IJIR: Your Sexual Medicine Journal
https://doi.org/10.1038/s41443-020-0326-5

REVIEW ARTICLE

Penile implant infection prevention part 1: what is fact and what is fiction? Wilson's Workshop #9

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Received: 26 April 2020 / Revised: 22 May 2020 / Accepted: 29 June 2020

Abstract

Inflatable penile prosthesis (IPP) infections are undeniably devastating for patient and surgeon alike. While less common in this modern era, the landscape of prosthesis infection is shifting. Continued examination of risk factors for infection and re-evaluation of common practices remain critical should we aim to advance the field. Quality research on this topic is limited by several factors, among which small sample size and lack of coordinated effort pose the most precarious of challenges. Nonetheless, careful analysis of available data in conjuncture with judicious utilization of established research from other prosthetic fields can help us better grasp the issue at hand. In this review, we aim to do exactly that—to examine available evidence in an effort to discern fact from fiction. In this first part of the three part series, we aim to summarize our understanding of the pathogenesis behind prosthesis infections, explore known preoperative risk factors, and discuss intraoperative considerations for infection prevention. In the second part of this series, we will examine the game changing effect of infection retardant implant coatings. Part three of the series details postoperative prevention strategies, reviews salvage techniques, and discusses additional key considerations.

Introduction

Penile implant infection remains a dreadful and feared complication. The patient is miserable with pain and regrets having undergone a purely elective quality of life surgery. He now faces several future operations on his docket replete with higher complication risk and the dreaded shorter penis outcome. His medical bill will be markedly higher than the original surgery [1]. All implanters have had insomniac nights second guessing choices that might have avoided this disaster. Despite the field of modern microbiology and about 50 years of inflatable penile prosthesis (IPP) literature, our understanding of implant infection remains rudimentary and our practices too often anecdotal. For example, how can this former Köhler patient who was 3 months post-op be completely asymptomatic and using his device without incident (Fig. 1)? By historic definition his device is infected and should be removed in entirety.

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Several factors coalesce to cloud understanding of implant infections. By conservative estimates, about 25,000 implants are placed in the US per year [2]. An additional similar number is done in the rest of the world. Of these, using a 3% infection rate, 750 US IPP infections will occur annually. Seventy-five percent of all US implants are done by implanters who do <4 per year, so the vast majority of these infections will occur to a specific surgeon none to once per year [3]. According to Darouiche's statistical power calculation, "a prospective, randomized clinical trial that would sufficiently assess the efficacy of strategies for decreasing infection rates by 25% (from 3 to 2.25%) would require over 34,000 study subjects [4]." Without a national penile implant registry tracking all cases, it becomes readily apparent that because of the paucity of implantations coupled with the rarity of the complication, we will never have high-level penile implant infection specific evidence.

The issue is further confounded by methodological issues from prior and future studies. High-volume academic centers are more likely to publish their data, which are not representative of most implanters who are occasional i.e., <4 per year. High-volume centers also have a publication bias for positive results and, conversely, attrition bias is very common [5]. Attrition bias results from patients lost to follow-up. A not uncommon scenario is that a patient will

Published online: 21 July 2020

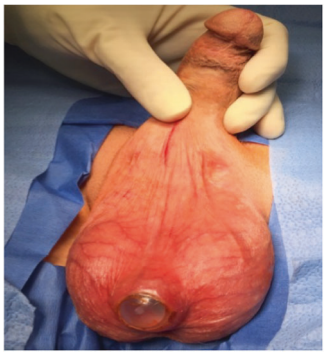

Fig. 1 A 2015 Köhler patient 3 months post-op. He was actively using device with zero symptoms or signs of infection. Patient states that the device was often rubbing on his deer stand while hunting and believes this led to this problem. Management: only the pump removed. The tubing was capped in partially inflated state good enough for intercourse. At last follow-up patient was doing well and using device without issue.

Table 1 Reasons why penile implant evidence is lacking.

- Implant infection is rare—it takes enormous studies to adequately power data [4].
- Most implants (and therefore, most infections) are done by occasional implanters who do a few per year and will never publish or contribute their findings to multicenter data gathering [3].
- Surgeons are remiss to publish negative results about infection.
- Infected patients may not return to their initial surgeons who publish results.
- PIF data is often incomplete because reps are rarely present at explants.
- Timing of IPP infection, sometimes years later, leads to loss to follow-up.
- Component erosion is misclassified as infection and vice-versa.
- Swabs of IPP at time of removal for infection are often negative [7].
- Devices removed for mechanical reasons reveal bacteria ≥80% of the time [7].

be dissatisfied with his first surgeon and go to someone else; consequently the initial surgeon never learns of his or her own complications. Implant companies attempt to track data using Product Information Forms (PIF). Unfortunately, company representatives are rarely present for infected device explant. Since they are usually the designated PIF scribe, existence of the infection is lost. Last but not least, diagnosing infection is not always straightforward. Device erosion can present as infection and vice-versa. For example, should the case from Fig. 1 be classified as an infection? Table 1 summarizes the difficulties in obtaining high-level evidence regarding penile prosthesis infection.

Despite the existing challenges, this article attempts to summarize and synthesize fact from fiction surrounding IPP infection. The authors in this paper often rely on evidence adapted from outside urology, most often the field of orthopedics. And … yes … to some degree our anecdotal

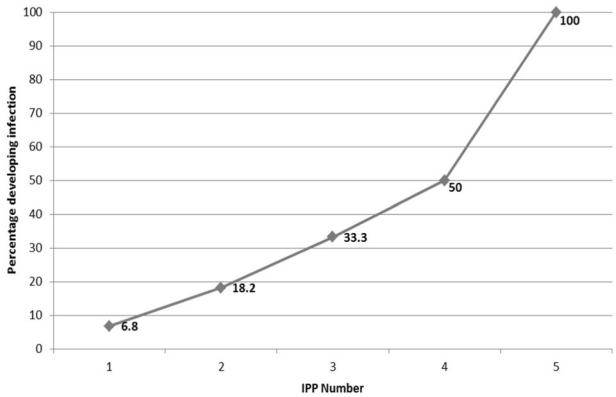

Fig. 2 Percentage of patients developing an infection with most recent device based on the IPP device number (Pearson correlation coefficient $R^2 = 0.90, p = 0.01$). As an example, 50% (4/8) of 4th IPPs in this historical series developed infection [33].

impressions are forged from >13,000 IPPs performed by the senior authors. Forty-eight years after the invention of the IPP, we do have some new insights. For example, it appears imperative to minimize risk for the first implant surgery; the best outcome always accompanies the initial operation. Moreover, infection risk rises with each subsequent IPP surgery performed and becomes inevitable by the 4th reoperation (Fig. 2) [6].

Understanding penile implant infection: a war between shifting pathogens and host factors

No matter what precautions are taken or how compulsive the surgeon's preparation, it is safe to assume the implant wound is contaminated. Up to 80% of capsules of explanted devices grow positive cultures, and examination under confocal electron microscopy detects presence of biofilm and bacteria on virtually all clinically uninfected devices [7]. After the pollution, the bacteria attempt to attach to the component surface and reproduce. At the same time the body's defense mechanisms tries to stop the colonization of the implant. If the bacteria win and reach a critical mass, they produce a protective substance resembling slime called biofilm [8]. This barrier prevents the penetration of antibiotics or the host's macrophages. With time, the biofilm structure becomes like a complicated city with nutrient and water channels feeding the quiescent bugs. Bunkered in this city are antibiotic resistant organisms in a lowered state of metabolism like bears in hibernation. These entities breed, undergo genetic transformation, and most often do not cause clinical evidence of their existence, i.e., clinical symptoms of device infection, then one day they do. The process of how and why quiescent organisms living in symbiosis with virtually every IPP recipient suddenly manifest clinically remains poorly understood.

Table 2 Penile implant infection: a war between host and pathogen.

Theoretical factors for host success		Theoretical factors for pathogen success
• Host immune system factors • Diabetes status • Immunocompromised status • Body habitus • Wound healing • Integrity and stress of closure • Host wound healing factors • Smoking • Diabetes • Tissue quality (salvage) • Host colonization and concomitant infection • Skin and nares preparation • Urine status (CIC) • Antibiotic selection • Pathogen appropriate • Adequate concentration • Tissue penetration and duration • Mechanical wound irrigation	vs.	• Bacterial type: wimpy vs. aggressive • Skin conditions and contact • Break in sterility (device prep and surgeon related) • Already present bacteria (revision) • Bacterial load: physical number of bacteria • Time wound open from case • Case complexity • Surgeon speed • Biofilm aegis and persister cells • Antibiotic resistance • Revision surgery • Pro-pathogen environments • Hematoma • Multi pathogen synergy • Foreign body surface area and type • Humidity, time of year, room pressure • Host tissue damage (cytotoxic irritant)

Prior to the availability of infection retardant-coated devices (2001), the classic organisms isolated from infected implants 75% of the time were coagulase negative staphylococcal species like *S. epidermidis*, *S. lugdunensis* [9]. These skin flora are wimpy opportunistic pathogens that do not cause any other known disease states except in the presence of a prosthetic device. The clinical appearance of this form of device infection is a local or a subacute presentation with pump tethering or wound dehiscence [7]. The infection is confined to the implant spaces and the patient is not particularly "sick." Seldom, these bugs would be the cause of an aggressive infection.

At the turn of this century, introduction of antibiotic-coated implants changed the microbial landscape of IPP infections. AMS introduced the rifampin and minocycline impregnated IPP in 2001 designed specifically to target *S. epidermidis* [10]. Following suit a year later, Coloplast unveiled the hydrophilic-coated IPP that can be dipped in a saline solution containing antimicrobial agents of choice at time of implant. Initially it was suggested that the optimum antibiotics to place in the dip were Rifampin and gentamicin or trimethoprim–sulfamethoxazole for gram positive coverage [11].

With the transition to use of coated devices, infection rates dropped by over 50% in the hands of high-volume implanters [9–11]. The two senior authors have not had a single virgin patient without risk factors that develop infection of a coated device in the past 19 years. Unfortunately, while the overall device infection rate plummeted, infected coated implant patients are declaring themselves quicker and much sicker. The unintended effect of the wide adaptation of coated devices was changing the flora of infections with more virulent organisms such as *S. aureus*, *pseudomonas*, *E. coli*, *Enterobacter*, and *Serratia* [12]. In addition to the ability to adhere to synthetic materials and produce protective biofilms like the wimpy skin bacteria, the majority of organisms found on coated devices secrete toxins and have cytotoxic capacities [13]. Infection with these bugs tends to present in an acute manner very early after implant in contrast to the skin organisms that presented months, even years later. While device infections are much rarer in the era of coated implants, these patients have both local and systemic symptoms so toxic to the patient that 83% of these devices are emergently removed [12].

Ultimately, all IPP infection literature falls into two general categories: factors that impact the host and those that influence the pathogen. Table 2 summarizes these factors and can be used to help organize various IPP infection prevention strategies. Both host and pathogen factors are critical in IPP infection prevention. Let's consider the three most important literature supported infection factors: implant revision, diabetes, and use of coated antibiotics:

- Implant revision was the single highest risk factor for infection when comparing virgin (3%) to revision cases (10%) [14]. This was prior to knowledge that washout and component exchange lowered the rate to nearing that of virgin implant [15]. Revisions are almost purely a pathogen-mediated risk factor as the host factors rarely changes from 1st to 2nd case. Instead, the pathogens which survive the first surgery with the succor of biofilm preservation and mutation rear their ugly heads. Alternately, the biofilm-protected bugs have altered the implant spaces making it receptive to bacterial growth, which is sheltered by capsule formation. When the toxic organism drops into the implant space during contamination in the revision surgery, it finds an encouraging environment. Think of it like chumming the water to attract the sharks!

Table 3 Preoperative IPP infection mitigation: host factors.

Effect on Risk • Smoking Cessation • Nares Treatment of *Staphylococcus aureus*	**Oxford Evidence Level: Practical Implications** 1: Patients should quit 4 weeks prior to reduce SSI by 57% (19) 2: Treated Patients with *S aureus* reduce SSI by 56% (21)
No Effect on Risk • Pre-operative cleansing with antiseptic • History of pelvic radiation • Age > 75 years • HIV Status • History of urinary diversion	 1: No change in SSI with home chlorhexidine scrub (28, 29) 4: No increased risk with IPPs, unlike artificial urethral sphincters (14, 30) 4: Advanced age affects immunity, no increased risk for infection (14) 4: CD4 count < 300 incurs risk, not HIV status itself 4: We typically isolate stomas out of the field (14, 30)
Increases Risk • Smoking • Diabetes Status • HbA1c level, peri-operative glucose control • *Staphylococcus aureus* nasal carriage • Spinal Cord Injury • Low CD4 count	 1: 79% higher SSI risk in active smokers (19) 2: 20-30% increased SSI risk, high diabetes prevalence (17,24) 2: Mixed data, favors much higher SSI with worse DM control (25) 2: Patients with history of MRSA skin infection should be treated (21) 3: Mixed data, neurogenic bladder + erosion likely raise risk (14,27, 28) 4: CD4 count < 300 incurs risk, not HIV status itself (32)
IPP Risk Unknown or Controversial • Obesity • Immunosuppression • Fungal infection of groin • Pre-operative urine culture	 4: No SSI increase shown except for IPP fungal infections (14) 4: Mixed data but most show no increased SSI risk (33, 34) 5: No data, IPP manufacturers state case contraindicated with any infection 5: Little data, AUA guidelines advocate urine culture and treatment (35, 36)

- In contrast, diabetic status as a host factor raises infection risk by 30%, with an overall incidence of 2–3% in high-volume surgeons' hands [16].
- Finally, coated IPP devices decrease infection risk by 50% (with overall rate of <1% in most high-volume surgeons' series)—a factor that augments both host and pathogen success in infection prevention [14, 17].

Preoperative factors: host optimization

Optimization of the implant recipient is an important component of infection risk reduction. A detailed history is essential in both first time and revision implants, with old operative reports, and recent imaging important to the latter. Table 3 summarizes preoperative IPP infection mitigation strategies stratified by risk effect and Oxford evidence level where level 1 is the best and 5 is the weakest [18].

Two factors definitively decrease IPP infection based on high levels of evidence from non-urologic literature: smoking cessation and treatment of patients with nares positive culture of *S. aureus*. In a meta-analysis of 479,150 patients, smoking cessation at least 4 weeks preoperatively led to an infection risk OR of 0.43 [19]. Paradoxically, smoking cessation within 4 weeks of surgery can promote respiratory infection due to the transient increase lung secretions during this period. Methicillin-resistant *Staphylococcus aureus* (MRSA) infection rates in patients undergoing IPP are higher than the national average [20]. In MRSA (+) patients, 5 days of chlorhexidine scrub and mupirocin nasal treatment reduce infection rates from 7.7 to 3.3% based on a large, randomized prospective trial [21]. We know very few high-volume implanters who routinely perform MRSA nares testing. However, patients should be asked about previous MRSA infection/exposure and be cultured and treated appropriately.

Diabetes is highly prevalent in patients suffering from erectile dysfunction (ED) and is a major cause of phosphodiesterase type 5 inhibitor refractory ED [22, 23]. We feel it is the most important preoperative risk factor, increasing infection rates by 23–32% [17, 24]. While the diabetic status confers risk, there is conflicting data that are reported as to the importance of preoperative Hemoglobin A1C (HbA1c), a measure of glycemic control over months, or fasting blood sugar on the morning of surgery. One recent study determined a threshold of 8.5% conferred a statistically significant risk for IPP infections [25] while a recent large series felt neither A1C or FBS level predicted risk [26].

Although the literature for spinal cord injury is mixed, overall it appears to increase IPP infection risk [14, 27]. Patients with spinal cord injury often have neurogenic bladders and decreased or absent perineal sensation. Thus, increased infection risk may be related to both the need for catherization and higher risk of device component erosion [28].

Based on trials with >10,000 patients, preoperative home patient cleansing with chlorhexidine for several days prior to the operation does not reduce infection risk [29, 30]. Nonetheless, it is always beneficial to engage the patient in

Table 4 Intraoperative IPP infection mitigation: pathogen factors.

Effect on Risk	Oxford Evidence Level: Practical Implications
• Operative site scrub	1: Chlorhexidine-alcohol over povidone-iodine (8 vs 32% skin Cx + after prep) (39, 40)
• Hair removal	1: Clippers superior for infrapubic incision, no scrotal incision data (38)
• Parenteral antibiotics	2: Based on RCTs from other prosthetic surgical specialties (43)
• Infection retardant coated devices	2: Decrease infection rate by 50% based on retrospective data (9)
• Surgeon experience	2: High volume surgeons (> 32 IPPs/year) ~ 50% less likely to get infection (50)
• "No touch technique"	3: Targets seeding with skin flora in the pre-coated device era (53)
No Effect on Risk	
• Surgeon hand washing approach	1: Avaguard® or Sterilium® equivalent instead of standard 10-minute sink scrub (37)
• Scrotal drain	1: No effect on postop infection rate based on retrospective & meta-analysis data (48, 49)
• Concomitant circumcision	3: No increase infection based on multiple retrospective reports (46, 47)
• Surgical approach	4: Not statistically different between penoscrotal and infrapubic (45)
Increases Risk	
• Low IPP volume surgeon	2: Annual IPP rate of <2 have highest infection rate (OR 2.5x vs > 32 IPP/year) (50)
• Increased operative time	2: Ortho study with 35% higher risk at 90 days for each additional 20 minutes (51)
IPP Risk Unknown or Controversial	
• Intraoperative glove change	2: No direct link to infection, but gloves often contaminated on culture (54)

their own care and create the perception of infection control vigilance. Similarly, no contemporary study has linked device infection with either a prior history of radiation treatment or urinary diversion [14, 31]. When controlled for diabetes, neither obesity nor advanced age predispose patients to infection after implant [14].

The broad term of immunosuppression remains poorly understood as a risk factor for IPP. CD4 count <300 does increase postoperative infections [32]. However, examination of solid organ transplant patients and those on long-term steroid therapy did not yield statistically convincing elevations in risk for IPP infection [33, 34]. Both device companies list active infections elsewhere in the body (e.g., UTI, skin infection) as a contraindication to proceeding with surgery. Similarly, AUA guidelines recommend treatment of concomitant infection prior to surgery [35]. However, no quality study has proven the utility of preoperative urine culture for the general IPP population. A large retrospective review found no statistical difference in infection rate between patients with and without pre-op urine culture [36].

Intraoperative factors: pathogen minimization

Traditional hand-washing: the surgeon need not bother with the traditional 10-min hand scrub, as level 1 evidence deems aqueous alcohol solutions e.g., Avagard® or Sterilium® equivalent [37]. The operative canvas should be cleared of any debris, which may harbor insidious organisms prior to incision. For infrapubic incisions, high-level evidence recommends trimming hair with clippers rather than razors to minimize disruptions to the skin barrier [38]. Given the irregular contours of the scrotum, intraoperative hair removal technique is best left to the discretion of the surgeon. Next, scrub the skin with an antiseptic solution to sterilize the field as much as possible. Povidone-iodine solutions were historically favored until relatively recently. In a randomized prospective trial of patients undergoing genitourinary prosthesis implant, an astonishing 32% of skin cultures remained positive after preparation with povidone-iodine scrub [39]. As such, the 2016 ICSM guideline [40] favors the use of chlorhexidine-alcohol skin paint (not scrub) prior to implant surgery based on results of several large randomized studies [41, 42]. By contrast, the previous study showed an 8% positive culture rate even with the use of chlorhexidine. This data illustrates how IPP infection mitigation rarely involves absolute prevention of bacterial wound contamination, but rather limitation of total bacteria bolus and the targeting of bacteria with greater virulence. Table 4 summarizes the intraoperative IPP infection mitigation strategies stratified by risk effect.

What about perioperative antibiotics? The critical role of parenteral antibiotics at time of prosthetic surgery is well established thanks to our orthopedic surgery colleagues [43]. Interestingly, since the introduction of the coated IPP devices almost 20 years ago, patients are now presenting with increasingly more virulent bugs [12]. A recent study showed that only 62–86% of organisms cultured from explanted devices following device infection were susceptible to the prophylactic parenteral antibiotic administered at time of surgery [44]. In today's era of infection retardant-coated implants, routine coverage of indolent skin organisms that usually caused infections in the non-coated devices is no

longer enough. Rather, careful consideration of patient risk factors e.g., diabetes, immunosuppression should prompt coverage with antimicrobials specific for the situation.

Does incision site make a difference? Neither penoscrotal nor infrapubic surgical approach appears to be superior to the other in infection prevention. A small retrospective study hints at a potential increase in infection rate with the infrapubic incision, but this cohort lacked the necessary power to achieve statistical significance [45]. Conceptually the idea of concomitant circumcision, hydrocele, epididymectomy at time of implant raises possible infection concerns, but small retrospective data have not yielded clinical significance on coincident surgeries [46, 47]. Wilson suggests doing the clean surgery (IPP) first and then through a separate incision accomplish the accompanying surgery.

To drain or not to drain? Studies show no appreciable association between scrotal drain placement and infection rates [48]. A recent high-level Cochrane database review verified this supposition [49].

Does surgical experience matter? Surgical experience and frequency, on the other hand, does appear to play a critical role in terms of overall outcomes. Those who perform IPP surgeries infrequently are afflicted with higher complication rates, including infections [50]. Relative risk of IPP infection was 2.5, 2.4, and 2.1 times higher in implanters performing 0–2, 3–7, 8–31 IPPs per year respectively compared with those who performed >31 cases per year. In addition, by extrapolating data from our orthopedic colleagues, it appears that infection rates increase with every passing minute under the knife [51]. Operative efficiency is a skill honed over many arduous years and requires a significant surgical volume to develop. Given that 75% of IPP surgeries are being done by surgeons who perform less than four IPPs a year [3], many surgeons, no matter how gifted their skills are, will simply not have enough repetition to truly minimize infection risk.

Does IPP revision increase infection rate? No discussion of IPP infection prevention is complete without visiting the topic of revision surgeries. Each revision on an IPP automatically elevates the postoperative infection rates until by the 5th operation the accompanying device infection rate approaches 100% [6]. We know from Henry and Wilson's multicenter studies that revision IPP infection rate is lowered by washing out the spaces with antiseptic solutions and performing component exchange [15]. We have learned that the classic irrigation solutions should be optimized [52]. During the washout, because of the risk of air embolism, our traditional Mulcahy irrigation with 50% hydrogen peroxide should be discontinued. In addition, the betadine solution must be drastically diluted from a 50% blend. Instead, a 0.35–3.5% betadine solution should be used with 3 min of dwell time followed by copious saline irrigation. Remember, as Dr Mulcahy always says, "the solution to pollution is dilution."

With the inevitability of microbial seeding in mind, if we can limit the absolute number of bacterial clusters to contact the device at time of implant, we may prevent them from reaching the critical mass needed to trigger clinically significant symptomology [8]. The "no touch" technique was introduced by Eid and Wilson in 2012 to prohibit the sterile device components from touching the patient's skin, contaminated gloves, or soiled instruments [53]. Eid's penoscrotal approach employs frequent glove changes with the surgical field re-draped and new instruments opened prior to opening Buck's fascia. By meticulously following this protocol over the course of 4 years, this single surgeon was able to reduce his infection rate from 2.0 to 0.46%. The principles of this discipline have significantly impacted our prosthetic urology field. Nevertheless, the "no touch" technique development was aimed at skin organisms; not the toxic bugs we find ourselves dealing with in the rare device infection we encounter in the era of the coated implant. Level 2 evidence shows high level of glove positive culture rates and micro-tearing in implant cases arguing for glove change prior to handling the implant [54]. However, no studies to date have demonstrated an increased risk of infection as a result of positive glove cultures or glove microtears. As such, the authors routinely double glove but only seldom perform glove changes intraoperatively.

In institutions where there are multiple prosthetic surgeons, e.g., a university hospital, it is useful to implement mandatory protocols of patient preparation. Of course, disciplines to prevent infection only work if implemented. To quote Dr Gawande's apt observation from The Checklist Manifesto [55], "A further difficulty, just as insidious, is that people can lull themselves into skipping steps even when they remember them." Katz and colleagues demonstrated in their 2014 report that compulsive adherence to a mandatory presurgical checklist by multiple surgeons effectively brought their hospital wide IPP infection rate down to almost zero [56]. By adhering to a systematic process for IPP infection prevention, the determined surgeon can seek to overcome this innate desire to simplify repetitive processes, reducing infection rates along the way.

High-volume implanters share a common low infection rate but utilize very different techniques. Careful analysis of Tables 3 and 4 reveal competing statements. Is a "no touch" technique with frequent glove changes worth the additional risk of keeping the wound open longer? Similarly, is the convenience of concomitant circumcision with IPP worth the theoretical increased risk? Each surgeon must choose their one best technique and seek to perfect it. Our approach favors operative efficiency and speed so we do not utilize the "no touch" technique. If a case runs longer than expected only then will we delay additional concomitant procedures or maneuvers.

Conclusion

This Wilson Workshop has attempted to separate the facts of diminishing the risk of implant infection from the fictions and anecdotal opinions of our audience, the implanting surgeons. Admittedly, high-level evidence for penile prosthetics is scarce based on several described methodological factors. The extensive reference listing of historical milestones in IPP infections and cutting-edge recent investigations from other surgical disciplines employing devices combine to allow us to pass judgment on fact or fiction. Fortunately, the infection rate for IPPs has plummeted in recent years. We believe this is due to widespread adoption of infection retardant-coated devices, improved skin preparation, and complete component exchange coupled with copious washout during revision for noninfectious reasons. The very rare (<1%) device infection seen by high-volume implanters has different organisms and a different clinical picture than during the era of the non-coated implants. IPP infection results from pathogens gaining enough momentum to overcome the defenses of the host. We advocate following a standardized procedure to optimize the resistance of the host based on high-level evidence coupled with an emphasis on surgical speed and efficiency. Part 2 of this surgical series will focus on device coatings. Part 3 of this series will focus on postoperative infection prevention, recognition, and current thoughts on salvage surgery.

Compliance with ethical standards

Conflict of interest TSK is a consultant for Boston Scientific and Coloplast. SKW is a consultant for AMT, Coloplast, and International Medical Devices. He is also a lecturer for Boston Scientific, and a NeoTract Stockholder. The other author declare that he has no conflict of interest.

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