All port sites were diffusely erythematous, indurated, and surrounded by patches or ecchymosis. After resuscitation, the women received two surgical explorations of the wound, but despite the aggressive treatment, the infection spread all over the abdomen, with a circumferential full-thickness skin and subcutaneous fascia loss from her costal margins distally to her suprapubic region and medial thighs [70].

In another case, during the operative procedure for abdominal wall debridement, extensive necrosis of abdominal wall fascia with leakage of bilious fluid from defects in the rectus sheath was found. Subsequent peritoneal cavity exploration showed two perforations in the mid ileum with gross peritoneal cavity contamination [73].

Bacteria are the most frequent etiologic agents of postoperative infections after liposuction and, among these, group A streptococci, *Streptococcus pyogenes*, and synergistic infections (anaerobic and facultative anaerobic bacteria) are by far the most frequently isolated [71,72]. However, sporadic cases of infections due to *Mycobacteria* (*Mycobacterium chelonae*, *M. fortuitum*, and *M. abscessus*) outbreaks also have been described [78–82]. Soft tissue infection caused by *M. chelonae* has manifested initially as slightly tender nodules with scant drainage and minimal surrounding cellulites, whereas systemic manifestations were absent or minimal (low fever) [78,80]. Lesions have occurred in areas of liposuction or contiguous areas, usually not at incision sites, and ranged in diameter from 0.5 to 7 cm. In most cases, patients had multiple lesions (mean diameter, 15–20 cm) with pink, red, or purple nonpruritic and not painful subcutaneous nodules. The indolent course, together with a low index of suspicion and failure to request or perform the appropriate diagnostic tests (e.g., acid-fast staining), has rendered timely diagnosis difficult [78,80].

Infections also have been caused by *M. fortuitum* and *M. abscessus* [81]. Strains of *M. fortuitum* are resistant to many disinfectants, including 10% povidone-iodine, 2% aqueous formaldehyde, and 2% alkaline glutaraldehyde. The clinical features of surgical infections usually manifest several weeks to some months after the procedure. Patients predominantly exhibit local erythema, induration, microabscesses, and serous drainage. Fever, chills, and other manifestations of sepsis are infrequent. The absence of clinical response after the administration of antimicrobial agents against commonly invading bacteria and the sterility of routine cultures taken from the infected sites highlight

the need for special isolating microbiologic procedures. Surgical debridement and drainage of pus and collections together with antimycobacterial therapy usually are required. Wounds are left open and packed to prevent early closure of the skin, which can result in reaccumulation of pus and the appearance of new draining fistulas [81].

Few studies have tried to find possible risk factors for the occurrence of infections. Lázaro Cárdenas et al. [68] investigated the influence of the amount of fat aspirated as well as associated procedures on the occurrence of postoperative infections. In a retrospective study, they divided patients into four groups according to procedure as follows: small lipoaspirations (<5 l), large lipoaspirations (>5 l), lipoaspirations combined with abdominoplasties, and lipoaspirations combined with other surgical procedure. All patients received a first- or second-generation cephalosporin for prophylaxis 6 h before the procedure. Although a 21.7% incidence for minor complications (palpable and visible irregularities, seromas, cutaneous hyperpigmentation, overcorrection, cutaneous slough, and local infections) and 0.38% incidence for major complications (fat embolism syndrome, cutaneous necrosis and extended infections) were reported, no differences in occurrences were found among the groups [68]. For the *Mycobacterium chelonae*, the best predictors were the number of liposuction sites, the duration of the procedure, and weight (inversely associated with the risk of infection) [80].

Other Procedures

Although an initial study by Baker described an infection incidence of 1% [82], actually face-liftings have lower values, with an approximate range of 0% to 0.3% [83–85]. Even with the subperiosteal approach, in which bacteria can enter the entire undermined space through the unsutured stab wound in the incisor fossae, the incidence of infection is low (0.3%), probably due to the extensive vascularization of the thick facial flap, the lack of dead space, and the atraumatic technique that prevent infections [86].

One study reported an infection incidence of 4.9% (2/41). However, this study investigated restorative mid-face-liftings with hand-carved and expanded polytetrafluoroethylene orbital rim implants [87]. In this series, one patient experienced a draining fistula 15 months after the operation due to a recurrent squamous cell carcinoma. Treatment consisted of implant removal. A second patient underwent implantation after resection for neurofibromatosis, and 4 months later experienced an atypical mycobacterial infection that necessitated explantation of the implant [87]. However, this was not the only case in the literature of a mycobacterial infection after face-lifting. A 35-year-old, HIV-negative woman experienced a *Pseudomonas aeruginosa* infection that manifested with spontaneous ulceration and violaceous discoloration of the skin adjacent to the surgical wound. The infection persisted despite treatment. An additional bacteriologic examination showed *Mycobacterium smegmatis* to be a concomitant causative agent, and the appropriate combination therapy was instituted [88].

Regarding treatment, the only patient in the study of Jones and Grover [83] who experienced an infection was treated with a combination of surgical drainage and antibiotic therapy, and in the series of Ullmann and Levy [84], the patient with a β-hemolytic streptococcal infection healed spontaneously after appropriate antibiotic therapy.

Although rare, infections have been described also in highly vascularized areas such as the brow and the eyelids [89–90]. The incidence of infections after brow-liftings was lower than 0.1% in a survey of Elkwood et al. [89]. Even in this study, the low occurrences were attributable to the prominent vascularization and the lack of dead spaces.

Furthermore, a case report of an infection after blepharoplasty was described [90]. A healthy 59-year-old woman underwent outpatient bilateral upper and lower blepharoplasty with mid face-lifting. After 30 h, she experienced marked pain and edema of the left eyelids and face and a violaceous eyelid bulla, which heralded early necrotizing fasciitis. Culture of the serosanguinous exudates from the left eyelid showed group A beta-hemolytic *Streptococcus* organisms. The patient was treated with intravenous antibiotics, intravenous corticosteroids, hyperbaric oxygen therapy, and wound debridement. The infection resolved with mild cicatrization of the left upper eyelid [90].

Conclusions

Infections remain an important problem in plastic surgery, with issues that still need to be clarified. First, although incidences vary according to the operation performed and the presence of specific risk factors, they usually are extremely small, and it is difficult to demonstrate a statistical significance between groups without recruiting a large number of patients. In many cases, the reader often is left questioning whether the small differences noted by the author in the surgical literature are indeed statistically significant.

Second, the efficacy of perioperative antibiotic prophylaxis was effectively demonstrated with large prospective studies involving only abdominoplasties. Breast augmentations were investigated using a small prospective study (with important differences of treatment among groups) and with a large retrospective survey, whereas all the studies on breast reductions consisted of old national

surveys or retrospective analyses of personal series. All surveys, although they quoted statistics, were inherently flawed because the data often were generated by querying surgeons based on their memory.

In contrast, retrospective studies showed important differences among groups. For this reason, such studies failed to reach any significant conclusions for breast augmentations and reductions, nor did they provide any significant recommendations regarding the use of perioperative antibiotics.

The analysis of the published literature interestingly found that an important, although rare, quota of infections derives from unusual microorganisms (i.e., mycobacteria and fungi). Such infections can manifest after different operations and, because of their rarity, need to be suspected from the caregiver so they can be diagnosed correctly and managed with appropriate antimicrobial therapy.

Finally, the analysis of the literature found that the risk factors for the occurrence of infections seem to be specific for each operation, suggesting that the anatomy of the involved area and the surgical technique adopted may influence them. For example, this is the case for the peculiar "T" incision of breast reductions and for smoking during operations in which the vascularization is reduced (i.e., abdominoplasties for the flap preparation).

References

1. LeRoy J, Given KS (1991) Wound infection in breast augmentation: The role of prophylactic perioperative antibiotics. Aesth Plast Surg 15:303–305
2. Brand KG (1993) Infection of mammary prostheses: A survey and the question of prevention. Ann Plast Surg 30:289–295
3. Pittet B, Montandon D, Pittet D (2005) Infection in breast implants. Lancet Infect Dis 5:94–106
4. Araco A, Gravante G, Araco F, et al (2007) Infections of breast implants in aesthetic breast augmentations: A single-center review of 3,002 patients. Aesth Plast Surg 2007 (Epub ahead of print)
5. Pajkos A, Deva AK, Vickery K, et al (2003) Detection of subclinical infection in significant breast implant capsules. Plast Reconstr Surg 111:1605–1611
6. Bernardi C, Saccomanno F (1998) Late Klebsiella pneumoniae infection following breast augmentation: Case report. Aesth Plast Surg 22:222–224
7. Macadam SA, Mehling BM, Fanning A, et al (2007) Nontuberculous mycobacterial breast implant infections. Plast Reconstr Surg 119:337–344
8. Wolfe JM, Moore DF (1992) Isolation of Mycobacterium thermoresistible following augmentation mammaplasty. J Clin Microbiol 30:1036–1038
9. Heistein JB, Mangino JE, Ruberg RL, et al (2000) A prosthetic breast implant infected with Mycobacterium fortuitum. Ann Plast Surg 44:330–333
10. Juang YC, Wang LS, Chen CH, et al (1989) Mycobacterium fortuitum mastitis following augmentation mammaplasty: report of a case. Taiwan Yi Xue Hui Za Zhi 88:278–281
11. Handel N, Jensen A, Black Q, et al (1995) The fate of breast Implants: A critical analysis of complications and outcomes. Plast Reconstr Surg 96:1521–1533
12. Clegg HW, Bertagnoll P, Hightower AW, et al (1983) Mammaplasty associated mycobacteryal infection: A survey of plastic surgeons. Plast Reconstr Surg 72:165–169
13. Kainer MA, Keshavarz H, Jensen BJ, et al (2005) Saline-filled breast implant contamination with Curvularia species among women who underwent cosmetic breast augmentation. J Infect Dis 192:170–177
14. Saray A, Kilic D, Kaygusuz S, et al (2004) Fungal growth inside saline-filled implants and the role of injection ports in fungal translocation: In vitro study. Plast Reconstr Surg 114:1170–1178
15. Tian HH, Tan SM, Tay KH (2007) Delayed fungal infection following augmentation mammoplasty in an immunocompetent host. Singapore Med J 48:256–258
16. Niazi ZB, Salzberg CA, Montecalvo M (1996) Candida albicans infection of bilateral polyurethane-coated silicone gel breast implants. Ann Plast Surg 37:91–93
17. Truppman ES, Ellenby JD, Schwartz BM (1979) Fungi in and around implants after augmentation mammaplasty. Plast Reconstr Surg 64:804–806
18. Spear SL, Boehmler JH, Clemens MW (2006) Augmentation/mastopexy: A 3-year review of a single surgeon's practice. Plast Reconstr Surg 118(7 Suppl):136S–147S
19. Persoff MM (2003) Vertical mastopexy with expansion augmentation. Aesth Plast Surg 27:13–19
20. Spear SL, Howard MA, Boehmler JH, et al (2004) The infected or exposed breast implant: Management and treatment strategies. Plast Reconstr Surg 113:1634–1644
21. Harel M (1998) Late postoperative infection in a breast reduction case. Plast Reconstr Surg 102:1775
22. Kompatscher P, von Planta A, Spicher I, et al (2003) Comparison of the incidence and predicted risk of early surgical site infections after breast reduction. Aesth Plast Surg 27:308–314
23. Davis GM, Ringler SL, Short K, et al (1995) Reduction mammaplasty: Long-term efficacy, morbidity, and patient satisfaction. Plast Reconstr Surg 96:1106–1110
24. Serletti JM, Davenport MS, Herrera HR, et al (1994) Efficacy of prophylactic antibiotics in reduction mammoplasty. Ann Plast Surg 33:476–480
25. O'Grady KF, Thoma A, Dal Cin A (20005) A comparison of complication rates in large and small inferior pedicle reduction mammaplasty. Plast Reconstr Surg 115:736–742
26. Culver DH, Horan TC, Gaynes RP, et al (1991) Surgical wound infection rates by wound class, operative procedure, and patient risk index. Am J Med 91:152S–157S
27. Beer GM, Spicher I, Cierpka KA, et al (2004) Benefits and pitfalls of vertical scar breast reduction. Br J Plast Surg 57:12–19
28. Platt R, Zucker JR, Zaleznik DF, et al (1992) Prophylaxis against wound infection following herniorrhaphy or breast surgery. J Infect Dis 166:556–560
29. Dabbah A, Lehman JA, Parker MG, et al (1995) Reduction mammaplasty: An outcome analysis. Ann Plast Surg 35:337–341
30. Menke H, Eisenmann-Klein M, Olbrisch RR, et al (2001) Continuous quality management of breast hypertrophy by the German Association of Plastic Surgeons: A preliminary report. Ann Plast Surg 46:594–600
31. Asplund OA, Davies DM (1996) Vertical scar breast reduction with medial flap or glandular transposition of the nipple-areola. Br J Plast Surg 49:507–514
32. Lejour M (1999) Vertical mammaplasty: Early complications after 250 personal consecutive cases. Plast Reconstr Surg 104:764–770
33. Lassus C (1996) A 30-year experience with vertical mammaplasty. Plast Reconstr Surg 97:373–380

34. Hammond DC (1999) Short scar periareolar inferior pedicle reduction (SPAIR) mammaplasty. Plast Reconstr Surg 103:890–902
35. Andenaes K, Amland PF, Lingaas E, et al (1995) A prospective, randomized surveillance study of postoperative wound infections after plastic surgery: A study of incidence and surveillance methods. Plast Reconstr Surg 96:948–956
36. Pickford MA, Boorman JG (1993) Early experience with the Lejour vertical scar reduction mammaplasty. Br J Plast Surg 46:516–522
37. Berg A, Palmer B, Stark B (1995) Early experience with the Lejour vertical scar reduction mammaplasty technique. Eur J Plast Surg 18:214–218
38. Chaouat M, Levan P, Lalanne B, et al (2000) Abdominal dermolipectomies: Early postoperative complications and long-term unfavorable results. Plast Reconstr Surg 106:1614–1618
39. Krizek TJ, Gottlieb LJ, Koss N, et al (1985) The use of prophylactic antibacterials in plastic surgery: A 1980s update. Plast Reconstr Surg 76:953–963
40. Baran CN, Sensoz O, Ulusoy MG (1999) Prophylactic antibiotics in plastic and reconstructive surgery. Plast Reconstr Surg 103:1561–1566
41. Manassa EH, Hertl CH, Olbrisch RR (2003) Wound healing problems in smokers and nonsmokers after 132 abdominoplasties. Plast Reconstr Surg 111:2082–2087
42. Dillerud E (1990) Abdominoplasty combined with suction lipoplasty: A study of complications, revisions, and risk factors in 487 cases. Ann Plast Surg 25:333–338
43. Rogliani M, Labardi L, Silvi E, et al (2006) Smokers: Risks and complications in abdominal dermolipectomy. Aesth Plast Surg 30:422–424
44. Rees TD, Liverett DM, Guy CL (1984) The effect of cigarette smoking on skin flap survival in the face-lift patient. Plast Reconstr Surg 73:911–915
45. Krueger JK, Rohrich RJ (2001) Clearing the smoke: The scientific rationale for tobacco abstention with plastic surgery. Plast Reconstr Surg 108:1063–1073
46. Chan LK, Withey S, Butler PE (2006) Smoking and wound healing problems in reduction mammaplasty: Is the introduction of urine nicotine testing justified?. Ann Plast Surg 56:111–115
47. Kryger ZB, Fine NA, Mustoe TA (2004) The outcome of abdominoplasty performed under conscious sedation: Six-year experience in 153 consecutive cases. Plast Reconstr Surg 113:1807–1817
48. Spiegelman JI, Levine RH (2006) Abdominoplasty: A comparison of outpatient and inpatient procedures shows that it is a safe and effective procedure for outpatients in an office-based surgery clinic. Plast Reconstr Surg 118:517–522
49. Kaufman T, Eichenlaub EH, Levin M, et al (1984) Tobacco smoking: Impairment of experimental flap survival. Ann Plast Surg 13:468–472
50. Lawrence WT, Murphy RC, Robson MC, et al (1984) The detrimental effect of cigarette smoking on flap survival: An experimental study in the rat. Br J Plast Surg 37:216–219
51. Van Adrichem LN, Hoegen R, Hovius SE, et al (1996) The effect of cigarette smoking on the survival of free vascularized and pedicled epigastric flaps in the rat. Plast Reconstr Surg 97:86–96
52. Chang LD, Buncke G, Slezak S, et al (1996) Cigarette smoking, plastic surgery, and microsurgery. J Reconstr Microsurg 12: 467–474
53. Black CE, Huang N, Neligan PC, et al (2001) Effect of nicotine on vasoconstrictor and vasodilator responses in human skin vasculature. Am J Physiol Regul Integr Comp Physiol 281:R1097–R1104
54. Astrup P, Kjeldsen K (1974) Carbon monoxide, smoking, and atherosclerosis. Med Clin North Am 58:323–350
55. Birnstingl MA, Brinson K, Chakrabarti BK (1971) The effect of short-term exposure to carbon monoxide on platelet stickiness. Br J Surg 58:837–839
56. Dintenfass L (1975) Elevation of blood viscosity, aggregation of red cells, haematocrit values, and fibrinogen levels with cigarette smokers. Med J Aust 1:617–620
57. Meade TW, Chakrabarti R, Haines AP, et al (1979) Characteristics affecting fibrinolytic activity and plasma fibrinogen concentrations. Br Med J 1:153–156
58. Van Uchelen JH, Werker PM, Kon M (2001) Complications of abdominoplasty in 86 patients. Plast Reconstr Surg 107: 1869–1873
59. Mast BA (2005) Safety and efficacy of outpatient full abdominoplasty. Ann Plast Surg 54:256–259
60. El-Khatib HA, Bener A (2004) Abdominal dermolipectomy in an abdomen with preexisting scars: A different concept. Plast Reconstr Surg 114:992–997
61. Hensel JM, Lehman JA Jr, Tantri MP, et al (2001) An outcomes analysis and satisfaction survey of 199 consecutive abdominoplasties. Ann Plast Surg 46:357–363
62. Vastine VL, Morgan RF, Williams GS, et al (1999) Wound complications of abdominoplasty in obese patients. Ann Plast Surg 42:34–39
63. Lahiri A, Duff CG, Brown TL, et al (2006) Anthropometric measurements and their value in predicting complications following reduction mammaplasty and abdominoplasty. Ann Plast Surg 56:248–250
64. Sevin A, Senen D, Sevin K, et al (2007) Antibiotic use in abdominoplasty: Prospective analysis of 207 cases. J Plast Reconstr Aesthet Surg 60:379–382
65. Cook WR (1997) Utilizing external ultrasonic energy to improve the results of tumescent liposculpture. Dermatol Surg 23: 1207–1211
66. Katz BE, Bruck MC, Coleman WP (2001) The benefits of powered liposuction versus traditional liposuction: A paired comparison analysis. Dermatol Surg 27:863–867
67. Zocchi M (1998) New perspective in lipoplasty: The ultrasonic-assisted lipectomy (UAL). Presented at the French Society of Aesthetic Surgery Congress in Paris, France
68. Cardenas-Camarena L (2003) Lipoaspiration and its complications: A safe operation. Plast Reconstr Surg 112:1435–1441
69. Hurwitz DJ (2004) Single-staged total body-lift after massive weight loss. Ann Plast Surg 52:435–441
70. Gibbons MD, Lim RB, Carter PL (1998) Necrotizing fasciitis after tumescent liposuction. Am Surg 64:458–460
71. Beeson WH, Slama TG, Beeler RT, et al (2001) Group A streptococcal fasciitis after submental tumescent liposuction. Arch Facial Plast Surg 3:277–279
72. Heitmann C, Czermak C, Germann G (2000) Rapidly fatal necrotizing fasciitis after aesthetic liposuction. Aesth Plast Surg 24:344–347
73. Sharma D, Dalencourt G, Bitterly T, Benotti PN (2006) Small intestinal perforation and necrotizing fasciitis after abdominal liposuction. Aesth Plast Surg 30:712–716
74. Anwar UM, Ahmad M, Sharpe DT (2004) Necrotizing fasciitis after liposculpture. Aesth Plast Surg 28:426–427
75. Heitmann C, Czermak C, Germann G (2000) Rapidly fatal necrotizing fasciitis after aesthetic liposuction. Aesth Plast Surg 24:344–347
76. Umeda T, Ohara H, Hayashi O, Ueki M, Hata Y (2000) Toxic shock syndrome after suction lipectomy. Plast Reconstr Surg 106:204–207
77. Barillo DJ, Cancio LC, Kim SH, Shirani KZ, Goodwin CW (1998) Fatal and near-fatal complications of liposuction. South Med J 91:487–492

78. Giannella M, Pistella E, Perciaccante A, Venditti M (2005) Soft tissue infection caused by Mycobacterium chelonae following a liposculpture and lipofilling procedure. Ann Ital Med Int 20: 245–247
79. Fisher EJ, Gloster HM Jr (2005) Infection with Mycobacterium abscessus after Mohs micrographic surgery in an immunocompetent patient. Dermatol Surg 31(7 Pt 1):790–794
80. Meyers H, Brown-Elliott BA, Moore D, Curry J, Truong C, Zhang Y, Wallace Jr RJ (2002) An outbreak of Mycobacterium chelonae infection following liposuction. Clin Infect Dis 34:1500–1507 Epub 2002 May 10
81. Murillo J, Torres J, et al (2000) Skin and wound infection by rapidly growing mycobacteria: An unexpected complication of liposuction and liposculpture. The Venezuelan Collaborative Infectious and Tropical Diseases Study Group. Arch Dermatol 136:1347–1352, 2000
82. Centers for Disease Control and Prevention (CDC) (1998) Rapidly growing mycobacterial infection following liposuction and liposculpture-Caracas, Venezuela, 1996–1998. MMWR Morb Mortal Wkly Rep 47:1065–1067
83. Jones BM, Grover R (2004) Endoscopic brow-lift: A personal review of 538 patients and comparison of fixation techniques. Plast Reconstr Surg 113:1242–1250
84. Ullmann Y, Levy Y (2004) Superextended face-lift: Our experience with 3,580 patients. Ann Plast Surg 52:8–14
85. Matarasso A, Elkwood A, Rankin M, et al (2000) National plastic surgery survey: Face-lift techniques and complications. Plast Reconstr Surg 106:1185–1195
86. Scheflan M, Maillard GF, Cornette de St Cyr B, et al (1996) Subperiosteal face-lifting: Complications and the dissatisfied patient. Aesth Plast Surg 20:33–36
87. Steinsapir KD (2003) Aesthetic and restorative midface lifting with hand-carved, expanded polytetrafluoroethylene orbital rim implants. Plast Reconstr Surg 111:1727–1737
88. Pennekamp A, Pfyffer GE, Wüest J, et al (1997) Mycobacterium smegmatis infection in a healthy woman following a face-lift: Case report and review of the literature. Ann Plast Surg 39:80–83
89. Elkwood A, Matarasso A, Rankin M, et al (2001) National plastic surgery survey: Brow-lifting techniques and complications. Plast Reconstr Surg 108:2143–2150
90. Goldberg RA, Li TG (2002) Postoperative infection with group A beta-hemolytic streptococcus after blepharoplasty. Am J Ophthalmol 134:908–910