

Deep and Superficial Debridement Techniques in Lower Extremity Split-thickness Skin Grafting

Rachel N. Rohrich, BS*

Karen R. Li, BBA*†

Christian X. Lava, MS*†

Sami Alahmadi, MS†

Henry L. Stanton, BS†

Victoria H. Kim, MS†

Daisy L. Spoer, MS†

Karen K. Evans, MD*

John S. Steinberg, DPM†

Christopher E. Attinger, MD*

Background: Patients with nonhealing lower extremity (LE) wounds often require a split-thickness skin graft (STSG) for closure. Nonviable tissue must be debrided before STSG inset. Our study aimed to compare differences in debridement depth on STSG outcomes.

Methods: Chronic, atraumatic LE wounds receiving STSG from December 2014 to December 2022 at a single institution were reviewed. Demographics, wound characteristics, operative details, and outcomes were collected. Superficially debrided wounds were compared with wounds receiving deep debridement (DD), defined by debriding to the level of white tissue underlying the granulation tissue. Subanalysis was performed on wounds that had a negative and positive postdebridement culture. Primary outcome was graft failure.

Results: Overall, 244 wounds in 168 patients were identified. In total, 158 (64.8%) wounds were superficially debrided and 86 (35.3%) received DD. The cohort had a median Charlson Comorbidity Index of 4 [interquartile range (IQR): 3]. Diabetes (56.6%) and peripheral artery disease (36.9%) were prevalent. The only statically significant demographic difference between groups was congestive heart failure (SD: 14.9% versus DD: 3.0%, $P = 0.017$). Wound size, depth, and all microbiology results were similar between groups. Postoperatively, the DD group demonstrated significantly less graft failure (10.5% versus 22.2%, $P = 0.023$). In a multivariate regression, DD was independently associated with lower odds of graft failure (OR: 0.0; CI, 0.0–0.8; $P = 0.034$). Sub-analysis of graft failure supported this finding in culture-positive wounds (DD: 7.6% versus DD: 22.1%, $P = 0.018$) but not in culture-negative wounds (13.6% versus 22.2%, $P = 0.507$).

Conclusions: The DD technique demonstrates improved outcomes in chronic, culture-positive LE wounds receiving STSG. (*Plast Reconstr Surg Glob Open* 2024; 12:e6048; doi: [10.1097/GOX.0000000000006048](https://doi.org/10.1097/GOX.0000000000006048); Published online 12 August 2024.)

INTRODUCTION

Chronic wounds pose significant medical challenges to patient morbidity, quality of life, and healthcare costs.¹ In the atraumatic lower extremity (LE) wound population, a split-thickness skin graft (STSG) is commonly used when healing through secondary intention is not feasible.^{2,3} Common in this multimorbid population, wound beds with poor vascularity, necrotic tissue, and infection

contribute to STSG failure.³ Infection is a common cause of poor outcomes, with successful STSGs only able to withstand 10^5 bacteria per gram of tissue.³⁻⁵ To achieve a healthy granulation bed, surgical debridement is the gold standard for excising nonviable tissue and bacterial debris.^{1,2} A debate remains about the depth of debridement required to achieve an optimal wound bed for successful skin grafting.^{1,3,6} Our study compares the impact of deep and superficial debridement techniques in chronic, atraumatic LE wounds treated with STSG.

METHODS

Following institutional review board approval (STUDY00004145), we reviewed electronic medical records for chronic, atraumatic LE wounds receiving

Disclosure statements are at the end of this article, following the correspondence information.

Related Digital Media are available in the full-text version of the article on www.PRSGlobalOpen.com.

From the *Department of Plastic and Reconstructive Surgery, MedStar Georgetown University Hospital, Washington, D.C.; †Georgetown University School of Medicine, Washington, D.C.; and ‡Department of Podiatric Surgery, MedStar Georgetown University Hospital, Washington, D.C.

Received for publication March 20, 2024; accepted June 17, 2024.

Copyright © 2024 The Authors. Published by Wolters Kluwer Health, Inc. on behalf of The American Society of Plastic Surgeons. This is an open-access article distributed under the terms of the [Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 \(CCBY-NC-ND\)](https://creativecommons.org/licenses/by-nc-nd/4.0/), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

DOI: [10.1097/GOX.0000000000006048](https://doi.org/10.1097/GOX.0000000000006048)



Fig. 1. Comparative images of a wound before and after a deep debridement. A, Wound before debridement. B, Wound after deep debridement, evident by reaching the white wound base.

STSG between December 2014 and December 2022. Operations were performed at a single institution by a total of six senior surgeons. Patients were excluded if they had incomplete follow-up information or received a synthetic dermal substitute (SDS), such as Integra (Integra LifeSciences, Princeton, N.J.), before STSG. At our institution, wounds that are exceptionally large or have deep exposed structures are pretreated with SDS by our wound

Takeaways

Question: Does a deeper debridement before a split-thickness skin graft improve outcomes?

Findings: A retrospective review of 168 patients with 244 chronic lower extremity wounds showed that those receiving a deep debridement experienced significantly less graft failure (10.5% versus 22.2%, $P = 0.023$) and complications requiring reoperation (25.6% versus 39.2%, $P = 0.032$). When stratified by culture-negative and culture-positive wounds, this finding only remained significant in culture-positive wounds.

Meaning: In culture-positive chronic wounds colonized by bacteria, a deeper debridement is a valuable method to improve STSG outcomes.

clinic 3–4 weeks before STSG. Because this pretreatment enhances granulation tissue formation and leads to better STSG take rates, we excluded these wounds in our present investigation to reduce confounding factors and analyze a homogenous population.

Study Groups and Surgical Technique

Patients and wounds were compared by debridement technique. Patients requiring STSG were assigned to surgeons based on clinical scheduling without selection bias, resulting in random allocation to either the deep debridement (DD) technique, performed by one surgeon at our institution, or superficial debridement (SD) technique, performed by the remaining five surgeons. The DD technique completely removes the granulation tissue down to the base of the granulation buds, not just until bleeding tissue is reached, but until the white-colored base of the tissue is visible (Fig. 1). This white tissue layer, representing the very base of granulation tissue “buds,” serves as a clinical indicator of the depth of the DD method. The SD technique reflected a standard debridement where removal of granulation tissue was to healthy bleeding tissue, not reaching the white base layer (Fig. 2).

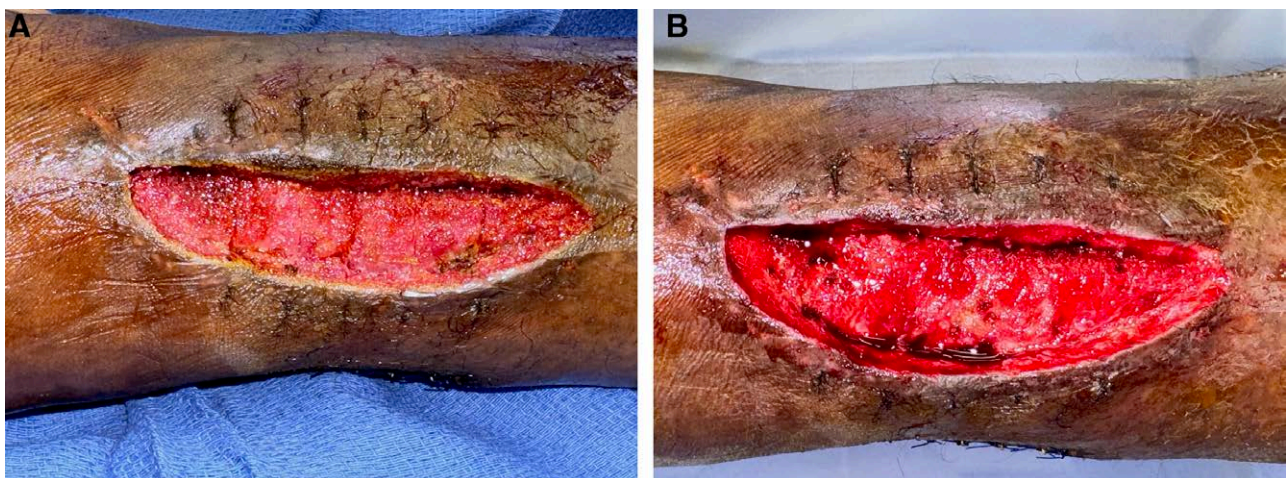


Fig. 2. An image of a wound (A) before and (B) after a superficial debridement.

All patients underwent debridement before STSG. For both the DD and SD methods, all surgeons utilized a knife, rongeur, and hydrosurgical blade (VERSAJET; Smith & Nephew, Fort Worth, Tex.) to excise necrotic and infected tissue until all wound borders were clean and showed substantial bleeding at the base. SD was achieved at this level by shaving off the top layer of granulation tissue but leaving the base of granulation buds intact. For patients who underwent DD, the granulation tissue was totally removed, leaving behind an intact white base layer of tissue. All wounds were irrigated with 3 L of normal saline. Drapes, gloves, and instruments were changed after irrigation. Pre- and post-debridement cultures were obtained via an intraoperative swab of the entire wound surface. STSG harvest and placement followed identical surgical technique for all wounds. All STSGs were sutured to the wound bed using 4-0 absorbable sutures and dressed with a silicone interface followed by a sponge and tie over a multilayer compression dressing or negative pressure wound therapy (NPWT). NPWT was used in cases of excessive edema or joint motion. Per institutional protocol, all patients were strictly immobilized in a neutral position.⁷

Data Collection

Charts were reviewed to gather patient demographics, microbiology data, wound characteristics, and postoperative outcomes. Demographic data included age, sex, body mass index (BMI), and medical history. The Charlson Comorbidity Index (CCI) was used to calculate comorbidity burden.⁶ Wound characteristics, determined on the day of STSG surgery, included location, dimensions, and depth. Bacterial presence in the wound on day of STSG inset, including bacterial load and type, was determined by qualitative microbiology results from intraoperative debridement cultures. *Pseudomonas aeruginosa* and *Staphylococcus aureus* were of interest due to their contribution to poor outcomes.^{8–11} Graft thickness was categorized into five categories for analysis: very thin (<0.15 mm), thin (0.15–0.3 mm), intermediate (0.3–0.45 mm), thick (0.45–0.6 mm), and very thick (0.6–1.0 mm).^{2,12,13} Postoperative dressing type as previously described was also collected.

Patients were assessed at our outpatient wound clinic on postoperative day 30 (POV-30) and 60 (POV-60). Patients lost to follow-up were excluded. The primary outcome was graft failure, defined by the attending surgeon's documentation of complete necrosis, total sloughing, or removal of the STSG, or if the clinical note included the International Classification of Diseases 10th Revision Code T86.821 for failure of an allograft or autograft skin graft. Time to graft failure was calculated as the days between STSG placement and graft failure documentation. Secondary outcomes included infection and healing rates. Healing rate was evaluated by the binary variable clinical healing, determined by documentation of the wound as healed in attending notes. Healing course was further evaluated by incidence of reoperation, when patients required further surgical intervention to heal the same wound site treated by the original STSG.

A subgroup analysis was performed on culture-negative and culture-positive wounds. Culture-negative

wounds were defined as those that had a negative result on the postdebridement culture before STSG while culture-positive wounds had a positive result on the final postdebridement culture before STSG.

Statistical Analysis

Summary statistics are presented for the overall sample and by study groups as means, medians, SDs, minimums, maximums, and proportions (if categorical). Two-sample *t* tests were used to examine differences in the averages of continuous variables between groups (SD versus DD) when the normality assumption was satisfied. The Wilcoxon rank-sum test was used when the normality assumption was not satisfied. Chi-square and Fisher exact tests (defined as cell counts less than 5) were used to investigate differences for categorical variables as appropriate. A univariate linear regression analysis across all collected variables was conducted to evaluate the influence of demographic, wound, and operative characteristics on the incidence of graft failure. Variables in this univariate that demonstrated statistical significance were included in a multivariate linear regression analysis to determine independent predictors of graft failure. Statistical significance was defined as a *P* value less than 0.05 for between group differences, univariate, and multivariate analyses. StataMP Software (StataCorp LLC, College Station, Tex.) was used to perform all analyses. Results are reported according to the Strengthening the Reporting of Observational Studies in Epidemiology checklist.¹⁴

RESULTS

Patient Demographics and Wound Characteristics

Of the 316 patient charts screened, 10 were excluded due to incomplete follow-up information and 138 were excluded due to preoperative SDS usage. Our remaining study cohort included 168 patients who received STSG coverage for 244 chronic LE wounds. Patient demographics are summarized in Table 1. In total, 101 (60.1%) patients received SD and 67 (39.9%) patients received DD. Overall, the cohort was 61.3% men with a mean age of 61.9 ± 15.1 years and BMI of 28.6 (IQR: 8.9) kg/m², with no differences between groups (*P* = 0.534 and *P* = 0.751, respectively). The median CCI was 4 (IQR: 3), with no differences between groups (*P* = 0.397). Diabetes (56.6%, *P* = 0.217), chronic kidney disease (CKD) (22.6%, *P* = 0.235), and peripheral artery disease (PAD) (36.9%, *P* = 0.285) were prevalent among both groups. Incidence of congestive heart failure was the only significant demographic difference between groups (SD: 14.9% versus DD: 3.0%, *P* = 0.017).

Wound characteristics and operative details are listed in Table 2. Median wound surface area was 28.0 (IQR: 66.0) cm², with no differences between groups (*P* = 0.378). While all wounds were located on or below the knee, distribution of wound location varied significantly between groups (*P* = 0.001) with the DD group having a higher incidence of knee wounds (DD: 11.6% versus SD: 2.3%), and the SD group having a higher incidence of forefoot wounds (SD: 26.0% versus DD: 8.1%). Of foot wounds,

Table 1. Patient Demographics

	Total, No. (%) [168 (100%)]	SD, No. (%) [N = 101 (60.1%)]	DD, No. (%) [N = 67 (39.9%)]	P
Demographics				
Age, mean ± SD	61.9 ± 15.1	61.6 ± 15.4	62.1 ± 14.7	0.751
Sex				0.534
Male	103 (61.3)	60 (59.4)	43 (64.2)	
Female	65 (38.7)	41 (40.6)	24 (36.8)	
BMI (kg/m ²), median [IQR]	28.6 [8.9]	28.7 [9.0]	28.4 [9.1]	0.661
Comorbidities				
Smoking*				0.492
Never	83 (50.6)	51 (51.0)	32 (50.0)	
Former	34 (20.7)	18 (18.0)	16 (25.0)	
Current	47 (28.7)	31 (31.0)	16 (25.0)	
CCI, median [IQR]	4 [3]	4 [4]	4 [3]	0.397
Diabetes	95 (56.6)	61 (60.4)	34 (50.8)	0.217
Peripheral artery disease	62 (36.9)	34 (33.7)	28 (41.8)	0.285
History of myocardial infarction	16 (9.5)	13 (12.9)	3 (4.5)	0.070
History of CVA or TIA	18 (10.7)	10 (9.9)	8 (11.9)	0.676
History of malignancy	23 (13.1)	12 (11.9)	10 (14.9)	0.567
Chronic kidney disease	38 (22.6)	26 (25.7)	12 (17.9)	0.235
Congestive heart failure	17 (10.1)	15 (14.9)	2 (3.0)	0.017†
History of hypertension	137 (81.6)	84 (83.2)	53 (79.1)	0.506
ASA classification				0.645
Class I	4 (2.4)	3 (3.0)	1 (1.5)	
Class II	10 (6.0)	5 (5.0)	5 (7.5)	
Class III	136 (81.0)	80 (79.2)	56 (83.6)	
Class IV	18 (10.7)	13 (12.9)	5 (7.5)	

*Smoking history available for only 164 overall: 100 for the SD group, and 64 for the DD group.

†Statistically significant ($P < 0.05$).

ASA, American Society of Anesthesiologists.

incidence of plantar defects was similar between groups (DD: 30.0% versus SD: 35.5%, $P = 0.588$). Wound depth was also similar between groups ($P = 0.888$), with most wounds extending to the subcutaneous (57.0%). Most STSGs (56.5%) were harvested at an intermediate thickness. SD was more commonly grafted with very thin STSGs, and DD was more commonly grafted with thin STSGs ($P < 0.001$). No significant differences in NPWT use were observed between groups (SD: 27.7% versus DD: 37.2%, $P = 0.106$).

Microbiology results for pre- and postdebridement cultures are summarized in [Table 3](#). Pre- and postdebridement cultures were available for 163 and 202 wounds, respectively. Incidence of positive predebridement cultures was similar among DD and SD wounds (73.2% versus 80.4%, $P = 0.295$), in addition to rates of polymicrobial colonization, pathogen type, and bacterial load. Both DD and SD groups experienced lower rates of positive postdebridement cultures (70.7% versus 62.9%, $P = 0.881$). All bacterial species cultured from DD and SD wounds are presented in Supplemental Digital Content 1. [See [table, Supplemental Digital Content 1](#), which displays (a) pre- and (b) postdebridement bacterial species, <http://links.lww.com/PRSGO/D418>.]

Forty-four bacterial species were identified with no significant differences between study groups for bacterial species grown from predebridement cultures. The DD group demonstrated significantly lower rates of positive *Enterococcus faecalis* (0.0% versus 5.4%, $P = 0.040$) and

Diphtheroids (10.7% versus 24.0%, $n = 0.019$) growth on postdebridement cultures.

STSG Clinical Outcomes

Outcomes are summarized in [Table 4](#). Overall, the rate of graft failure was 18.0%, in which the DD group demonstrated significantly lower rates (10.5% versus 22.2%, $P = 0.023$). The mean time to graft failure was 47.3 ± 23.7 days ($P = 0.404$).

Our univariate linear regression of all collected variables is displayed in Supplemental Digital Content 2. (See [table, Supplemental Digital Content 2](#), which displays univariate regression across collected variables, <http://links.lww.com/PRSGO/D419>.) We observed a history of cerebral vascular accident or transient ischemia attack (TIA); wound length, surface area, and depth; plantar location; a postdebridement culture positive for a multidrug resistant (MDR) organism; and DD as significant covariates with graft failure. We included these variables in our multivariate model ([Table 5](#)). We did not observe the use of NPWT, STSG thickness, wound depth, or type of bacterial species to influence graft failure in our univariate regression, and thus did not include these in our multivariate regression. In our model, DD was independently associated with decreased odds of graft failure (OR: 0.0, CI: (0.0, 0.8), $P = 0.034$).

The median final follow-up time was 9.0 (IQR: 23.0) months. There were no significant differences between groups in clinical healing at POV-30 or POV-60. However,

Table 2. Wound Characteristics and Perioperative Details

	Total, No. (%) [N = 244 (100%)]	SD, No. (%) [N = 158 (64.8%)]	DD, No. (%) [N = 86 (35.3%)]	P
<i>Wound Characteristics</i>				
Wound Dimensions, Median [IQR]				
Width (cm)	4.0 [5.6]	4 [4.5]	4 [6]	0.671
Length (cm)	6.0 [8]	7 [9]	6 [7]	0.166
Surface area (cm ²)	28.0 [66]	30 [71]	24 [56]	0.378
Wound Depth*				
Eschar	3 (1.2)	2 (1.3)	1 (1.2)	0.896
Dermis	7 (3.0)	4 (2.6)	3 (3.5)	
Subcutaneous	138 (57.0)	87 (55.8)	51 (59.3)	
Fascia	45 (18.6)	31 (19.9)	14 (16.3)	
Muscle	23 (9.5)	14 (9.0)	9 (10.5)	
Tendon	12 (5.0)	7 (4.5)	5 (5.8)	
Bone	14 (5.8)	11 (7.1)	3 (3.5)	
Wound Location				
Forefoot	48 (19.7)	41 (26.0)	7 (8.1)	0.001 †
Midfoot	19 (7.8)	12 (7.6)	7 (8.1)	
Hindfoot	34 (13.9)	18 (11.4)	16 (18.6)	
Ankle	45 (18.4)	29 (18.4)	16 (18.6)	
Lower leg	78 (32.0)	48 (20.4)	30 (34.9)	
Knee	15 (6.2)	5 (3.2)	10 (11.6)	
Transmetatarsal amputation site	5 (2.1)	5 (3.2)	0 (0.0)	
Foot Wound Surface ‡				
Dorsal	70 (66.0)	49 (64.5)	21 (70.0)	0.588
Plantar	36 (34.0)	27 (35.5)	9 (30.0)	
<i>Perioperative Details</i>				
STSG Thickness §				
Very thin (<0.15 mm)	51 (22.2)	50 (32.3)	1 (1.3)	<0.001 †
Thin (0.15–0.3 mm)	45 (19.6)	8 (5.2)	37 (49.3)	
Intermediate (0.3–0.45 mm)	130 (56.5)	96 (61.9)	34 (45.3)	
Thick (0.45–0.6 mm)	3 (1.3)	0 (0.0)	3 (4.0)	
Very thick (0.6–1.0 mm)	1 (0.4)	1 (0.65)	0 (0.0)	
Postoperative Dressing				
Multilayer compression wrap	169 (69.3)	115 (72.8)	54 (62.8)	0.106
NPWT	75 (30.7)	43 (27.2)	32 (37.2)	

*Depth available for 242 overall: 156 for the SD group and 86 for the DD group.

†Statistically significant ($P < 0.05$).

‡Percentages out of total wounds located on the foot (forefoot, midfoot, hindfoot, and transmetatarsal amputation site), 106 overall: 76 for the SD group and 30 for the DD group.

§Graft thickness available for 230 overall: 155 for the SD group and 75 for the DD group.

by final follow-up, the DD group demonstrated significantly higher rates of clinical healing (84.5% versus 70.5%, $P = 0.027$). In addition, wounds in the SD group demonstrated a higher incidence of reoperation (39.2% versus 25.6%, $P = 0.032$). Incidence of infection was similar between groups at POV-30 (3.0%), POV-60 (7.5%), and at final follow-up (6.4%).

Subgroup Analysis of Culture-negative versus Culture-positive Wounds

Primary outcomes for culture-negative and culture-positive wounds are outlined in Table 6. Of culture-negative wounds ($n = 58$), the graft failure rate was 19.0%, with no significant differences between groups ($P = 0.507$). In the culture-positive group ($n = 144$), the overall rate of graft failure was 17.4%, in which the DD group had a significantly lower rate (7.6% versus 22.1%, $P = 0.019$). Furthermore, in this culture-positive subgroup, the DD group exhibited significantly higher rates of clinical healing at final

follow-up compared with the SD group (84.1% versus 66.2%, $P = 0.034$).

DISCUSSION

Principal reasons for LE STSG failure are (1) poor vascularity, (2) infection, and (3) shearing forces.^{2,15–17} To decrease the risk of STSG failure due to improper wound bed preparation, we investigated the relationship between debridement technique and clinical outcomes. Currently, the general consensus is that a healthy granulation bed alone suffices for successful STSG take in all wounds.^{1,18–21} However, we found in our study that this method was adequate only if the wound was culture-negative. In culture-positive wounds colonized by bacteria, we found that a deeper debridement was more effective to improve STSG outcomes.

Wound Characteristics and Patient Population

It is well known that comorbidities such as diabetes, PAD, and CKD impair wound healing, due to microvascular

Table 3. Predebridement Wound Bed Microbiology

	Predebridement Culture			
	Total, n (%) [N = 163 (%)]	SD, n (%) [N = 107 (65.6%)]	DD, n (%) [N = 56 (34.4%)]	P
Culture positive	127 (78.9)	86 (80.4)	41 (73.2)	0.295
Polymicrobial	80 (49.1)	59 (55.1)	21 (37.5)	0.101
Multidrug-resistant organism	18 (14.2)	13 (15.1)	5 (12.2)	0.789
Pathogens of interest				
<i>Pseudomonas aeruginosa</i>	24 (14.7)	15 (14.0)	9 (16.1)	0.725
<i>Staphylococcus aureus</i>	29 (17.8)	19 (17.8)	10 (17.9)	0.987
Bacterial load*				0.620
None	36 (22.2)	21 (19.8)	15 (26.8)	
Broth	8 (4.9)	6 (5.7)	2 (3.6)	
Rare	2 (1.2)	2 (1.9)	0 (0.0)	
Scant, few, or light	65 (40.1)	41 (38.7)	24 (42.9)	
Moderate	28 (17.3)	18 (17.0)	10 (17.9)	
Heavy	23 (14.2)	18 (17.0)	5 (8.9)	
	Postdebridement Culture			
	Total, n (%) [N = 202 (100%)]	SD, n (%) [N = 127 (62.9%)]	DD, n (%) [N = 75 (37.1%)]	P
Culture positive	144 (71.3)	91 (71.7)	53 (70.7)	0.881
Polymicrobial	79 (39.1)	55 (43.3)	24 (32.0)	0.210
Multidrug-resistant organism	37 (25.7)	24 (26.4)	13 (24.5)	0.807
Pathogens of interest				
<i>Pseudomonas aeruginosa</i>	20 (9.9)	13 (10.2)	7 (9.3)	0.836
<i>Staphylococcus aureus</i>	37 (18.3)	20 (15.8)	17 (22.7)	0.219
Bacterial load†				0.382
None	58 (28.9)	36 (28.6)	22 (29.3)	
Broth	18 (9.0)	12 (9.5)	6 (8.0)	
Rare	3 (1.5)	3 (2.4)	0 (0.0)	
Scant, few, or light	110 (54.7)	65 (51.6)	45 (60.0)	
Moderate	12 (6.0)	10 (7.9)	2 (2.7)	
Heavy	0 (0.0)	0 (0.0)	0 (0.0)	

*Predebridement bacterial load available for only 162 overall: 106 for the SD group, and 56 for the DD group.

†Postdebridement bacterial load available for only 201 overall: 126 for the SD group, and 75 for the DD group.

disease, peripheral neuropathy, and impaired metabolism.²² Our study population had a diabetes rate of 56.6%, PAD rate of 36.9%, and CKD rate of 22.6%, with a median CCI burden of 4.0. To control for such a heavy comorbidity burden among our population, we included significant demographic and wound characteristics determined by our univariate model in a multivariate model. We found that the DD technique remained independently associated with a lower likelihood of graft failure. Taken together, despite concerns that large, chronic wounds cannot handle a reepithelization burden after an aggressive operative approach, our results show that the DD method reduces the rate of STSG failure and does not hinder wound healing.

In addition to patient comorbidities, wound characteristics play a role in STSG reconstruction.^{3,23} Wounds that are larger, deeper, grafted with thicker STSGs, and located on plantar surfaces are at higher risk for STSG failure.^{2,24} Furthermore, NPWT has been shown to improve STSG outcomes.^{25–29} Our results demonstrated no significant difference between groups in wound depth, dimensions, use of NPWT, or frequency of plantar defects. Only plantar surface emerged as a significant covariate for graft failure, and we thus controlled for this in our multivariate regression. DD and SD wounds did differ in location and STSG thickness, both of which

showed no significance in the univariate regression. In fact, SD wounds were more commonly grafted with thinner STSGs, which are known to contribute to better outcomes via improved nutrient diffusion.² However, despite utilization of thinner STSG, the SD group demonstrated less favorable outcomes.

Comprehensive Clearing of Infection

In chronic wounds, infected tissue is one of the primary predictors of skin graft failure, impeding angiogenesis and the formulation of healthy granulation tissue.^{5,30,31} The importance of clearing infected tissue is emphasized by Golinko et al, who state that traditional clinical judgment in debridement is inadequate and often leaves pathologic tissue behind. They recommend debriding until deep tissue samples are pathologically negative.³² Golinko’s recommendation may not be clinically realistic to achieve, but our method of a more aggressive approach may serve as a reliable and effective clinical indicator during debridement of culture-positive wounds.

A less-aggressive debridement may risk leaving behind infected tissue, especially in wounds with known colonization. Over time, the presence of biofilms develop in 90% of chronic wounds, compared with 6% of acute wounds.³³ Biofilms thrive on chronic inflammation and the hypoxic

Table 4. Postoperative Complications and Long-term Outcomes

	Total, n (%) [N = 244 (100%)]	SD, n (%) [N = 158 (64.8%)]	DD, n(%) [N = 86 (35.3)]	P
Time to final follow-up (mo), median [IQR]	9 [23]	8 [25]	10.5 [18]	0.659
Graft failure	44 (18.0)	35 (22.2)	9 (10.5)	0.023*
Time to graft loss (d), mean ± SD	47.3 ± 23.7	48.8 ± 25.0	41.3 ± 17.5	0.404
Infection				
Anytime during follow-up	32 (13.1)	23 (14.6)	9 (10.5)	0.366
POV-30†	7 (3.0)	6 (4.0)	1 (1.2)	0.426
POV-60‡	16 (7.5)	11 (7.9)	5 (6.9)	0.791
Final follow-up§	14 (6.0)	11 (7.1)	3 (4.0)	0.558
Percentage healed (%), median [IQR]				
POV-30¶	80.1 [58.4]	84.7 [44.5]	75.4 [58.6]	0.264
POV-60	97.0 [36.1]	97.7 [32.5]	94.4 [38.0]	0.391
Final follow-up**	100.0 [1.3]	100.0 [2.3]	100.0 [0]	0.037*
Clinically healed				
POV-30††	87 (40.1)	58 (41.4)	29 (37.7)	0.588
POV-60‡‡	105 (50.7)	68 (49.3)	37 (53.6)	0.555
Final follow-up§§	153 (75.4)	93 (70.5)	60 (84.5)	0.027*
Reoperation at original wound site	84 (34.4)	62 (39.2)	22 (25.6)	0.032*

*Statistically significant ($P < 0.05$).

†POV-30 infection available for only 234 overall: 151 for the SD group, and 83 for the DD group.

‡POV-60 infection available for only 213 overall: 140 for the SD group, and 73 for the DD group.

§Final follow-up infection available for only 232 overall: 156 for the SD group, and 76 for the DD group.

¶POV-30 percentage healed available for only 160 overall: 95 for the SD group, and 65 for the DD group.

||POV-60 percentage healed available for only 166 overall: 107 for the SD group, and 59 for the DD group.

**Final follow-up percentage healed available for only 157 overall: 98 for the SD group, and 59 for the DD group.

††POV-30 healing available for only 217 overall: 140 for the SD group, and 77 for the DD group.

‡‡POV-60 healing available for only 207 overall: 138 for the SD group, and 69 for the DD group.

§§Final follow-up healing available for only 203 overall: 132 for the SD group, and 71 for the DD group.

POV, postoperative visit at 30 (-30) or 60 (-60) days.

Table 5. Multivariate Analysis Using Significant Univariate Covariates

Outcome Variable	Graft Loss, Any OR (95% CI)	P
Debridement method	0.0 (0.0–0.8)	0.034*
Wound length	0.5 (0.2–1.1)	0.083
Wound size	1.0 (0.9–1.1)	0.807
Wound surface		
Dorsal	1 [reference]	NA
Plantar	70.5 (2.1–2344.9)	0.017*
Wound depth		
Eschar	1 [reference]	NA
Dermis	0.3 (0.0–22.7)	0.611
Subcutaneous	3808.3 (4.5–3233340.0)	0.017*
Fascia	3.5 (0.0–458.7)	0.619
Muscle	3.8 (0.0–487.6)	0.590
Tendon	1 [omitted]†	NA
Post-debridement Cx MDR organism	1.0 (0.1–10.9)	0.982
History of CVA or TIA	2041.1 (9.8–423964.6)	0.005*

*Statistically significant ($P < 0.05$).

†Omitted due to insufficient sample size.

CI, confidence interval; Cx, culture; MDR, multi-drug resistant.

microenvironment of diabetic patients, which is optimal for facultative anaerobes, such as *P. aeruginosa*, to flourish in deeper tissues.^{34–36} Furthermore, chronic biofilms are able to evade conventional antibiotic treatment, yet remain vulnerable to environmental stress and physical damage.^{9,35,37} As such, a DD method may be able to disrupt deeper rooted and more pathogenic biofilm communities

that a superficial approach cannot. Although we did not observe strong correlation with debridement cultures and graft failure, this represents an avenue for further research using more advanced quantitative culturing techniques.^{38,39}

Our subgroup analysis of culture-negative and culture-positive wounds place our findings in the context of the wound bed's microenvironment. At baseline, culture-negative wounds have lower levels of infection and necrotic tissue but higher levels of healing factors.⁴⁰ As such, in these wounds, we did not observe a difference in STSG failure between SD and DD groups. However, in sub-analysis of culture-positive wounds, the DD method demonstrated significantly lower rates of failure. These results suggest that culture-positive wound beds may indicate an increased bacterial colonization that is better handled by a more aggressive debridement technique.¹⁸ Studies suggest that superficial wound swabs may fail to detect invasive biofilm infections due to their limited reach.^{41,42} If these swabs miss these infections, we cannot expect a superficial debridement technique to adequately eliminate deep infection, common in our study's multimorbid population. Taken together, while culture-negative wounds may not necessitate a deep debridement, culture-positive wounds may represent infection that cannot be effectively cleared by a superficial debridement only.

Risks of Deep Debridement

Because they do not carry their own blood supply, STSGs require a well-vascularized wound bed.² A primary

Table 6. Subgroup Analysis of Culture-negative and Culture-positive Postdebridement Wounds

Final Follow-up Outcomes	Culture-negative Wounds			
	Total, n (%) [N = 58 (100%)]	SD, n (%) [N = 36 (62.1%)]	DD, n (%) [N = 22 (37.9%)]	P
Graft failure	11 (19.0%)	8 (22.2%)	3 (13.6%)	0.507
Healed at final follow-up*	37 (75.5)	19 (65.5)	18 (90.0)	0.089
Postoperative infection	6 (10.3)	6 (16.7)	0 (0.0)	0.073
Final Follow-Up Outcomes	Culture-positive Wounds			
	Total, n (%) [N = 145 (100%)]	SD, n (%) [N = 91 (63.2%)]	DD, n (%) [N = 53 (36.8%)]	P
Graft failure	25 (17.4)	21 (22.1)	4 (7.6)	0.018 †
Healed at final follow-up‡	89 (72.7)	51 (66.2)	37 (84.1)	0.034 †
Postoperative infection	20 (13.9)	13 (14.3)	7 (13.2)	0.857

*Final follow-up healing available for only 49 overall: 29 for the SD group, and 20 for the DD group.

†Statistically significant ($P < 0.05$).

‡Final follow-up healing available for only 121 overall: 77 for the SD group, and 44 for the DD group.

concern with a radical debridement is damaging the wound bed by removing too much healthy tissue, thereby hindering the reepithelization process.^{2,29} However, our results suggest that this may not be the case when practiced by an experienced surgeon, as we observed that DD wounds achieve superior graft take. A deeper debridement may provide more mechanical stimulation that triggers a number of mechanotransduction pathways in the wound bed.^{23,43–45} Literature suggests mechanical activation promotes angiogenesis and reepithelization by releasing acute-pro-inflammatory cytokines that recruit keratinocytes and macrophages.^{45–51} As a result, the increased mechanical stimulation to a deeper level of tissue from DD may actually facilitate local neo-angiogenesis and microvascular endothelial cell proliferation.⁵¹

This point is further supported by our secondary outcomes in rates of healing, as we observed no significant differences in healing rates at POV-30 and POV-60 between the two groups. In fact, DD wounds demonstrated a higher rate of clinical healing at POV-60, suggesting that the healing potential of the wound bed is not compromised when using a more aggressive debridement method. Our sub-analysis demonstrates that even in culture-negative wound environments, extensive removal of granulation tissue did not result in higher complication rates. Furthermore, a significantly higher rate of SD wound healing was complicated by reoperation. In cases of graft failure, the wound must be further managed by our wound care team, which often includes the application of an SDS, further debridements, repeat STSG applications, or topical dressing management, all of which pose significant burdens to the individual and healthcare system. We observed the DD method to be a valuable tool to not only mitigate STSG failure but also reduce reoperation. Taken together, our data support that DD is safe and efficacious to perform in this comorbid population with atraumatic LE wounds.

Limitations

This study has several limitations. Its retrospective nature relies on the quality of data collected and the consistency of clinical documentation. Although we did not measure the depth of tissue removed during debridement, the white base layer was a consistent clinical marker to indicate differences between debridement types. Furthermore, photographic analysis or blind

observation for graft failure was not available to our study design. Although the patients were not randomized into treatments, all patients were treated by attendings at our wound clinic by standard institutional protocols and our results showed no significant differences in patient selection, as evidenced in patient demographics and wound characteristics. Graft outcomes are affected by patient adherence to postoperative immobilization protocols and wound locations that may be more susceptible to shearing forces or tendon movements.^{2,52} However, our univariate analysis did not demonstrate any wound location as covariates for graft failure. Despite these limitations, which predominantly arise from the retrospective study design, our research remains valuable because it provides a foundation for randomized prospective studies which we suggest to build upon and further validate our retrospective findings.

CONCLUSIONS

In managing chronic LE wounds, the preparation of an optimal wound bed is essential for the success of STSG reconstruction. Deep surgical debridement stands out as a technique that may not only eradicate persistent biofilm but also stimulate a wound environment conducive to graft healing. Our study highlights the importance of a thorough debridement and advocates for a more aggressive wound bed preparation in contaminated wounds to attain superior clinical results in a highly comorbid population.

Christopher E. Attinger, MD
 Georgetown University Hospital
 3800 Reservoir Road, NW
 Washington, DC 20007

E-mail: prsg Georgetownresearch@gmail.com

DISCLOSURE

The authors have no financial interest to declare in relation to the content of this article.

REFERENCES

- Attinger CE, Janis JE, Steinberg J, et al. Clinical approach to wounds: débridement and wound bed preparation including the use of dressings and wound-healing adjuvants. *Plast Reconstr Surg.* 2006;117(7S):72S–109S.

2. Braza ME, Fahrenkopf MP. Split-thickness skin grafts. *StatPearls*. Treasure Island, Fla.: StatPearls Publishing; 2023.
3. Donegan RJ, Schmidt BM, Blume PA. An overview of factors maximizing successful split-thickness skin grafting in diabetic wounds. Review. *Diabet Foot Ankle*. 2014;5:24769.
4. Edwards R, Harding KG. Bacteria and wound healing. *Curr Opin Infect Dis*. 2004;17:91–96.
5. Turissini JD, Elmarsafi T, Evans KK, et al. Major risk factors contributing to split thickness skin graft failure. *Georgetown Med Rev*. 2019;3.
6. Sieggreen MY, Maklebus J. Debridement: choices and challenges. *Adv Wound Care*. 1997;10:32–37.
7. Naz I, Walters ET, Janhofer DE, et al. Outcomes of split-thickness skin grafting for foot and ankle wounds in patients with peripheral arterial disease. *Wounds*. 2019;31:272–278.
8. Geethabanu S, Vanaja R. A study to analyse the influence of bacterial bio-burden on the success rate of split thickness skin grafting. *J Clin Diagn Res*. 2018;12:DC23–DC26.
9. Høgsberg T, Bjarnsholt T, Thomsen JS, et al. Success rate of split-thickness skin grafting of chronic venous leg ulcers depends on the presence of *Pseudomonas aeruginosa*: a retrospective study. *PLoS One*. 2011;6:e20492.
10. Nsafu K, Paintsil A, Dakubo J, et al. Evaluation of bacterial infection of split-thickness skin grafts at the Korle Bu Teaching Hospital. *Bali Med J*. 2020;9:259.
11. Unal S, Ersoz G, Demirkan F, et al. Analysis of skin-graft loss due to infection: infection-related graft loss. *Ann Plast Surg*. 2005;55:102–106.
12. Johnson TM, Ratner D, Nelson BR. Soft tissue reconstruction with skin grafting. *J Am Acad Dermatol*. 1992;27(2 Pt 1):151–165.
13. Stephenson AJ, Griffiths RW, La Hausse-Brown TP. Patterns of contraction in human full thickness skin grafts. *Br J Plast Surg*. 2000;53:397–402.
14. Vandembroucke JP, von Elm E, Altman DG, et al. Strengthening the reporting of observational studies in epidemiology (STROBE): explanation and elaboration. *Int J Surg*. 2014;12:1500–1524.
15. Eriksson E, Liu PY, Schultz GS, et al. Chronic wounds: treatment consensus. *Wound Repair Regen*. 2022;30:156–171.
16. CADTH Rapid Response Reports. *Debridement procedures for managing diabetic foot ulcers: a review of clinical effectiveness, cost-effectiveness, and guidelines*. Canadian Agency for Drugs and Technologies in Health; 2014.
17. Reddy S, El-Haddawi F, Fancourt M, et al. The incidence and risk factors for lower limb skin graft failure. *Dermatol Res Pract*. 2014;2014:582080.
18. Attinger CE, Bulan EJ. Débridement. The key initial first step in wound healing. *Foot Ankle Clin*. 2001;6:627–660.
19. Attinger CE, Bulan E, Blume PA. Surgical Débridement: the key to successful wound healing and reconstruction. *Clin Podiatr Med Surg*. 2000;17:599–630.
20. Attinger CE, Steinberg JS, Meyr AJ. Debridement of the diabetic foot. In: Armstrong DG, Lavery LA, eds. *Clinical Care of the Diabetic Foot*. 2nd ed. American Diabetes Association; 2010.
21. Diefenbeck M, Haustedt N, Schmidt HG. Surgical debridement to optimise wound conditions and healing. *Int Wound J*. 2013;10(Suppl 1):43–47.
22. Spampinato SF, Caruso GI, De Pasquale R, et al. The treatment of impaired wound healing in diabetes: looking among old drugs. *Pharmaceuticals (Basel)*. 2020;13:60.
23. Rose JF, Giovinco N, Mills JL, et al. Split-thickness skin grafting the high-risk diabetic foot. *J Vasc Surg*. 2014;59:1657–1663.
24. Yeong EK, Yu YC, Chan ZH, et al. Is artificial dermis an effective tool in the treatment of tendon-exposed wounds? *J Burn Care Res*. 2013;34:161–167.
25. Jiang ZY, Yu XT, Liao XC, et al. Negative-pressure wound therapy in skin grafts: a systematic review and meta-analysis of randomized controlled trials. *Burns*. 2021;47:747–755.
26. Leong S, Lo ZJ. Use of disposable negative pressure wound therapy on split-thickness skin graft recipient sites for peripheral arterial disease foot wounds: a case report. *Int Wound J*. 2020;17:716–721.
27. Mo R, Ma Z, Chen C, et al. Short- and long-term efficacy of negative-pressure wound therapy in split-thickness skin grafts: a retrospective study. *Ann Palliat Med*. 2021;10:2935–2947.
28. Moisisidis E, Heath T, Boorer C, et al. A prospective, blinded, randomized, controlled clinical trial of topical negative pressure use in skin grafting. *Plast Reconstr Surg*. 2004;114:917–922.
29. Gupta S. Optimal use of negative pressure wound therapy for skin grafts. *Int Wound J*. 2012;9(Suppl 1):40–47.
30. Yammine K, Assi C. A meta-analysis of the outcomes of split-thickness skin graft on diabetic leg and foot ulcers. *Int J Low Extrem Wounds*. 2019;18:23–30.
31. Manna B, Nahiriak P, Morrison CA. Wound debridement. *StatPearls*. Treasure Island, Fla.: StatPearls Publishing; 2023.
32. Golinko MS, Joffe R, Maggi J, et al. Operative debridement of diabetic foot ulcers. *J Am Coll Surg*. 2008;207:e1–e6.
33. Attinger C, Wolcott R. Clinically addressing biofilm in chronic wounds. *Adv Wound Care (New Rochelle)*. 2012;1:127–132.
34. Lebeaux D, Ghigo JM, Beloin C. Biofilm-related infections: bridging the gap between clinical management and fundamental aspects of recalcitrance toward antibiotics. *Microbiol Mol Biol Rev*. 2014;78:510–543.
35. Donlan RM, Costerton JW. Biofilms: survival mechanisms of clinically relevant microorganisms. *Clin Microbiol Rev*. 2002;15:167–193.
36. Schulze A, Mitterer F, Pombo JP, et al. Biofilms by bacterial human pathogens: Clinical relevance—development, composition and regulation—therapeutical strategies. *Microb Cell*. 2021;8:28–56.
37. Goswami AG, Basu S, Banerjee T, et al. Biofilm and wound healing: from bench to bedside. *Eur J Med Res*. 2023;28:157.
38. Johnson AC, Buchanan EP, Khechoyan DY. Wound infection: a review of qualitative and quantitative assessment modalities. *J Plast Reconstr Aesthet Surg*. 2022;75:1287–1296.
39. Kallstrom G. Are quantitative bacterial wound cultures useful? *J Clin Microbiol*. 2014;52:2753–2756.
40. Halim AS, Khoo TL, Saad AZ. Wound bed preparation from a clinical perspective. *Indian J Plast Surg*. 2012;45:193–202.
41. Li S, Renick P, Senkowsky J, et al. Diagnostics for wound infections. *Adv Wound Care (New Rochelle)*. 2021;10:317–327.
42. Wu YK, Cheng NC, Cheng CM. Biofilms in chronic wounds: pathogenesis and diagnosis. *Trends Biotechnol*. 2019;37:505–517.
43. Oh S, Chung H, Chang S, et al. Effect of mechanical stretch on the DNCB-induced proinflammatory cytokine secretion in human keratinocytes. *Sci Rep*. 2019;9:5156.
44. Erba P, Ogawa R, Ackermann M, et al. Angiogenesis in wounds treated by microdeformational wound therapy. *Ann Surg*. 2011;253:402–409.
45. Rousselle P, Braye F, Dayan G. Re-epithelialization of adult skin wounds: cellular mechanisms and therapeutic strategies. *Adv Drug Deliv Rev*. 2019;146:344–365.
46. Xie F, Wen G, Sun W, et al. Mechanical stress promotes angiogenesis through fibroblast exosomes. *Biochem Biophys Res Commun*. 2020;533:346–353.
47. Sharifpanah F, Behr S, Wartenberg M, et al. Mechanical strain stimulates vasculogenesis and expression of angiogenesis guidance molecules of embryonic stem cells through elevation of intracellular calcium, reactive oxygen species and nitric oxide generation. *Biochim Biophys Acta*. 2016;1863:3096–3105.
48. Liu C, Cui X, Ackermann TM, et al. Osteoblast-derived paracrine factors regulate angiogenesis in response to mechanical stimulation. *Integr Biol (Camb)*. 2016;8:785–794.

49. Hughes GC, Biswas SS, Yin B, et al. A comparison of mechanical and laser transmyocardial revascularization for induction of angiogenesis and arteriogenesis in chronically ischemic myocardium. *J Am Coll Cardiol.* 2002;39:1220–1228.
50. Geris L, Vandamme K, Naert I, et al. Mechanical loading affects angiogenesis and osteogenesis in an in vivo bone chamber: a modeling study. *Tissue Eng Part A.* 2010;16:3353–3361.
51. Liu C, Lu Y, Du P, et al. Mesenchymal stem cells pretreated with proinflammatory cytokines accelerate skin wound healing by promoting macrophages migration and M2 polarization. *Regen Ther.* 2022;21:192–200.
52. Chang K-P, Tsai C-C, Lin T-M, et al. An alternative dressing for skin graft immobilization: negative pressure dressing. *Burns.* 2001;27:839–842.