Wound Bed Preparation - The Challenges of Wound Debridement and Cleansing

Dot Weir, RN, CWON, CWS
Buffalo, New York

Nov. 17, 2017 for HealthTrust Members

Disclosures

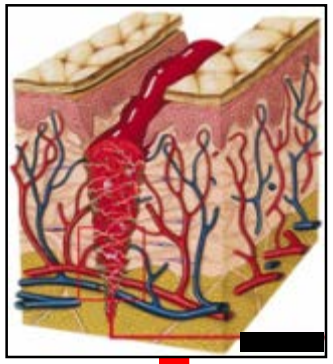
Company	Speaker's Bureau	Consulting / Advisory Board
Smith & Nephew Biotherapeutics	X	X
Organogenesis	X	
Hollister	X	
Lohmann & Rauscher	X	
Molnlycke	X	X
Acelity	X	
Medline		X

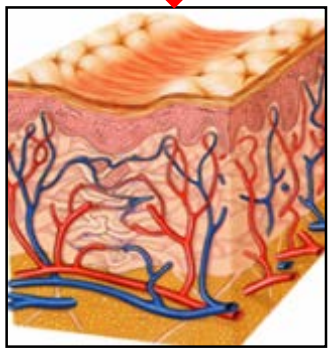
Learning Objectives

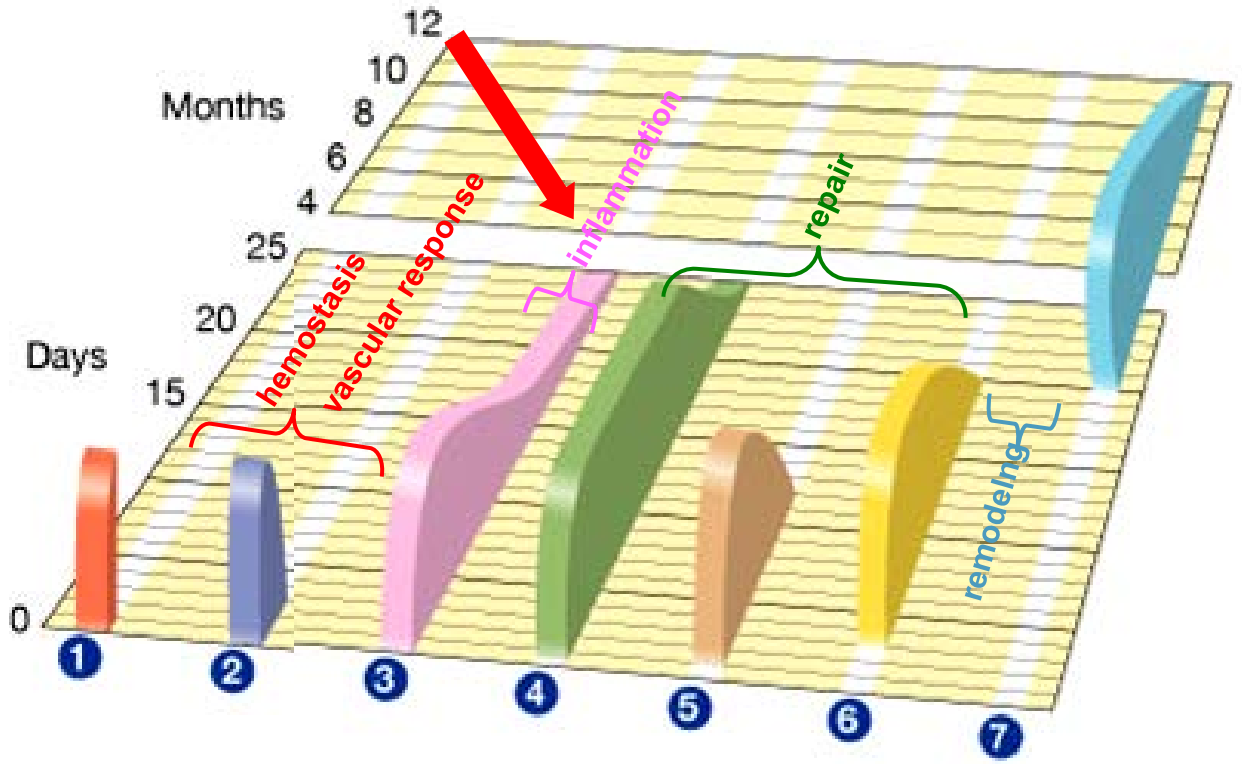
At the end of this session participants should be able to…

1. Explain wound bed preparation, challenges with biofilm and other factors preventing healing
2. Describe effective methods for debridement
3. Evaluate different wound care methodologies for the best possible patient outcome

Sequence of Molecular and Cellular Events in Skin Wound Healing







4 Phases of Healing

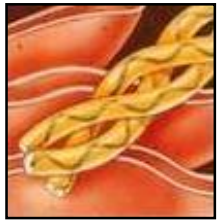
1. Hemostasis
2. Inflammation
3. Repair
4. Remodeling

1 Clotting

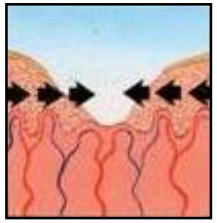
2 Vascular Response

3 Inflammation

4 Scar Formation

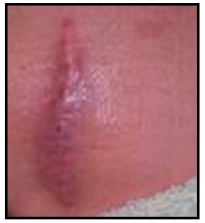


5 Epithelial Healing

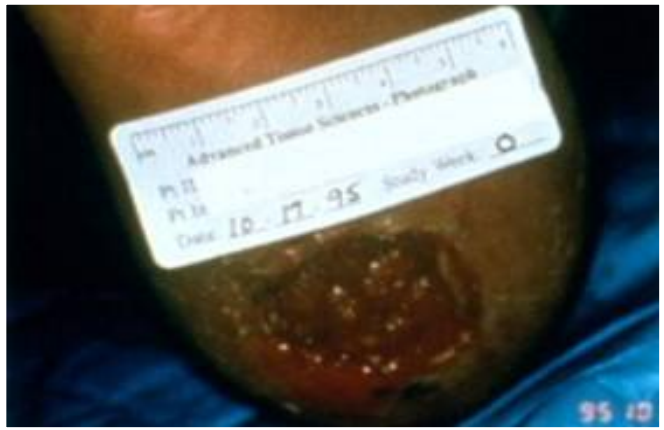


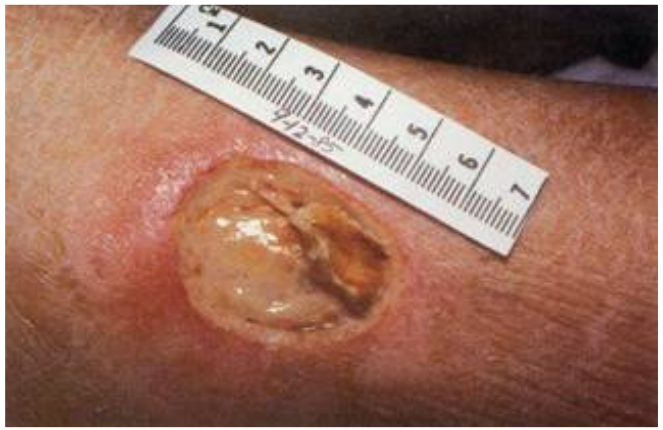
6 Contraction

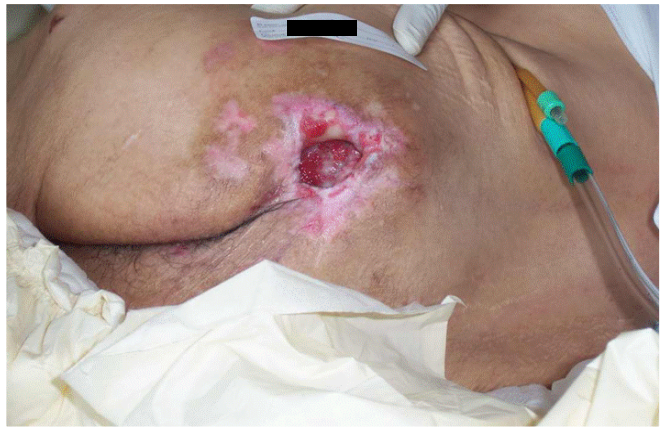
7 Scar Remodeling

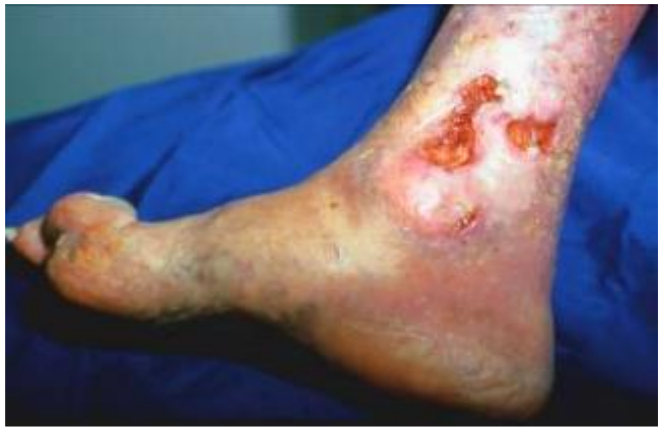


Is There a Common Molecular Pathology of Chronic Wounds?





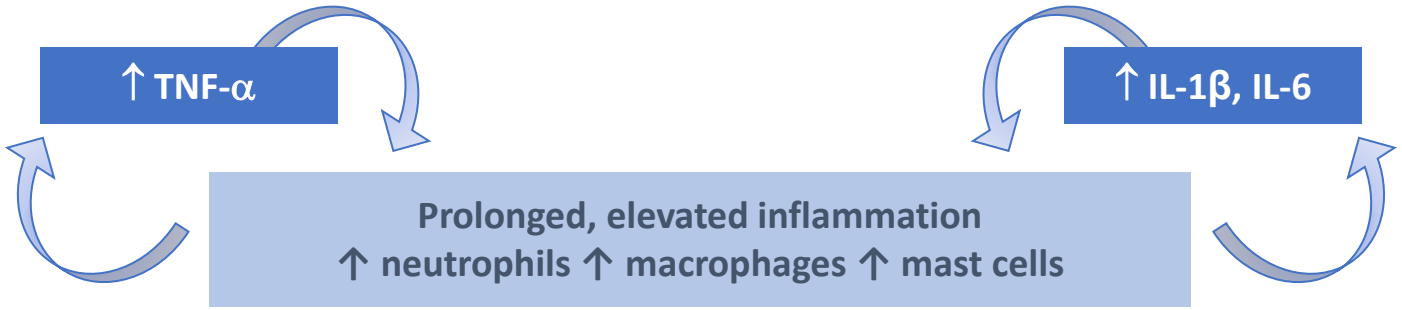




Hypothesis of Chronic Wound Pathophysiology

Repeated Tissue Injury, Ischemia, and Bioburden – Planktonic and Biofilms

- ECM = extracellular matrix; IL = interleukin; MMP = matrix metalloproteinase; TIMP = tissue inhibitor of metalloproteinase; TNF-α = tumor necrosis factor-alpha; ROS = reactive oxygen species



Mast BA, et al. Wound Repair Regen. 1996;4(4):411-420

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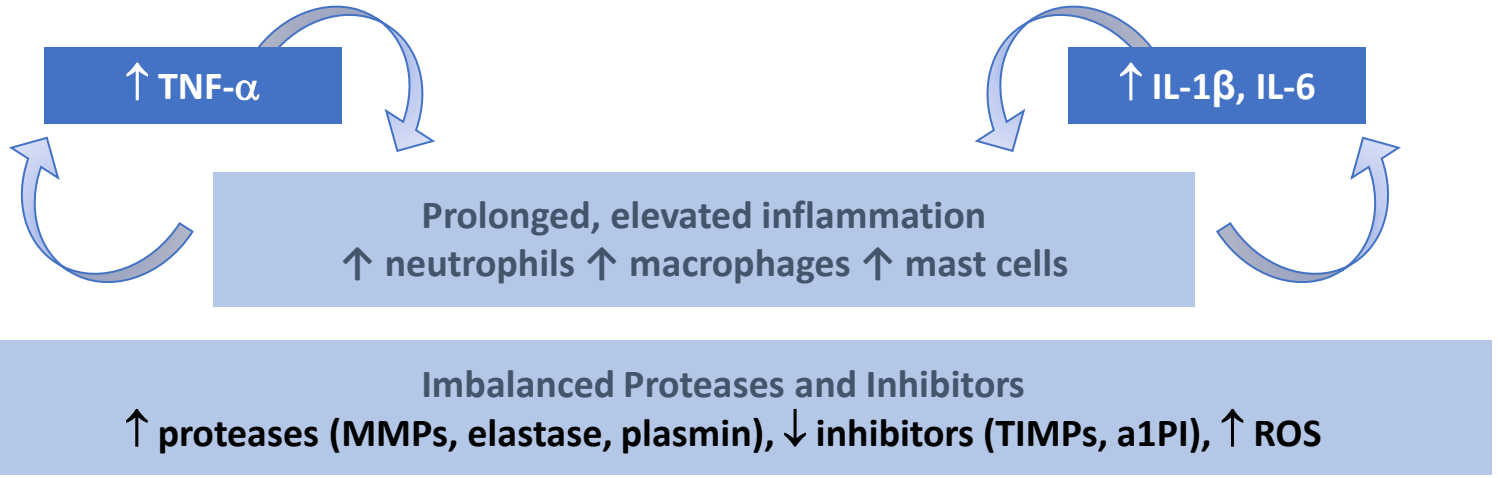

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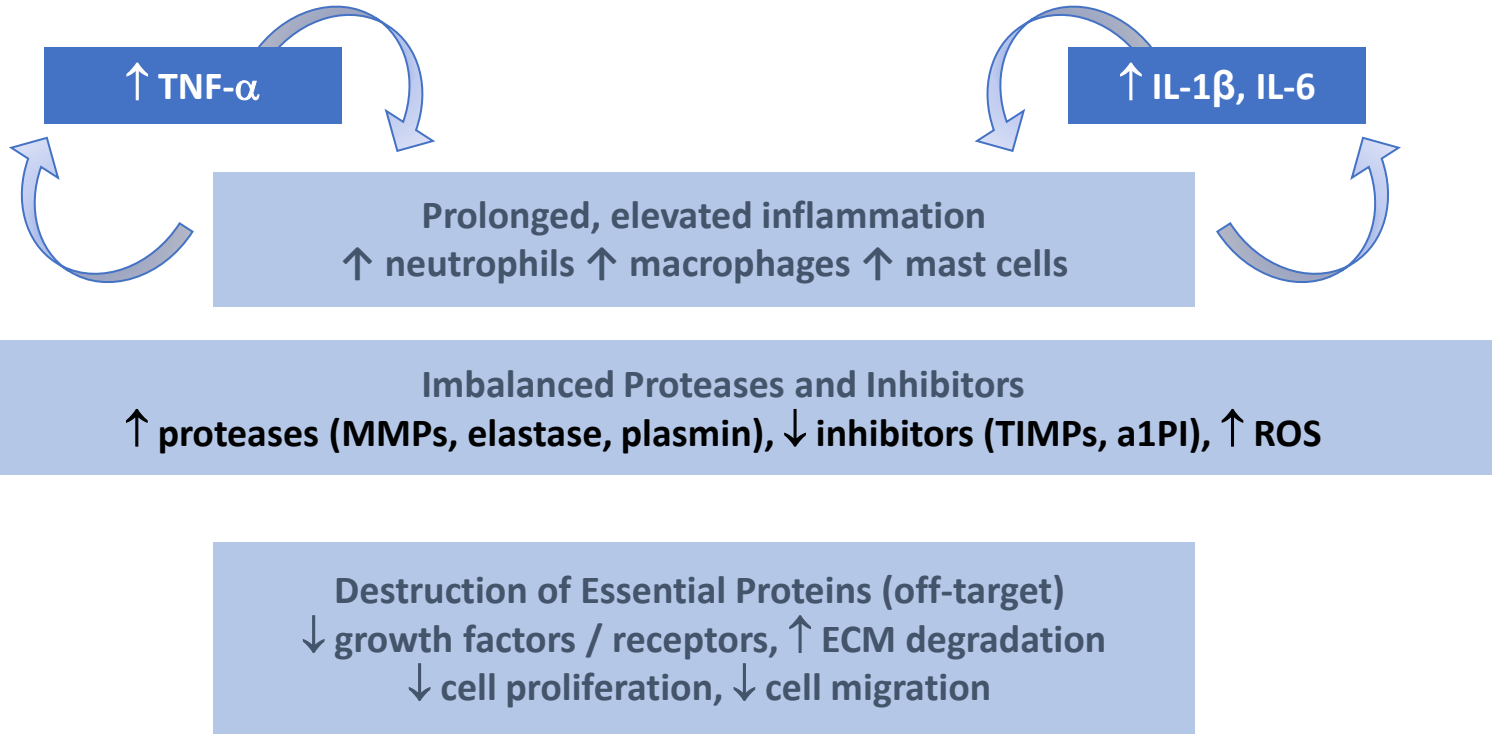

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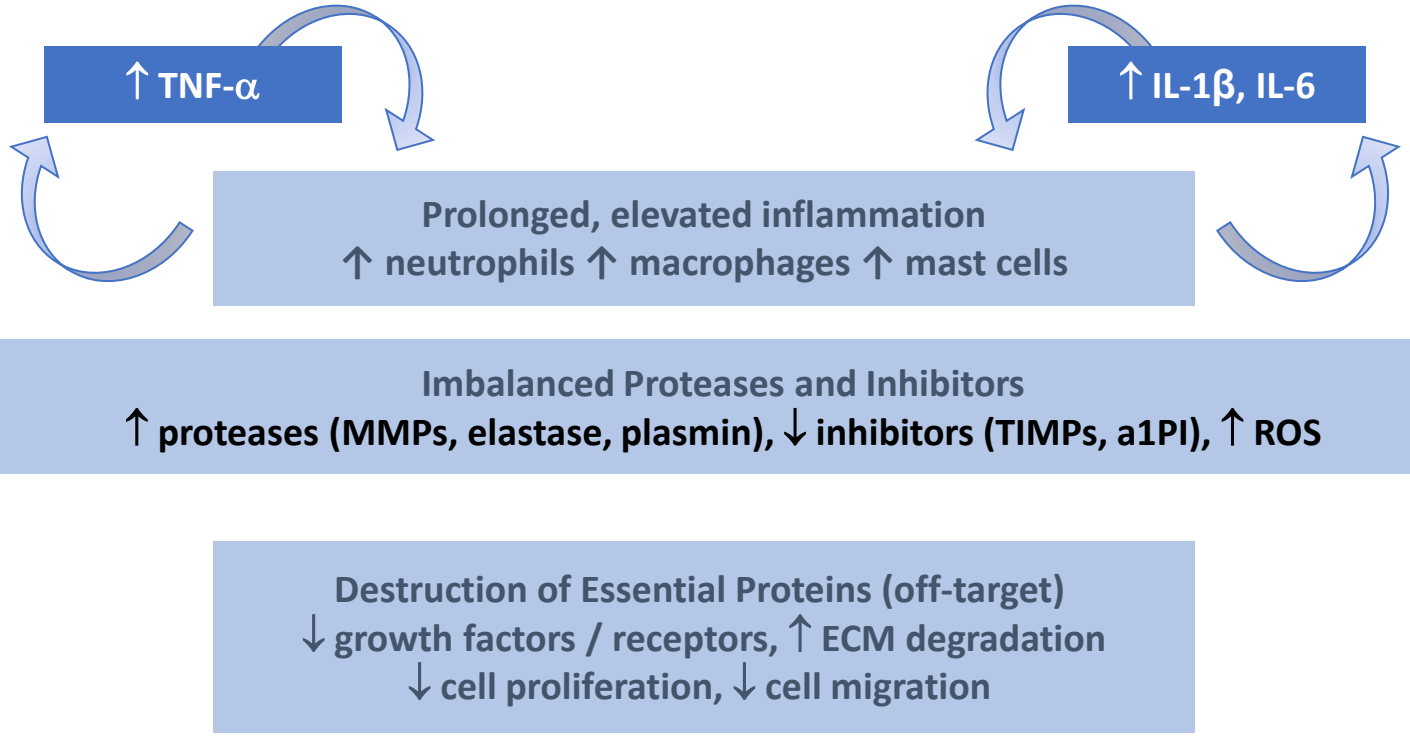

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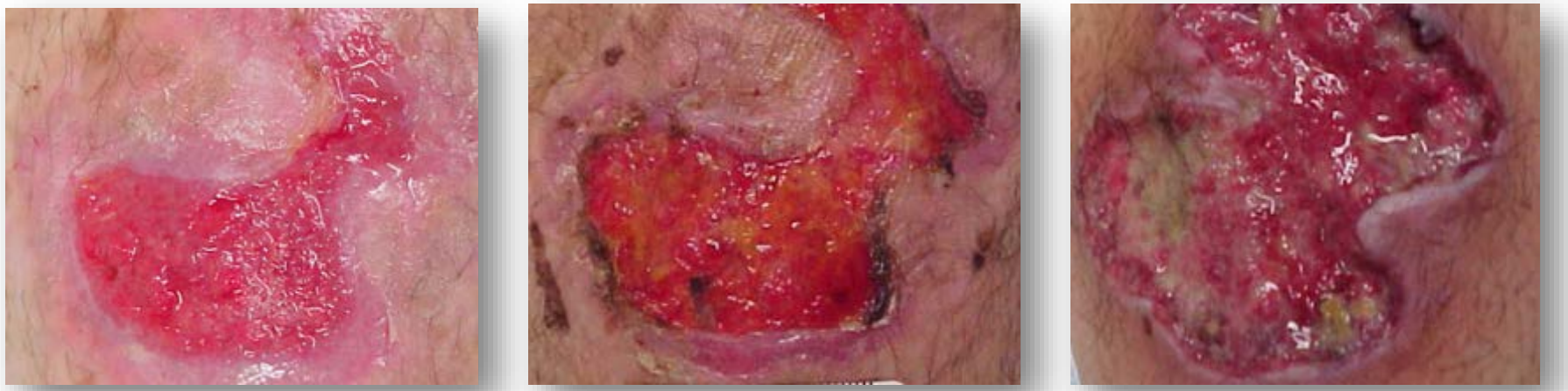
Acute Wound ⇒ Chronic Non-Healing Wound

Antimicrobial Stewardship: Judicious Use of Antimicrobial Dressings



Contamination	Colonization	Critical Colonization / Localized Infection	Spreading Infection	Systemic Infection

Topical antimicrobial dressings are not indicated because bioburden is not causing clinical problems

Must address the *bacteria*

Combined systemic antibiotics and topical antimicrobial dressings indicated

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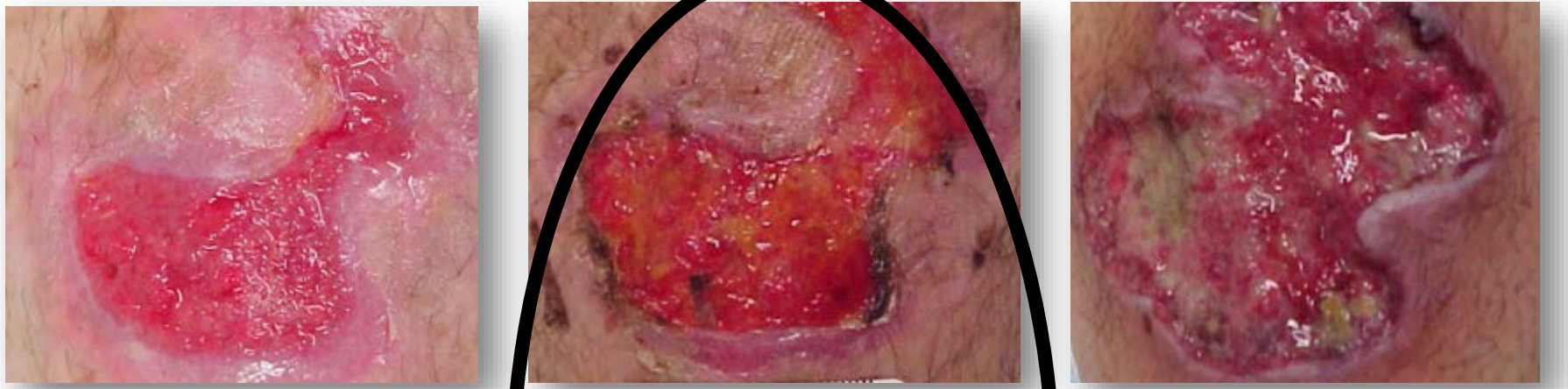

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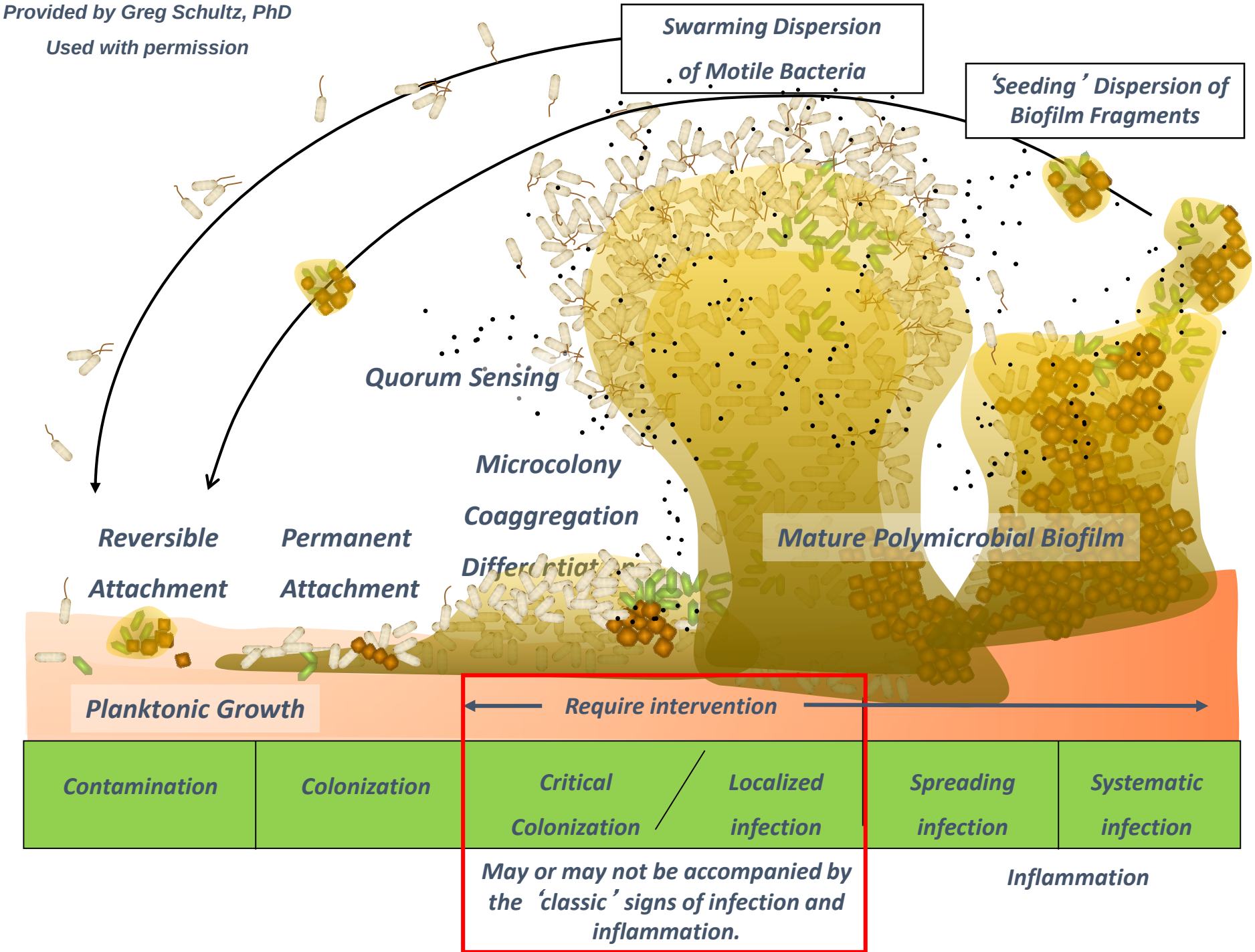
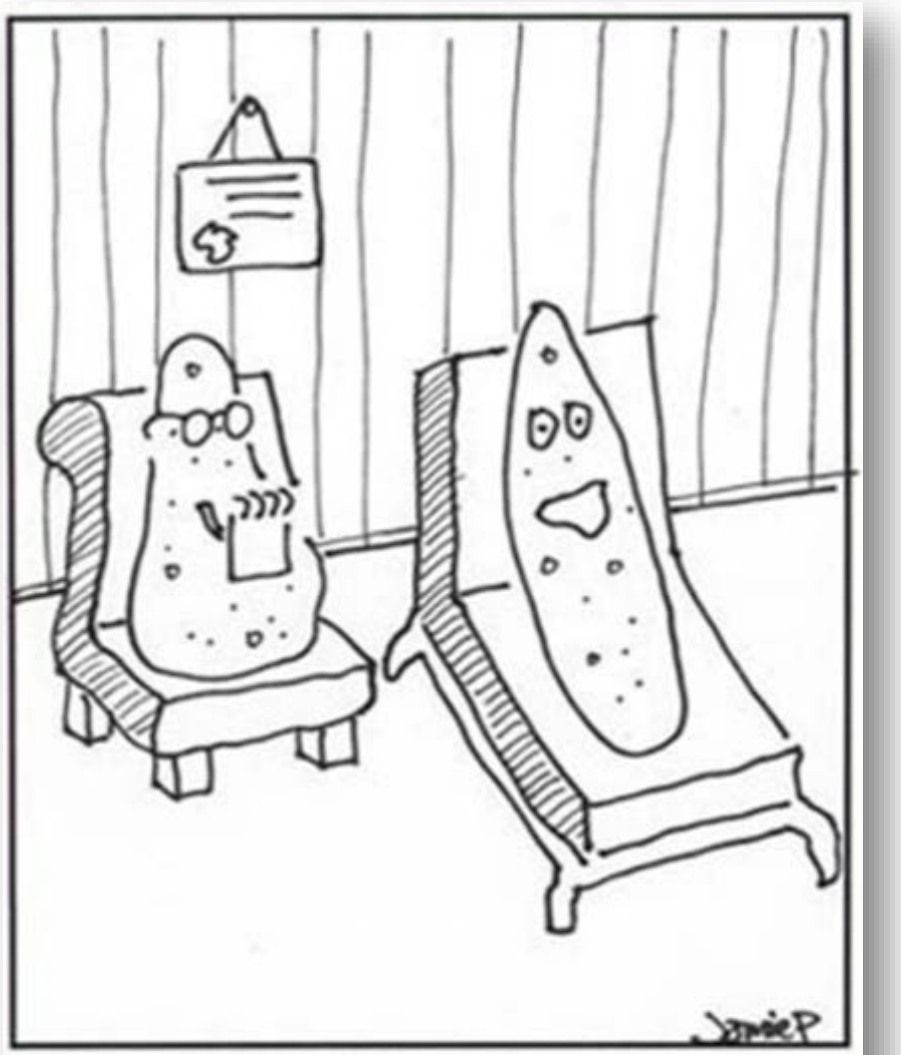
Provided by Greg Schultz, PhD

Used with permission



Bacteria are getting smarter…we must also.



biofilms made easy

Wounds

Introduction

This article describes what biofilms are and the important roles they appear to play in disrupting wound healing. In addition, it discusses potential interventions aimed at removing/reducing biofilms and preventing their reformation in wounds.

Authors: Phillips PL, Wolcott RD, Fletcher J, Schultz GS
Full author details can be found on page 5.

What are biofilms?

Biofilms are complex microbial communities containing bacteria and fungi. The microorganisms synthesise and secrete a protective matrix that attaches the biofilm firmly to a living or non-living surface[1].

Biofilms are dynamic heterogeneous communities that are continuously changing[2]. They may consist of a single bacterial or fungal species, or more commonly, may be polymicrobial, ie contain multiple diverse species[3,4]. At the most basic level a biofilm can be described as bacteria embedded in a thick, slimy barrier of sugars and proteins. The biofilm barrier protects the microorganisms from external threats.

How are biofilms relevant to wounds?

Biofilms have long been known to form on surfaces of medical devices, such as urinary catheters, endotracheal and tympanostomy tubes, orthopaedic and breast implants, contact lenses, intrauterine devices (IUDs) and sutures[5,6]. They are a major contributor to diseases that are characterised by an underlying bacterial infection and chronic inflammation, eg periodontal disease, cystic fibrosis, chronic acne and osteomyelitis[2,5,7].

Biofilms are also found in wounds and are suspected to delay healing in some. Electron microscopy of biopsies from chronic wounds found that 60% of the specimens contained biofilm structures in comparison with only 6% of biopsies from acute wounds[8]. Since biofilms are reported to be a major factor contributing to multiple chronic inflammatory diseases, it is likely that almost all chronic wounds have biofilm communities on at least part of the wound bed.

How do biofilms form?

Stage one: reversible surface attachment

Microorganisms are commonly perceived to be free-floating and solitary (ie planktonic). However, under natural conditions most microorganisms tend to attach to surfaces and eventually form biofilms[1,9] (Figure 1). The initial attachment is reversible.

Stage two: permanent surface attachment

As the bacteria multiply, they become more firmly attached (sessile) and differentiate, changing gene expression patterns in ways that promote survival[9,10]. This is usually the result of a type of bacterial communication known as quorum sensing[10].

Stage three: slimy protective matrix/biofilm

Once firmly attached, the bacteria begin to secrete a surrounding matrix known as extracellular polymeric substance (EPS)[11]. This is a protective matrix or 'slime'. Small bacterial colonies then form an initial biofilm[3,9].

The exact composition of EPS varies according to the micro-organisms present, but generally consists of polysaccharides, proteins, glycolipids and bacterial DNA[2,10,11]. Bacterial DNA released by living or dead bacteria is thought to provide an important structural component for biofilm EPS matrix[10]. Various secreted proteins and enzymes help the biofilm to become firmly attached the wound bed[9].

Fully mature biofilms continuously shed planktonic bacteria, microcolonies and fragments of biofilm, which can disperse and attach to other parts of the wound bed or to other wounds, forming new biofilm colonies[2,9].

Figure 1. Schematic representation of polymicrobial biofilm formation (adapted from [9])

Free download from *Wounds International*

Phillips PL, et al.
Biofilms Made Easy.
Wounds International.
2010;1(3):1-6.
www.woundsinternational.com/media/issues/288/files/content_8851.pdf.
Accessed March 28, 2017.

Biofilm in a Nutshell

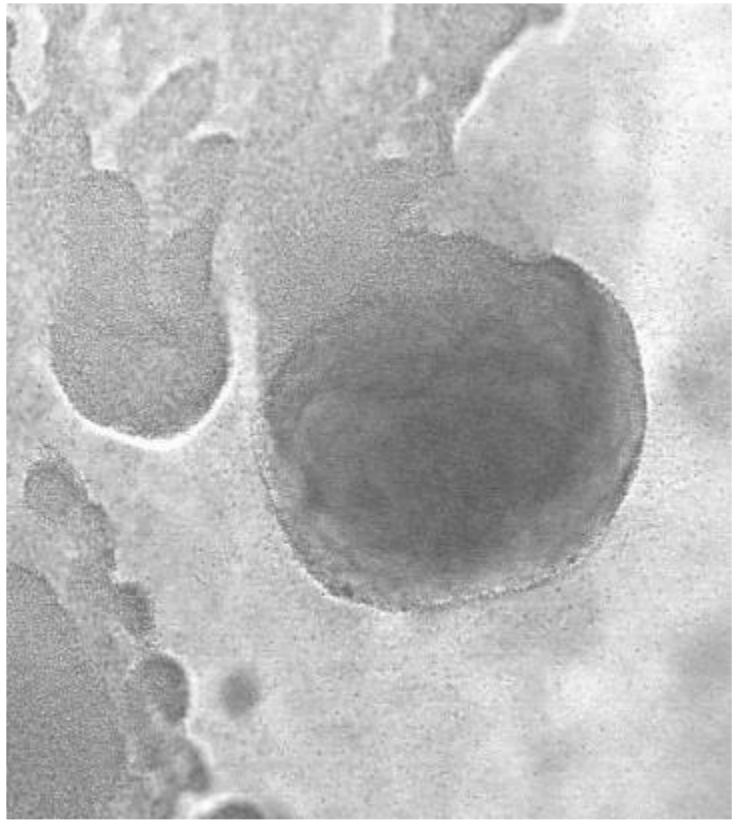
- Multiple species of bacteria and fungi
  - Gm + and -, aerobes and anaerobes
- Exudes film of extracellular polymeric substances (EPSs) composed of proteins, lipids, and polysaccharides.
  - The components of mature biofilm are approximately 5–25% bacterial cells and 75–95% glycocalyx matrix.
- Can begin to form within 2 hours, and reform rapidly after removal

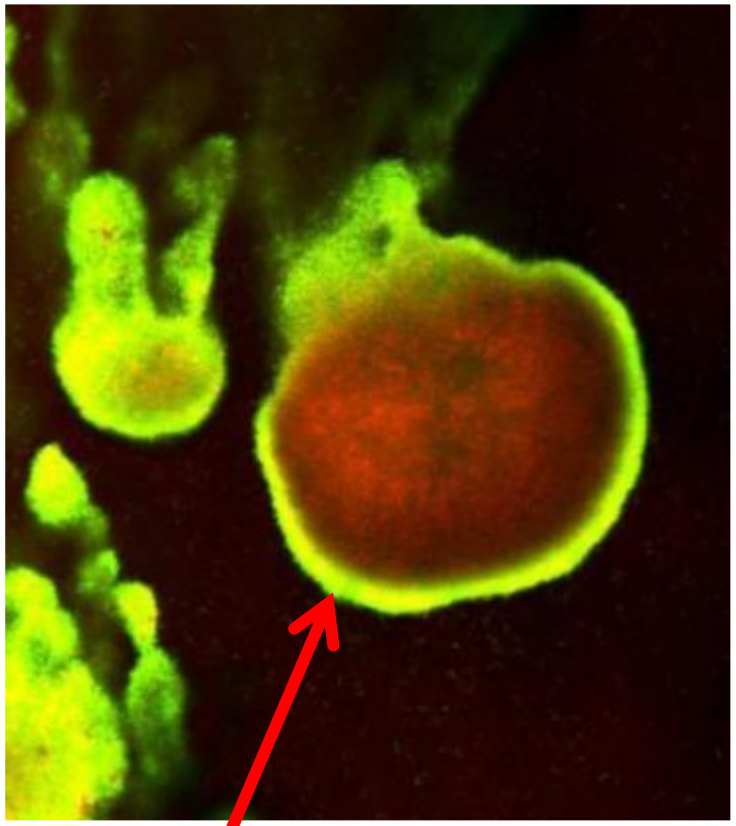
Why are Bacteria in Biofilms Hard to Kill?

- Exopolymeric material of the biofilm
  - Dense matrix impairs diffusion of large antibodies
  - EPM materials chemically react (neutralize) microbicides
  - Negative charges of polysaccharides and DNA bind cationic molecules like Ag+, antibiotics, PHMB+
- Persister bacteria have low metabolic activity
  - Metabolic needs are less
  - Antibiotics only kill metabolically active
- Oxygen diffusion to center of biofilm is limited
  - Promotes growth of anaerobic bacteria
- Synergism between different bacteria
  - Quorum sensing

Slide courtesy G. Schultz, PhD

Metabolic Activity of *P. aeruginosa* in Mature Biofilms is Limited to the Surface Layers

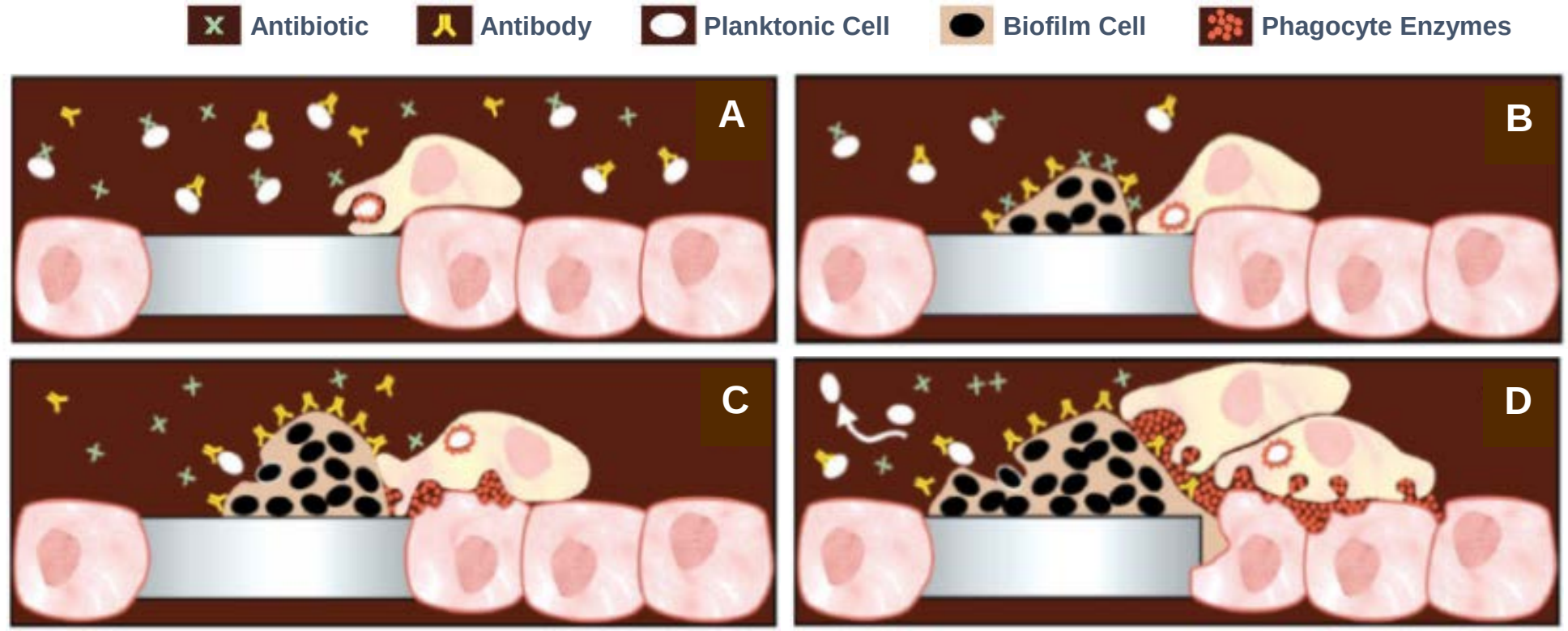




– Only fluorescent bacteria are metabolically active
– Only located in outer layers of the biofilm matrix
– **Antibiotics only kill metabolically active bacteria**

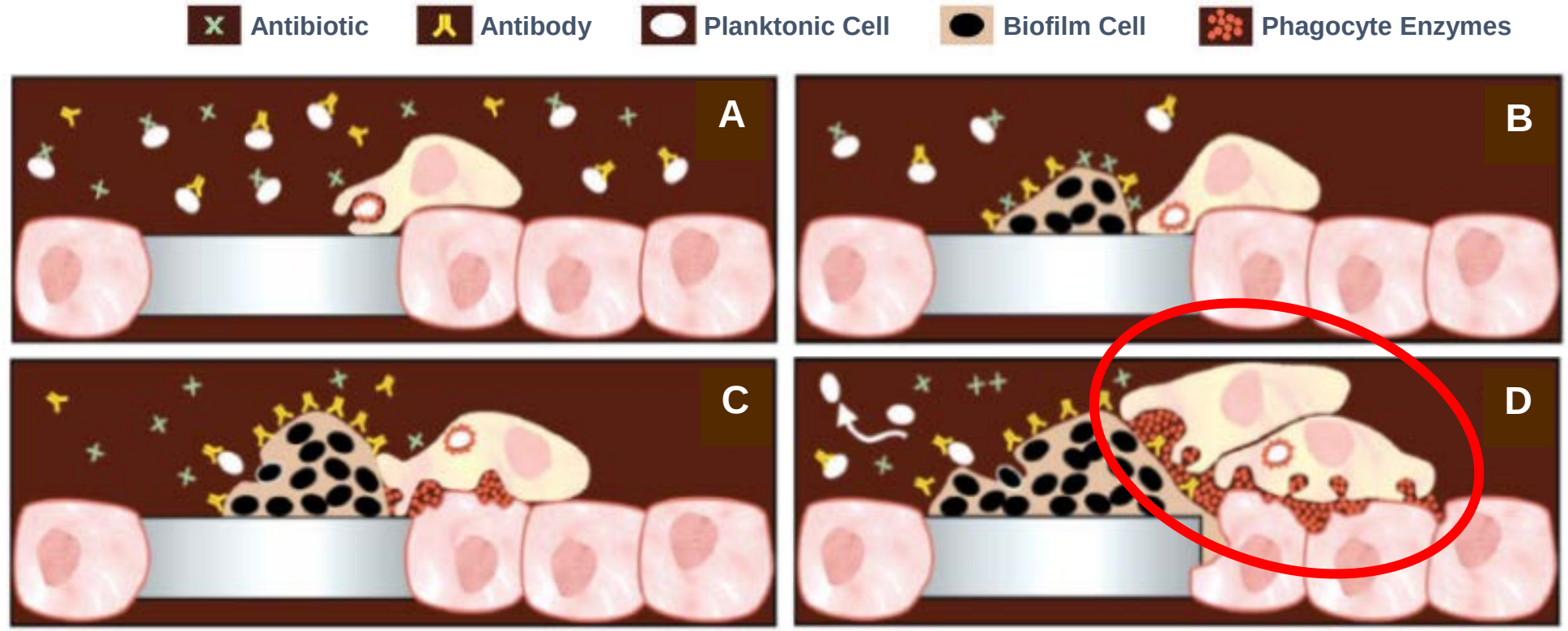
Phil Stewart, Montana State University Center for Biofilm Engineering.

How Does the Immunological Response to Biofilms Cause Tissue Damage and Impair Healing?



In **Panel A**, planktonic bacteria can be cleared by antibodies, phagocytosis, and are susceptible to antibiotics. Adherent bacterial cells **(Panel B)** form biofilms preferentially on inert surfaces or devitalized tissue, and these sessile communities are resistant to antibodies, phagocytosis and antibiotics. Neutrophils **(Panel C)** are attracted to the biofilms, but cannot engulf biofilm. Neutrophils still release proteases and reactive oxygen species. Phagocytic enzymes **(Panel D)** damage tissue around the biofilm, and planktonic bacteria are released from the biofilm, causing dissemination and acute infection in neighboring tissue.

How Does the Immunological Response to Biofilms Cause Tissue Damage and Impair Healing?



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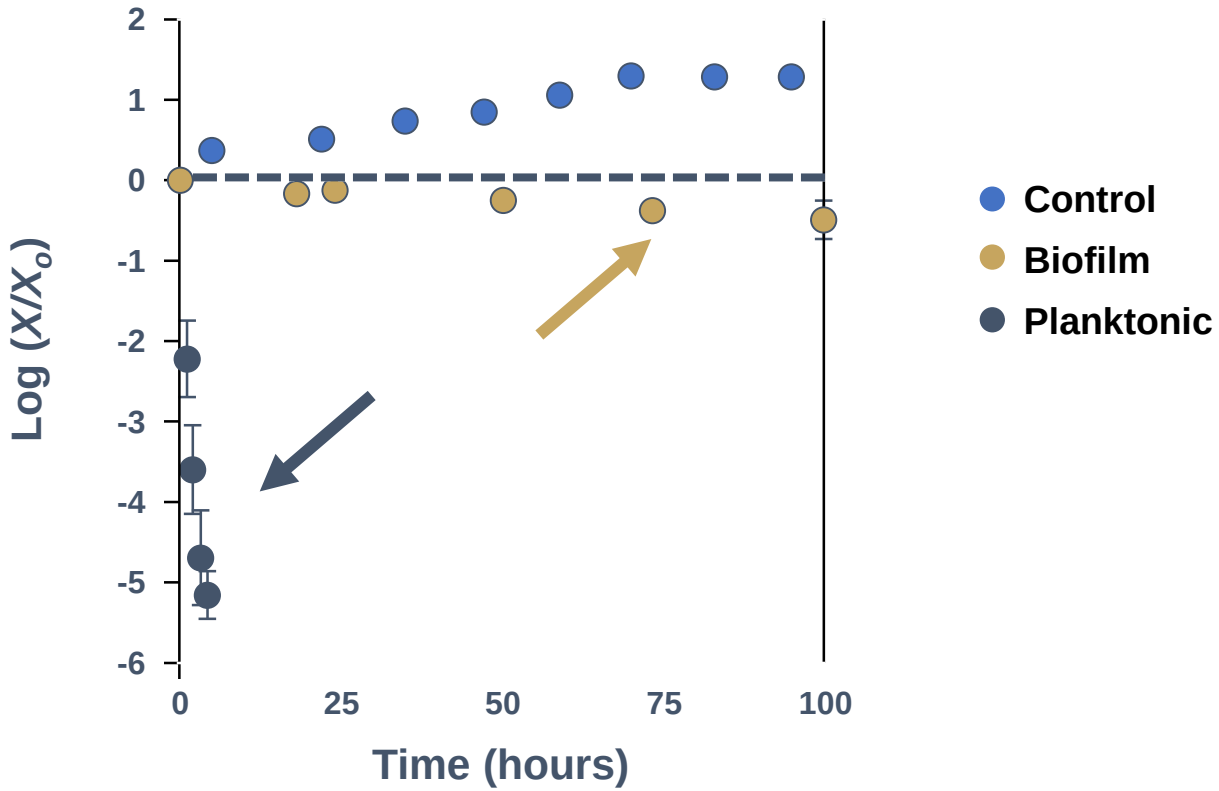
Biofilms are Highly Tolerant to Antibiotics

Tobramycin vs *P. aeruginosa* Biofilm



Tobramycin rapidly kills planktonic *Pseudomonas aeruginosa* (●) very effectively, but is not effective against biofilm (●).

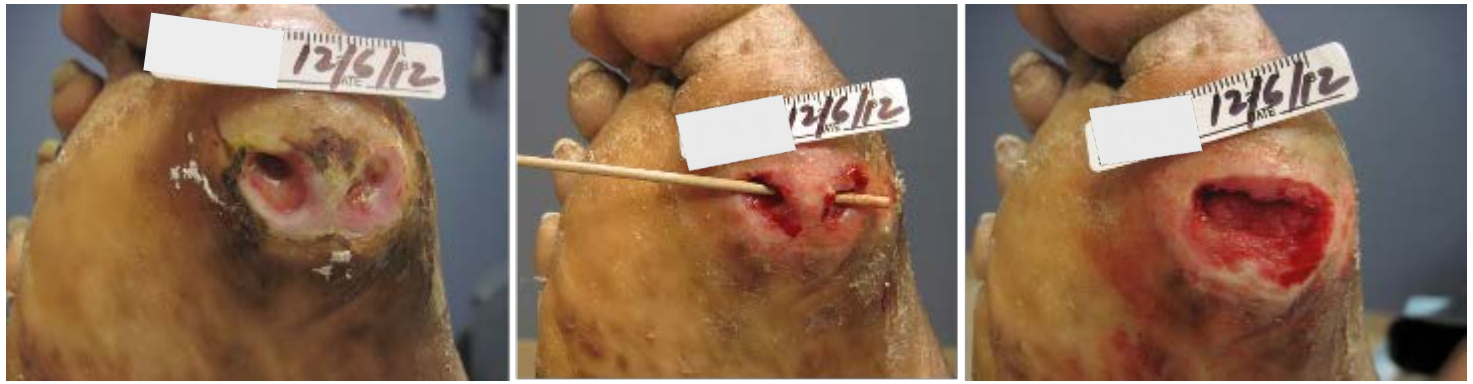
Walters MC 3rd, et al. *Antimicrob Agents Chemother.* 2003;47(1):317-323.

The Challenge of Diagnosis

- Remains a clinical observation versus a microbiological test
  - Persistent non-healing
  - Slough formation in well vascularized wounds
  - Reported observation of sheen, yellow gelatinous appearance but early biofilm is not visible
- Testing methods not readily available to average practitioner
  - PCR, SEM
  - MICs not helpful in treatment decisions
    - MBEC (Minimum Biofilm Eradication Concentration) reported but not available
- POC testing difficult to bring to clinical use
  - Blot test

- Percival, S. L., Hill, K. E., Malic, S., Thomas, D. W. and Williams, D. W. (2011), Antimicrobial tolerance and the significance of persister cells in recalcitrant chronic wound biofilms. Wound Repair and Regeneration, 19: 1–9. doi:10.1111/j.1524-475X.2010.00651.x

Management



- Cold hard steel (sharp debridement!)
  - Followed by antimicrobial dressings
- Monofilament pads
  - Followed by antimicrobial dressings
- Cadexomer Iodine / Iodine PVA foam dressings
- Anti-biofilm gels and agents

dotweir@aol.com

2003: A Concept Published: TIME

WOUND REPAIR AND REGENERATION
THE INTERNATIONAL JOURNAL OF TISSUE REPAIR AND REGENERATION

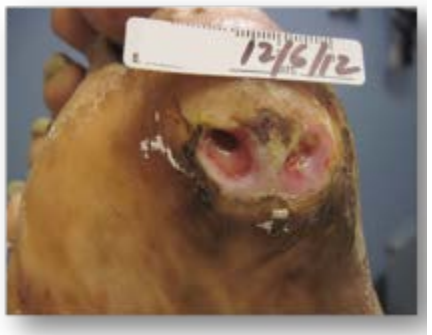
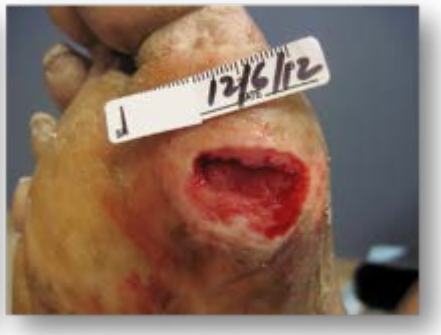
Wound bed preparation: a systematic approach to wound management

GREGORY S. SCHULTZ, PhD[1,]; R. GARY SIBBALD, MD[2,*]; VINCENT FALANGA, MD[3,*]; ELIZABETH A. AYELLO, PhD[4]; CAROLINE DOWSETT[5]; KEITH HARDING, MB, ChB[6]; MARCO ROMANELLI, MD, PhD[7]; MICHAEL C. STACEY, DS[8]; LUC TEOT, MD, PhD[9]; WOLFGANG VANSCHEIDT, MD[10]*

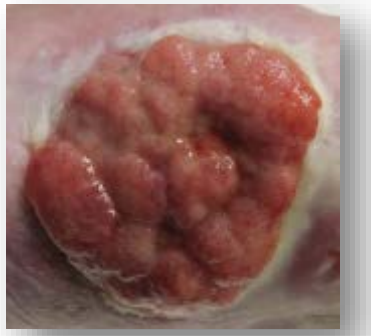
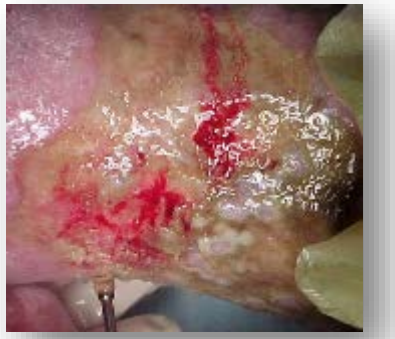
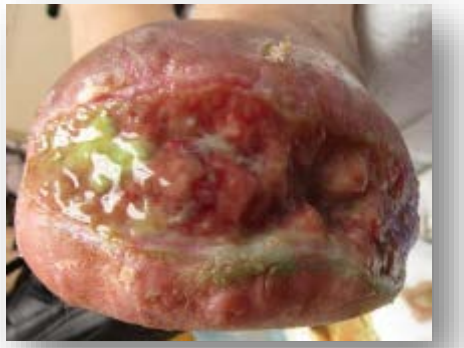
Schultz GS, Sibbald RG, Falanga V, et al. Wound bed preparation: a systematic approach to wound management. Wound Repair Regen 2003; 11(2): Suppl S1-28

Wound Bed Preparation: TIME

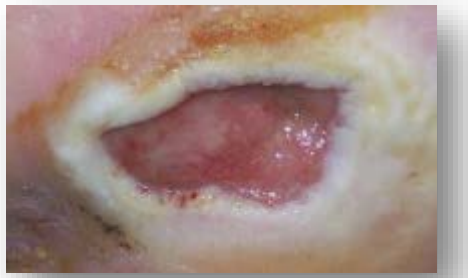
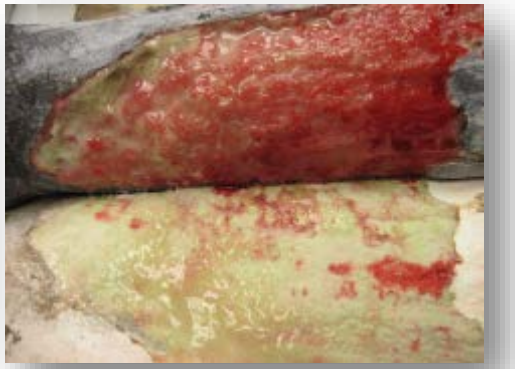
- ***T**issue (debridement)*

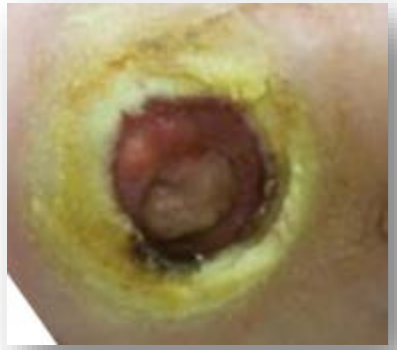
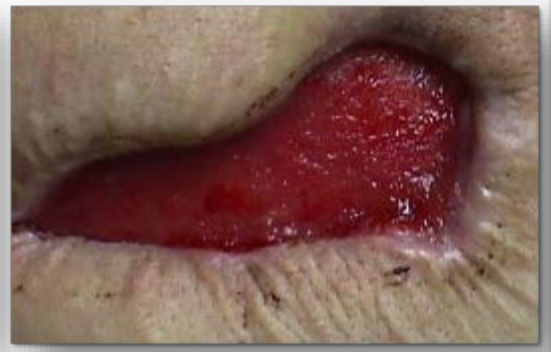
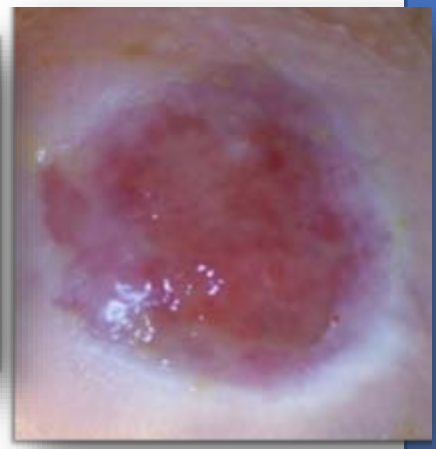
- ***I**nfection/inflammation/biofilm*

- ***M**oisture balance*

- ***E**dge of the wound*

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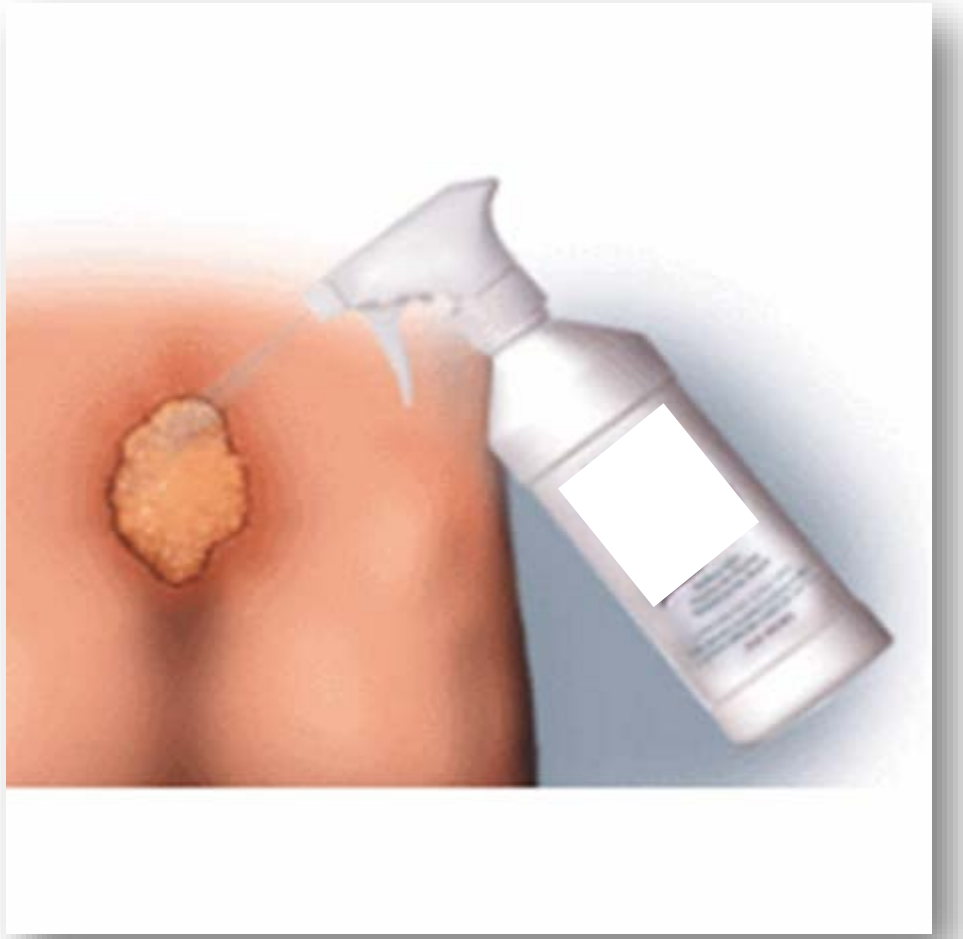

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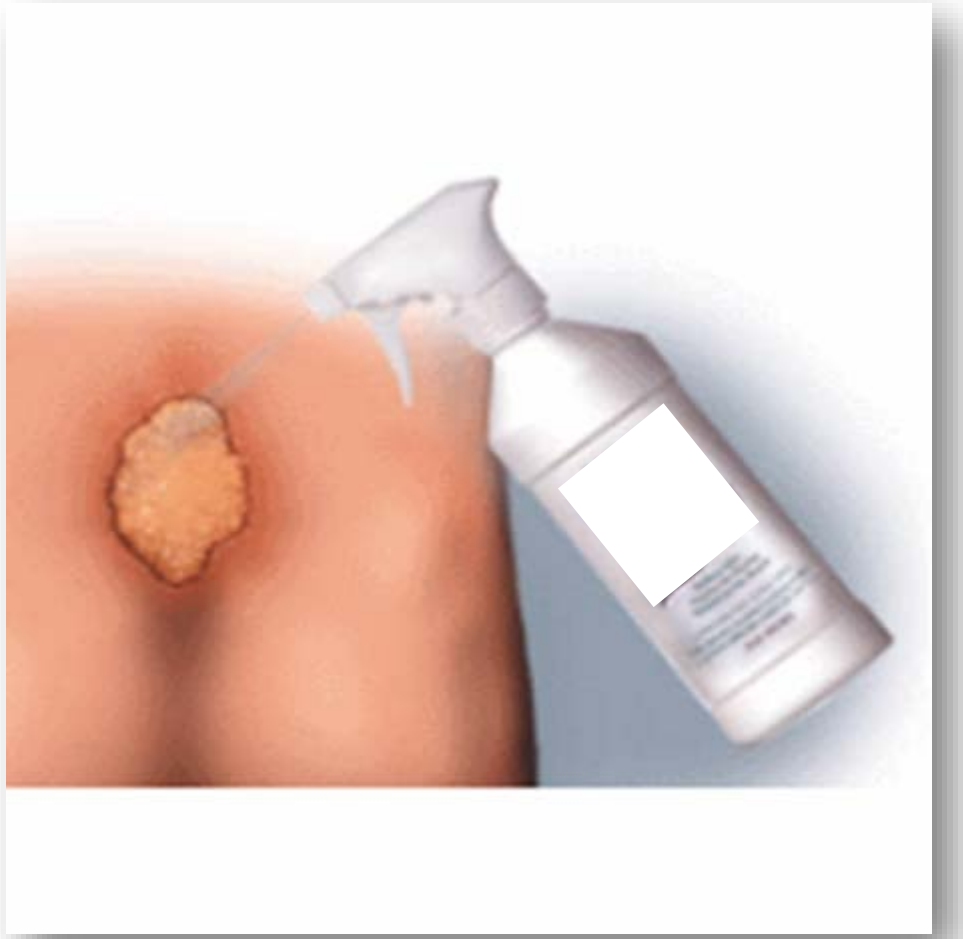

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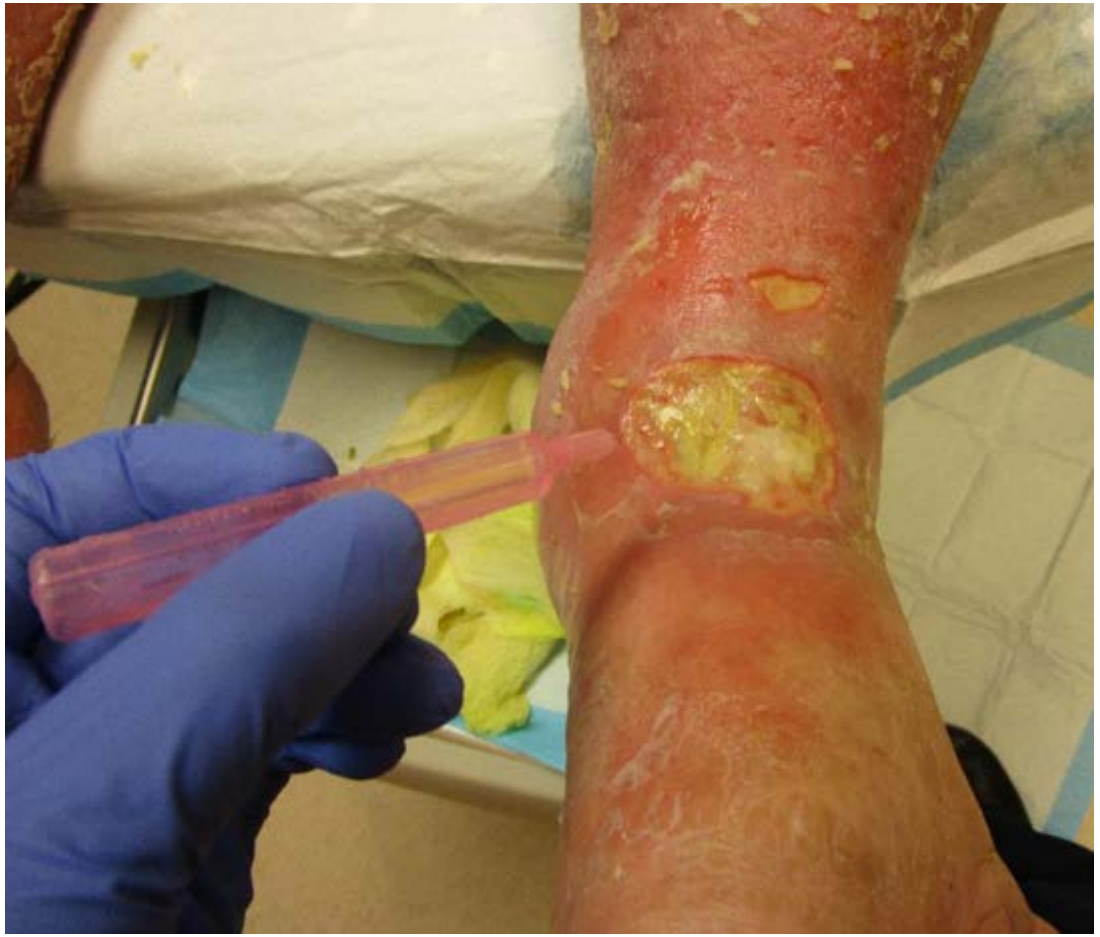

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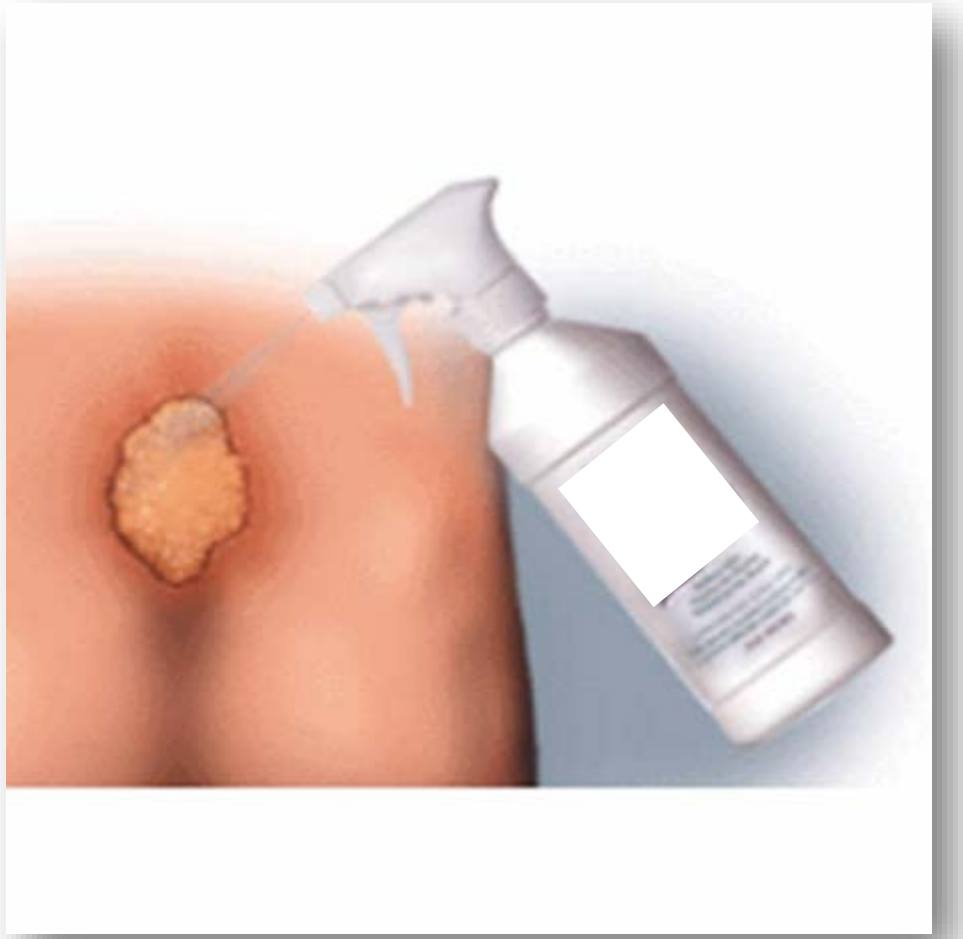
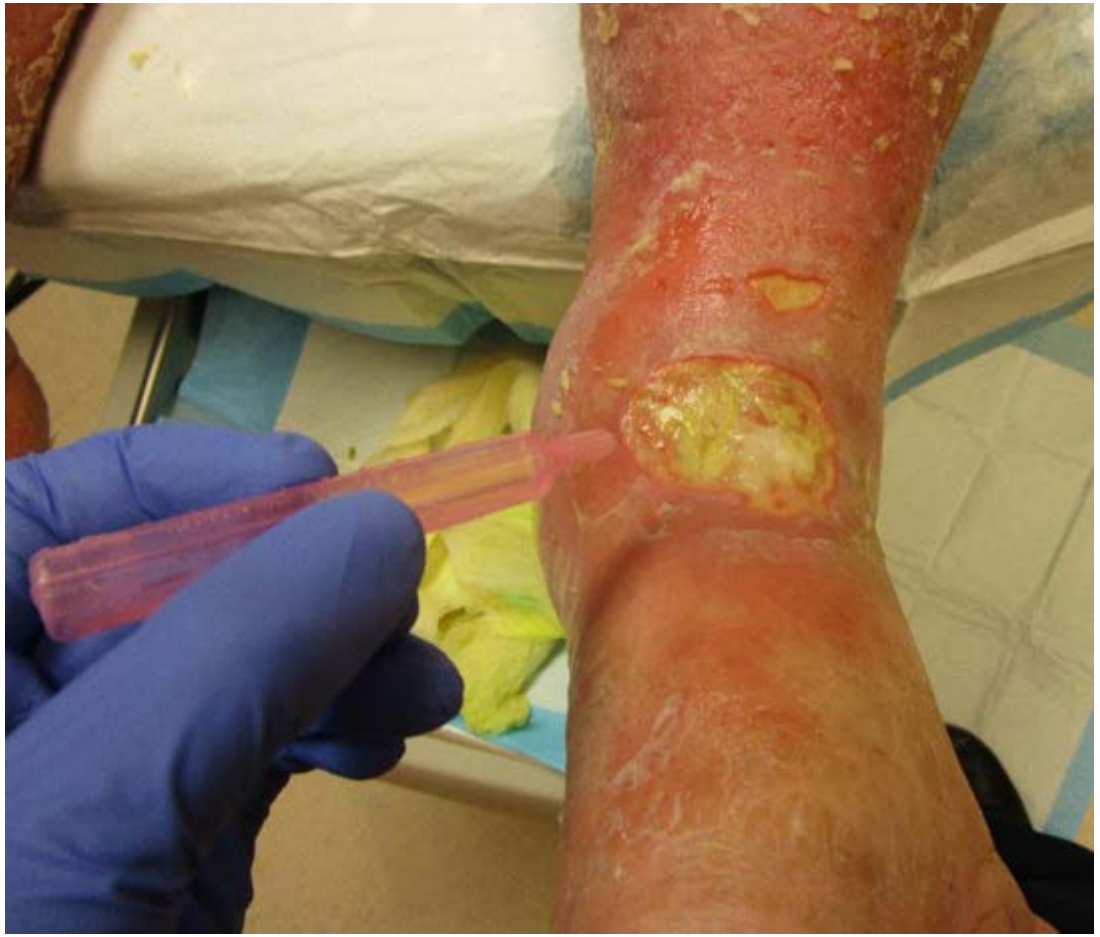
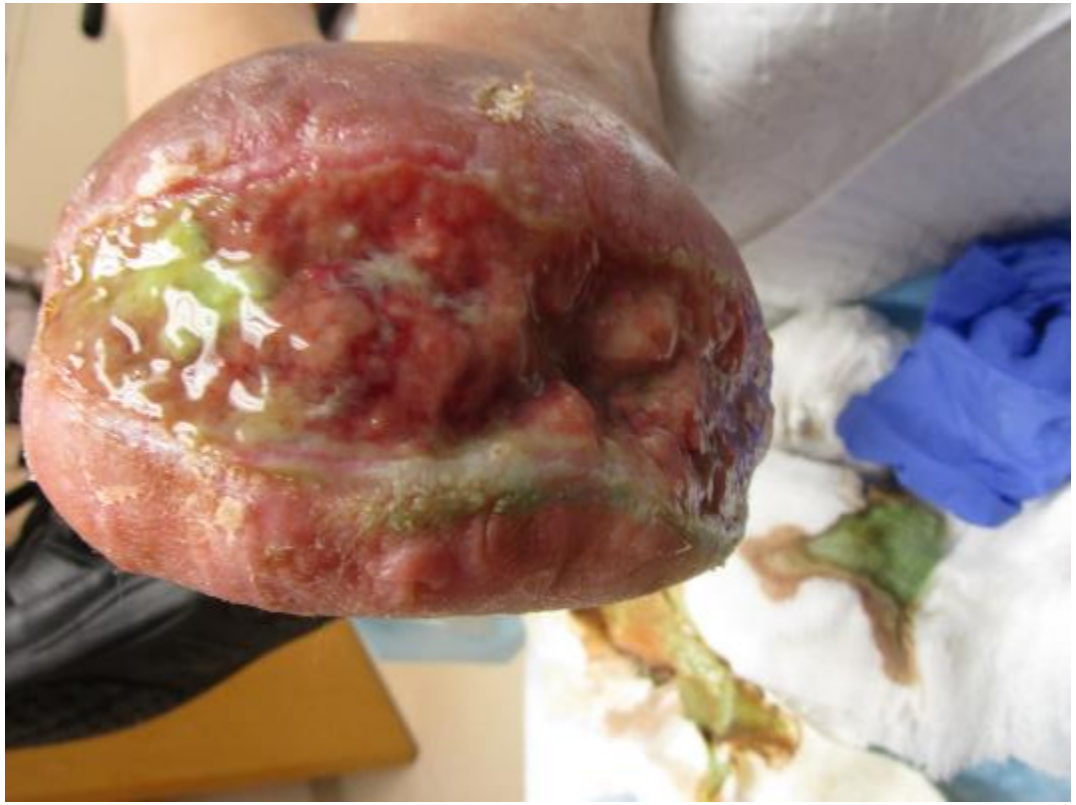


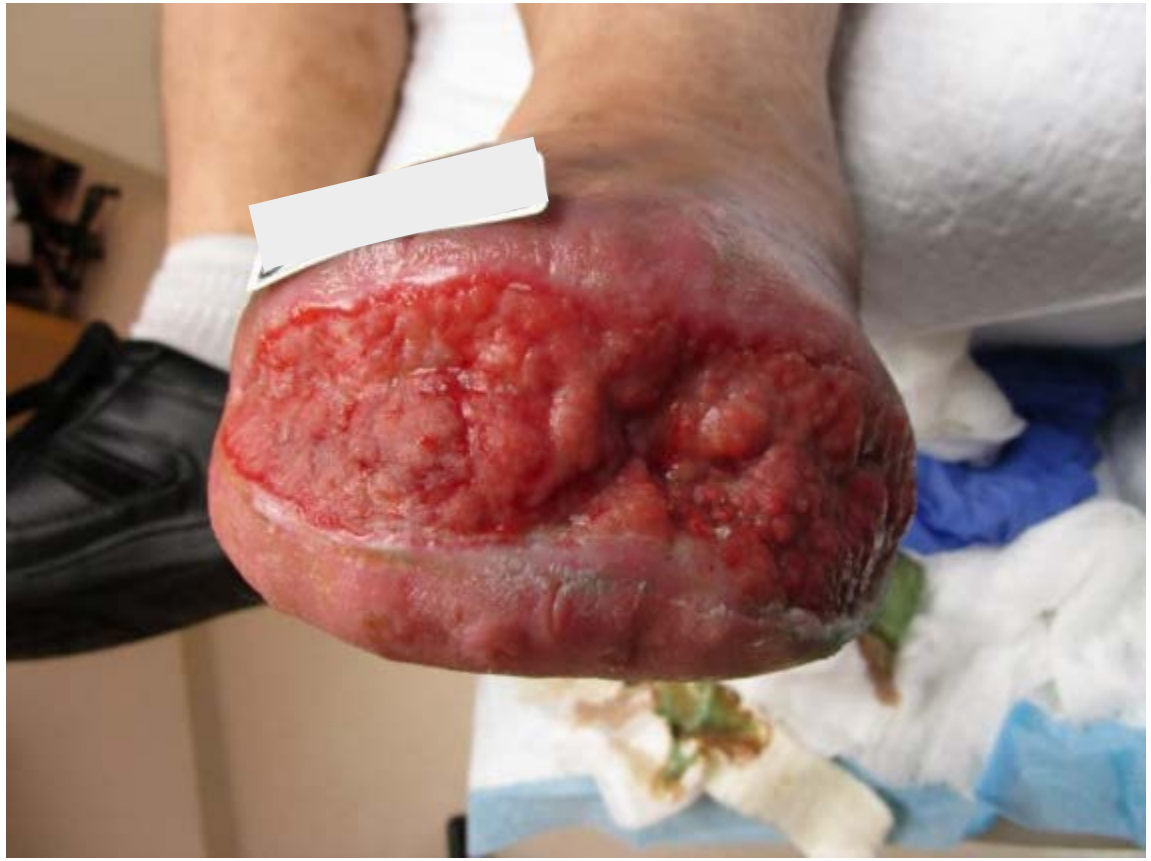
Not Anoint!



Clean it like you mean it!

Wound Cleansing Essential





dotweir@aol.com

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- Endogenous = invasion of indigenous microflora through any disruption in the body surface
  - Any rupture of the skin integument favors the development of infection
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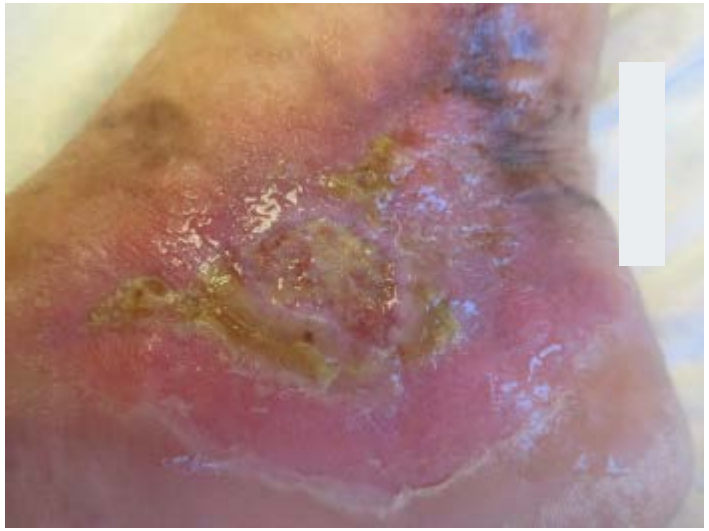

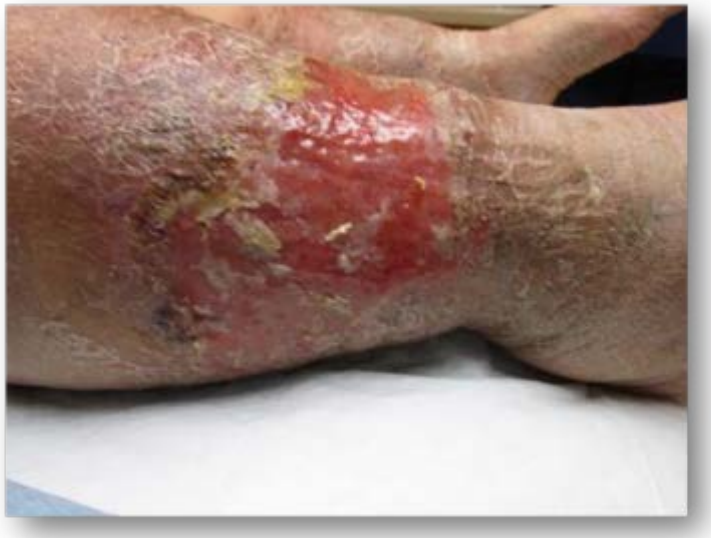

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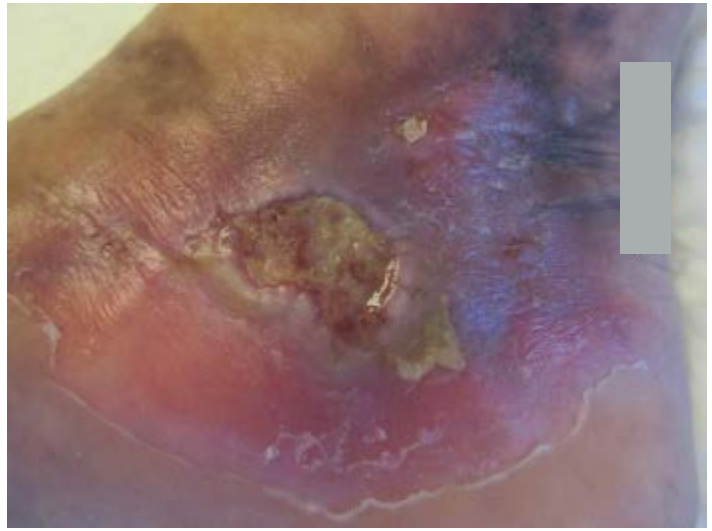

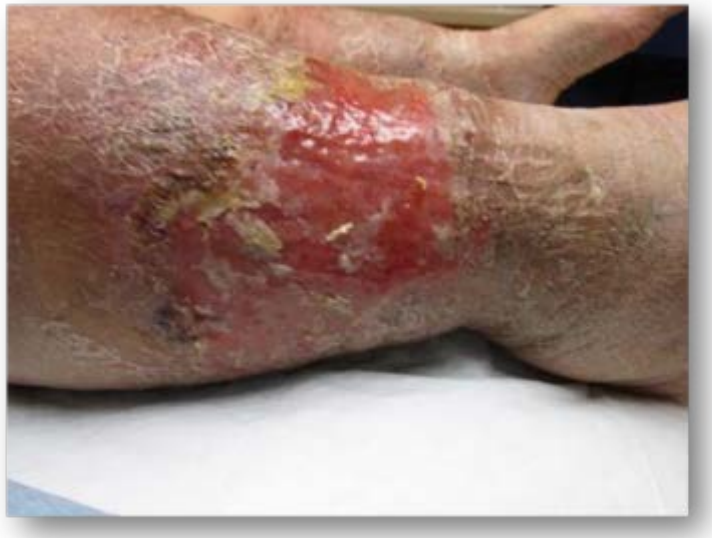

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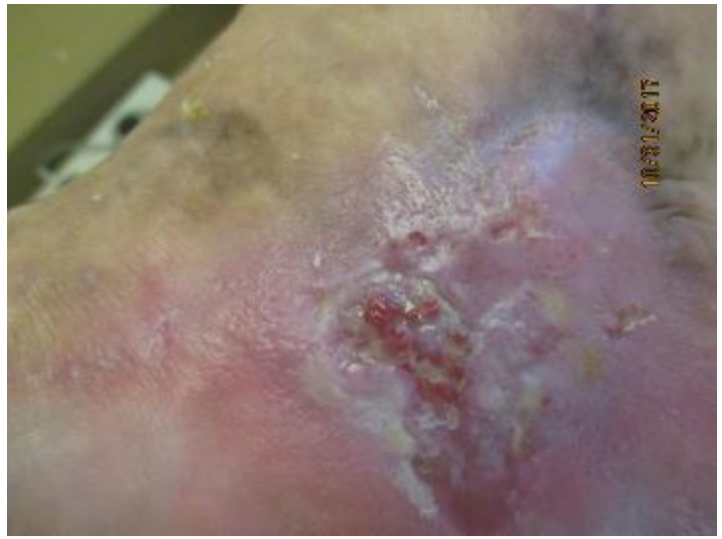

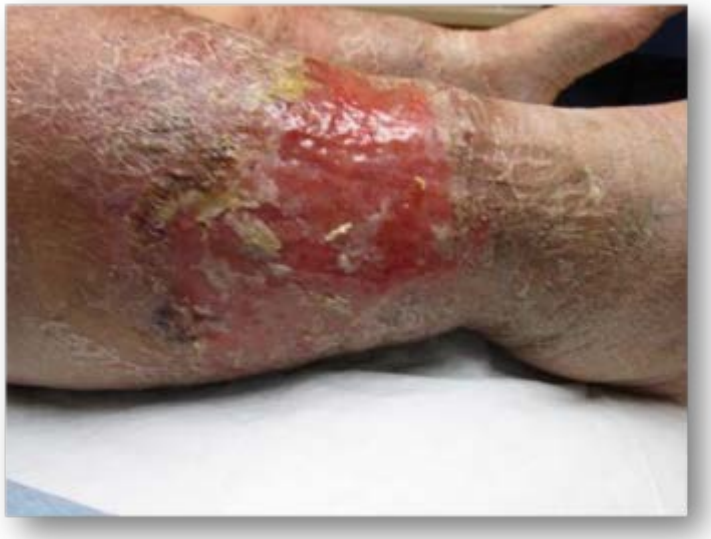

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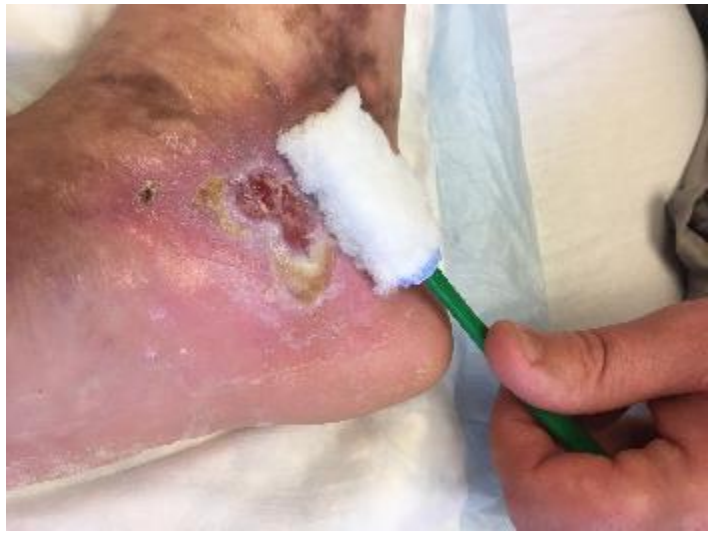

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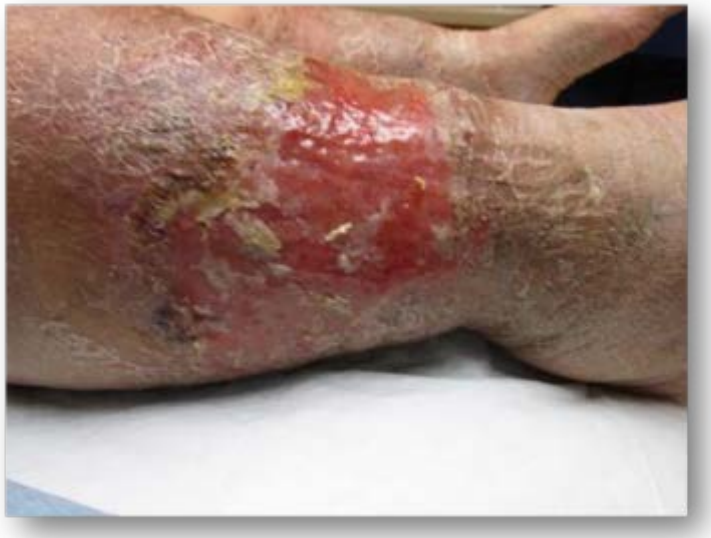

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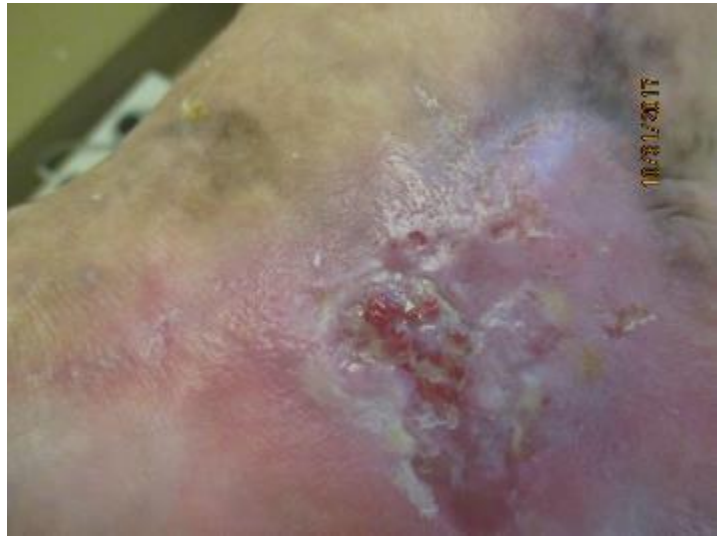

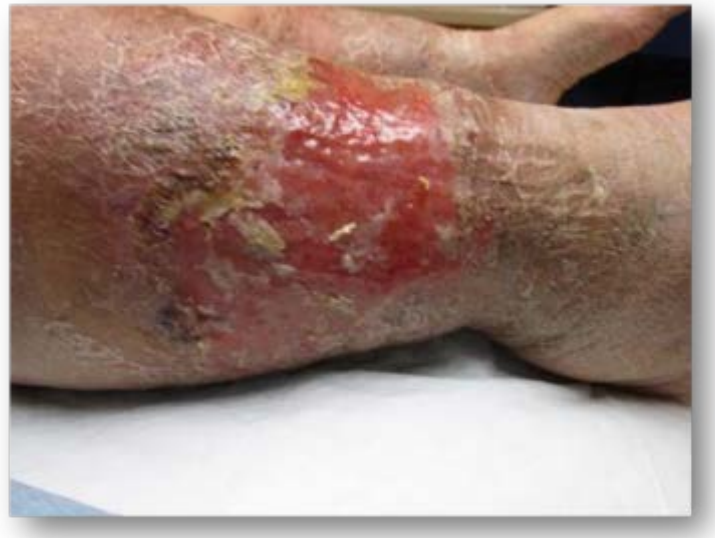

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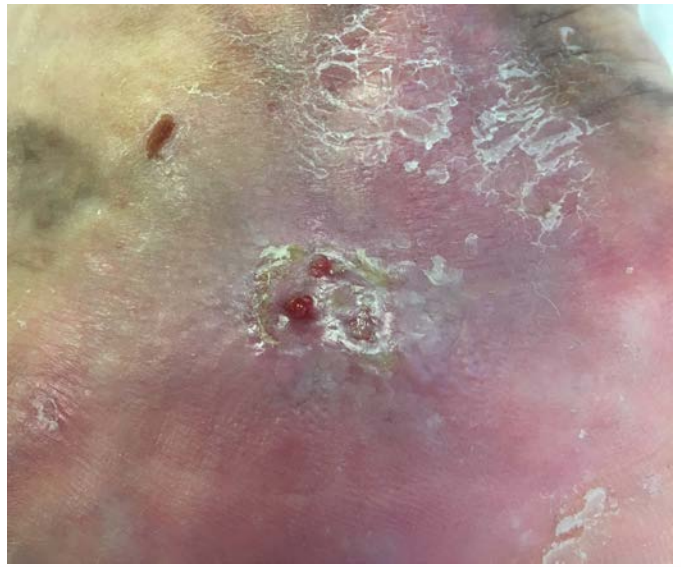

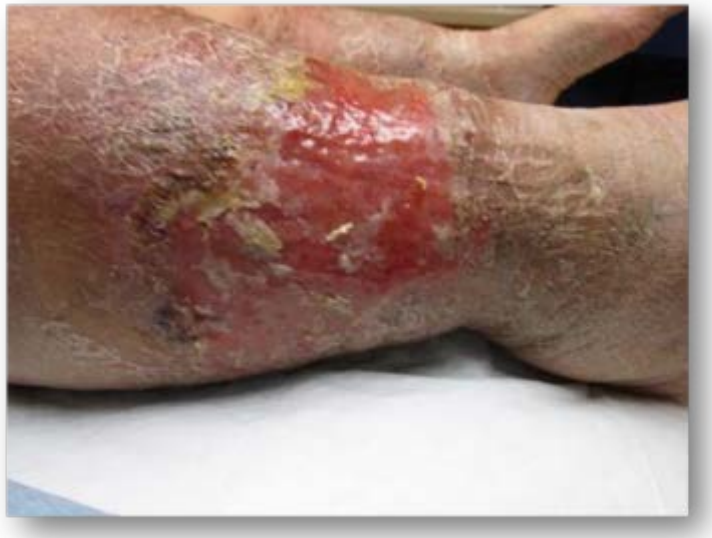

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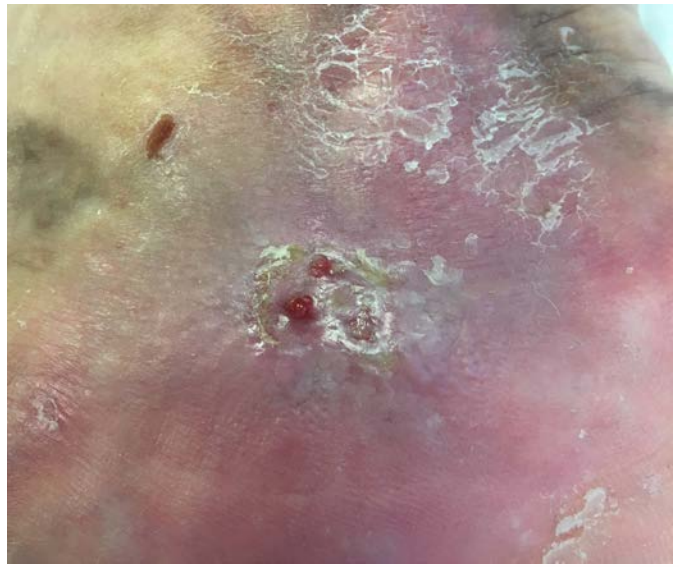
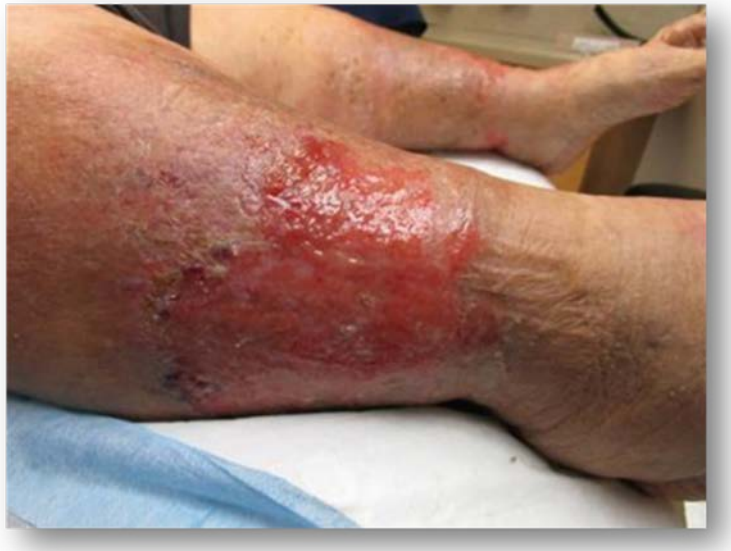




"T" – Tissue Debridement

Remove Non-Viable or Deficient Tissue

Goals are to remove necrotic tissue, microdebris, reduce bacterial burden

Strategy	Description	Examples
Surgical (Excisional/Sharp)	Removal by surgical instrument	Scalpel, scissors, hydrosurgery, lasers, curettes,
Mechanical	Removal of necrotic tissue by mechanical means	Wet- to dry dressings, hydrotherapy, ultrasound, abrasion
Biosurgical	Sterile larvae selectively digest necrotic tissue and bacteria	Sterile blowfly or housefly larvae
Autolytic	Uses the body's own enzymes to dissolve necrotic tissue; assisted with moisture-retentive dressings	Moisture retentive dressings
Enzymatic	Topical application of enzymes to liquefy necrotic tissue	Collagenase

The Wound Healing Society. Chronic Wound Care Guidelines. 2007. http://woundheal.org/documents/final_pocket_guide_treatment.aspx. Accessed March 29, 2017.

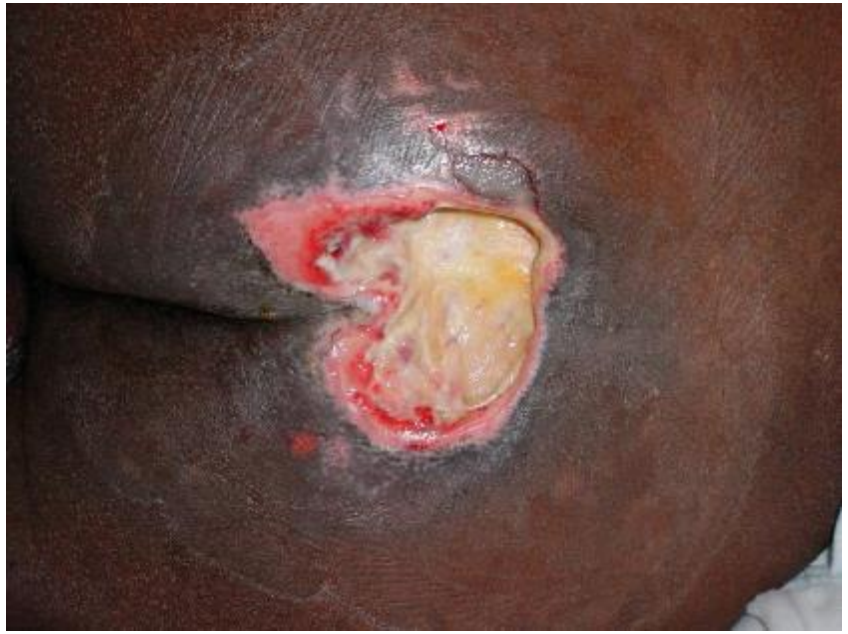
Debridement and Wound Healing

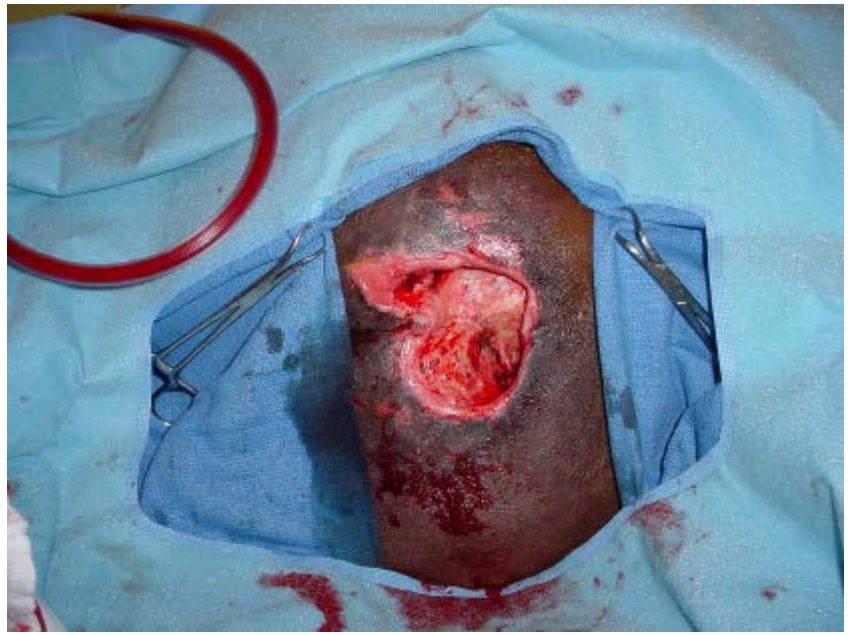
rhPDGF (%)			Placebo (%)	
Center	Debrided	Healed	Debrided	Healed
1	15	20	19	10
2	33	50	35	17
3**	37	64	43	36
4	45	50	58	17
5	68	53	59	32
6	81	83	87	25

***Combined data*

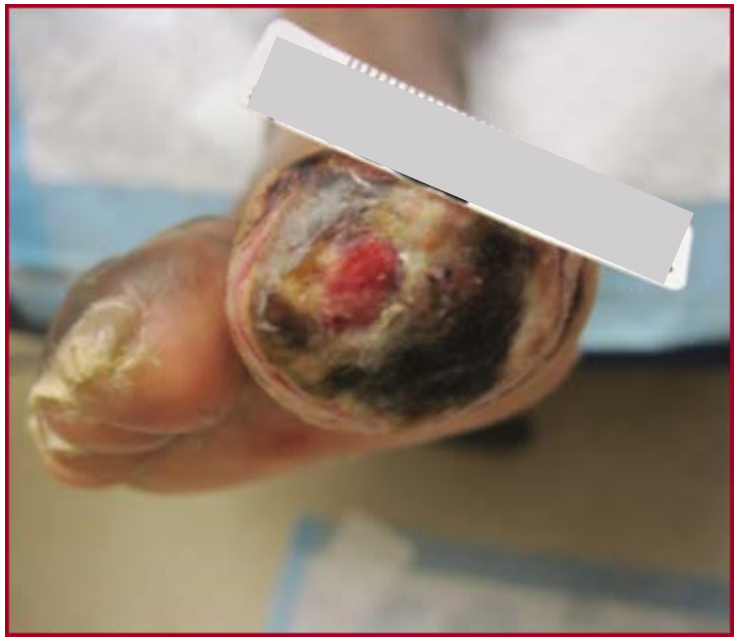
Steed DL, Donohoe D, Webster MW, Lindsley L. Effect of extensive debridement and treatment on the healing of diabetic foot ulcers. Diabetic Ulcer Study Group. J Am Coll Surg. 1996;183(1):61–4. [PubMed]

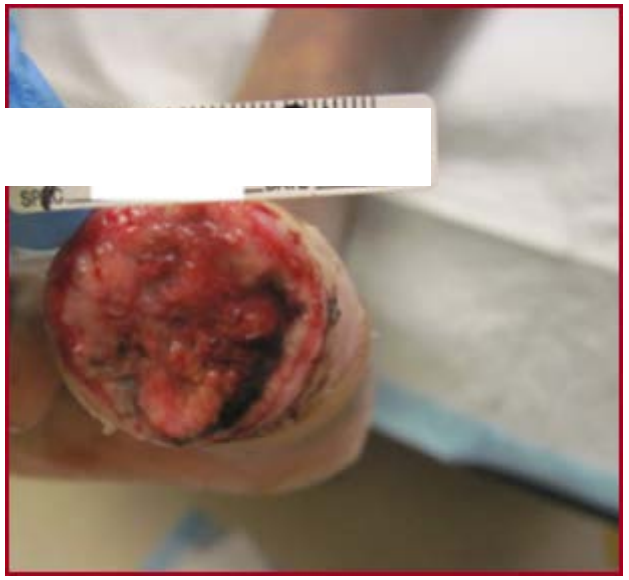
Hydrosurgical

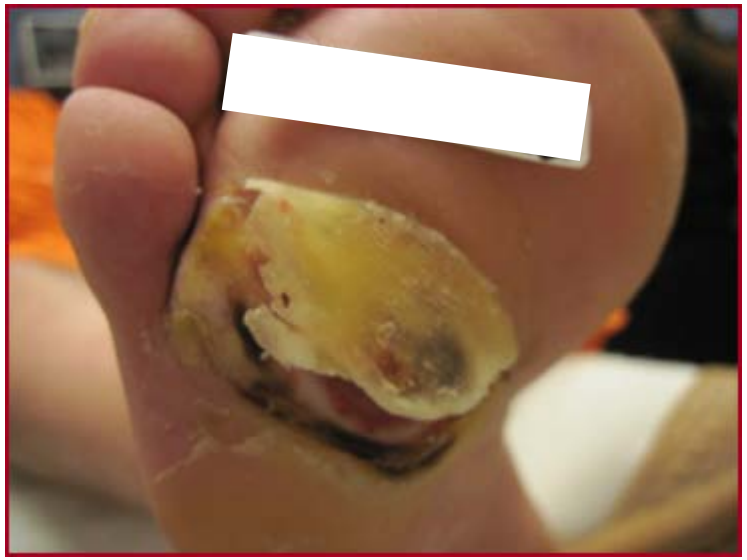


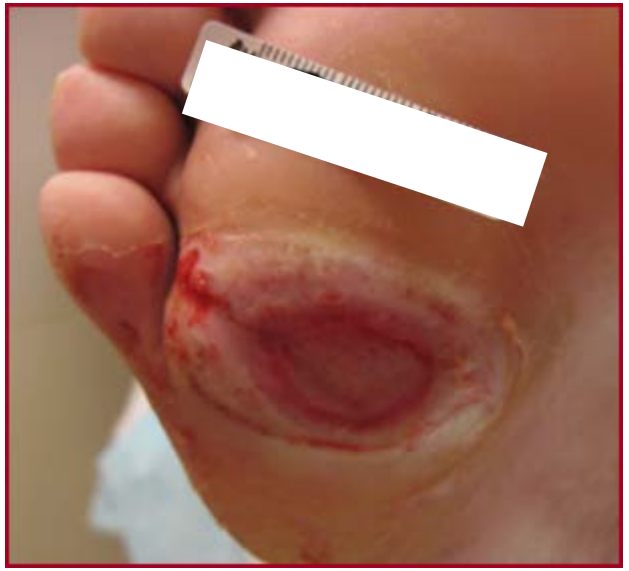


Bed / Chairside Debridement

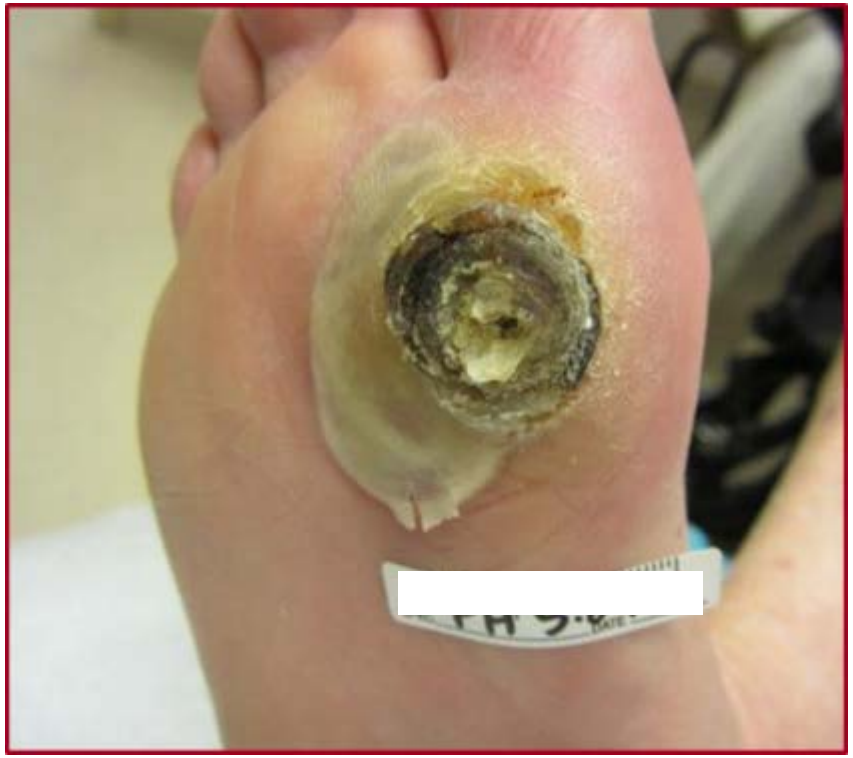


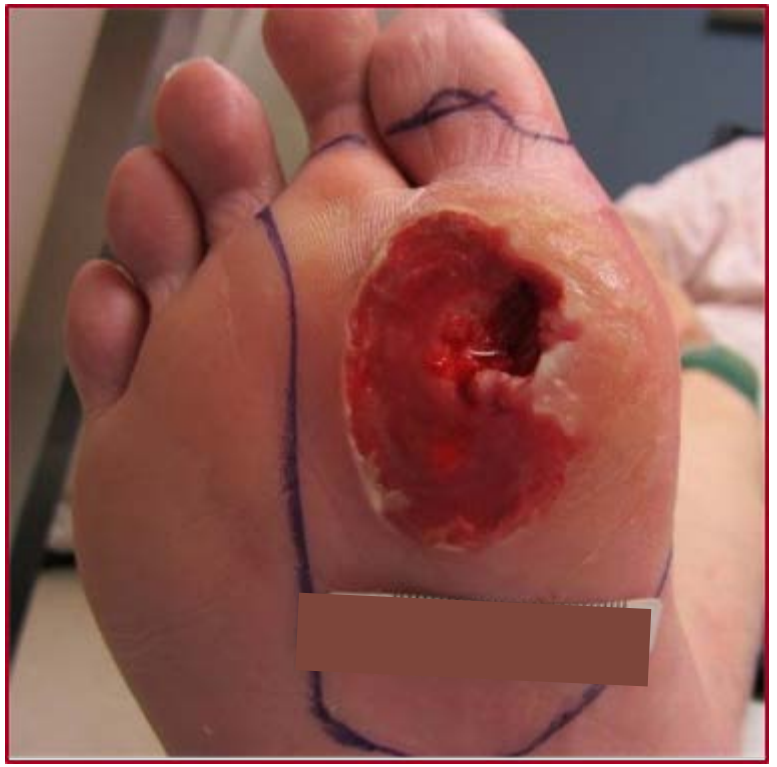




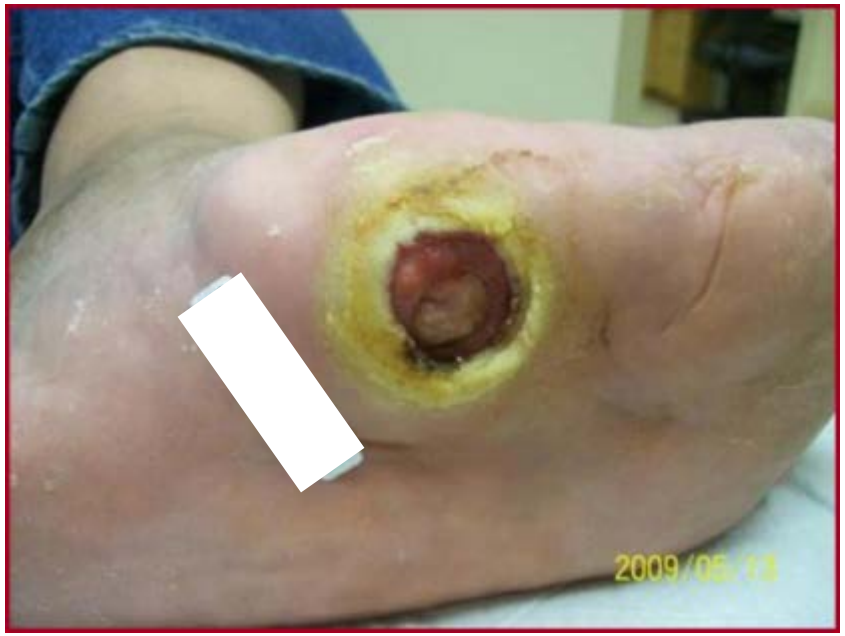


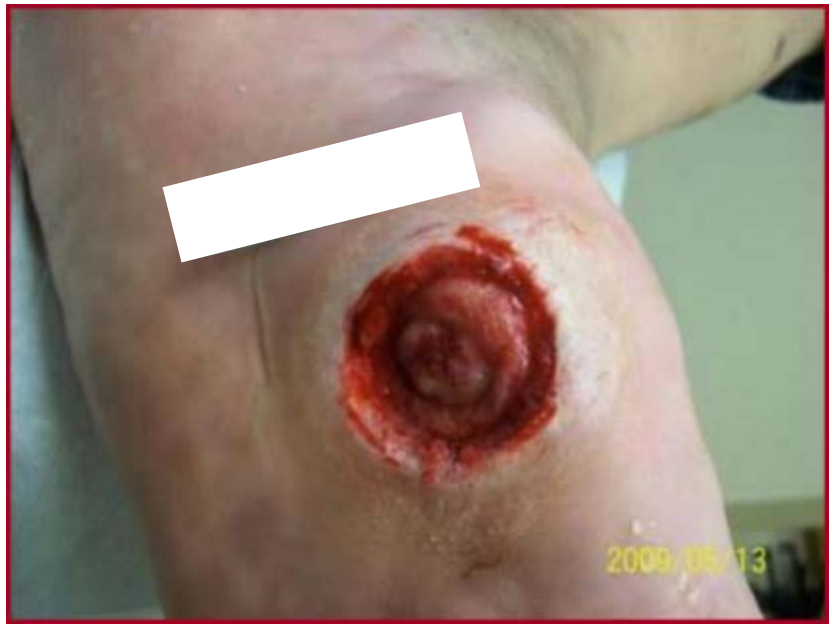
Leave no stone (or callus) unturned!





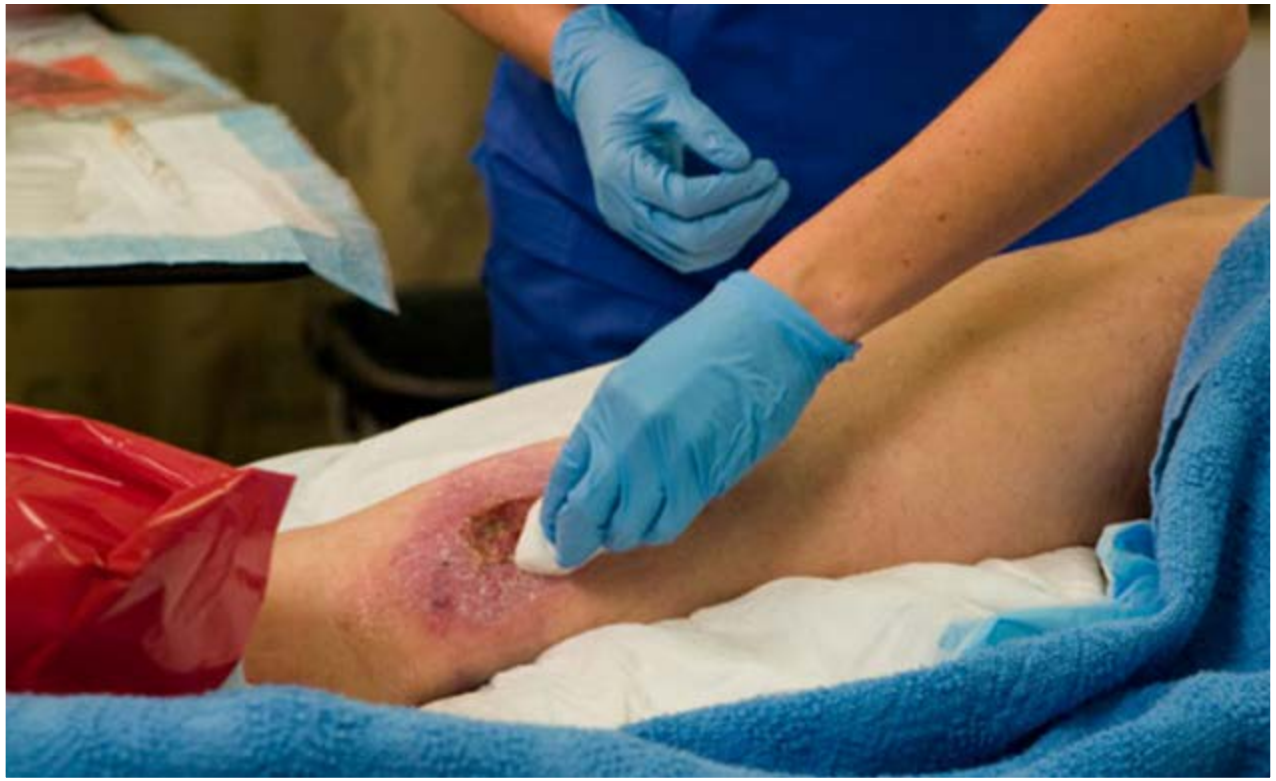
Edges must be excised……



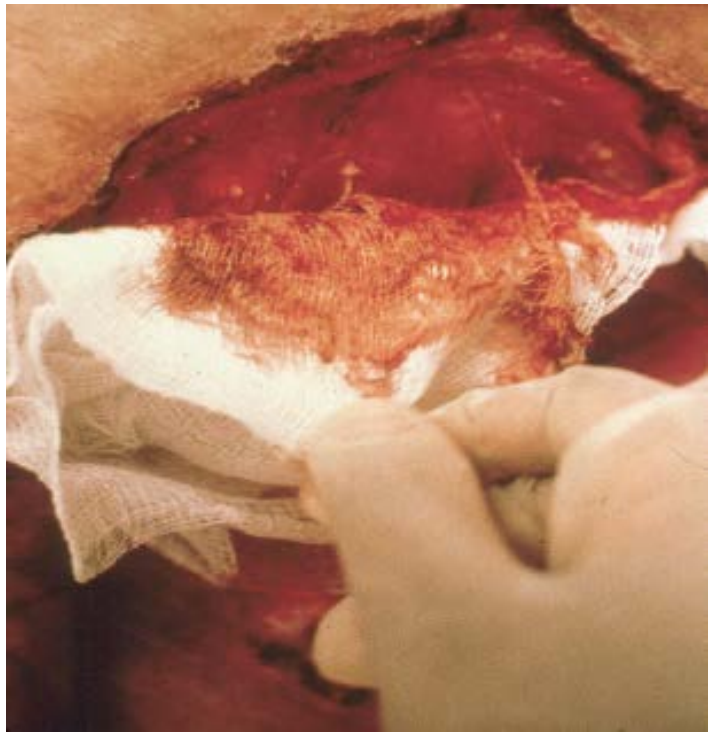


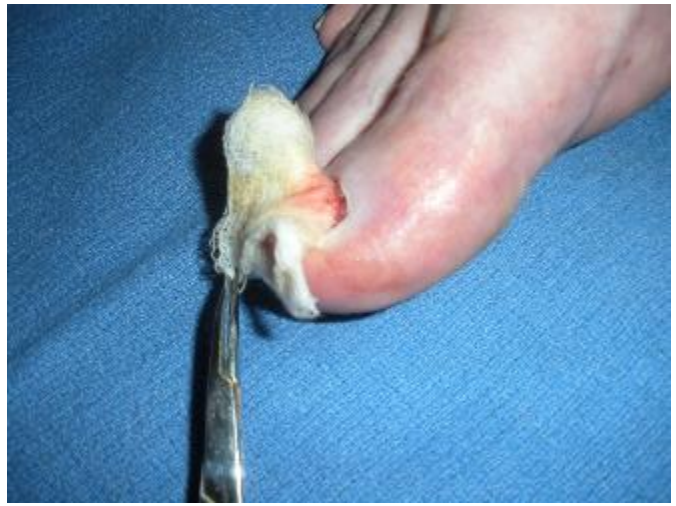
Mechanical

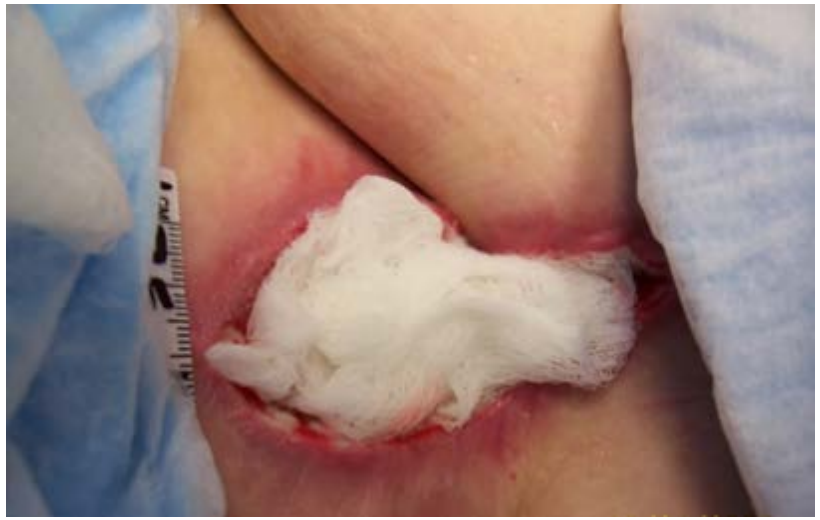
- Mostly nonselective, physical method of removing both viable and nonviable tissue and debris from a wound using a physical force such as wet to dry dressings, wound irrigation, pulsatile lavage and ultrasound and abrasion



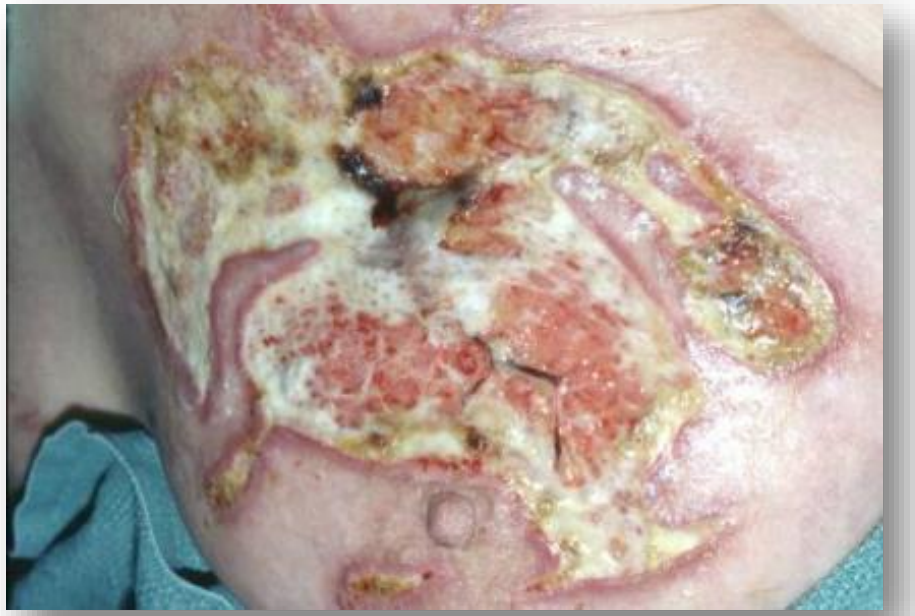
Wet to Dry/Moist Dressings

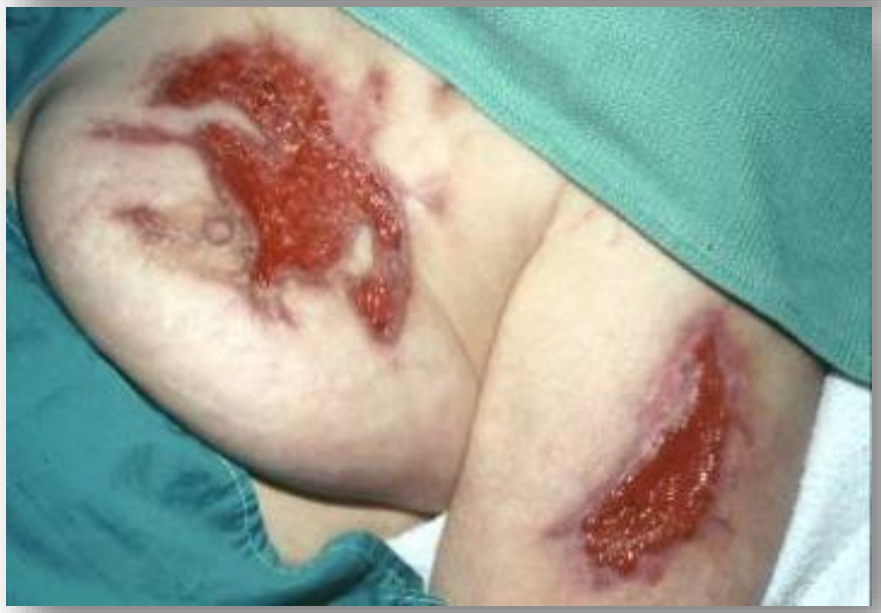


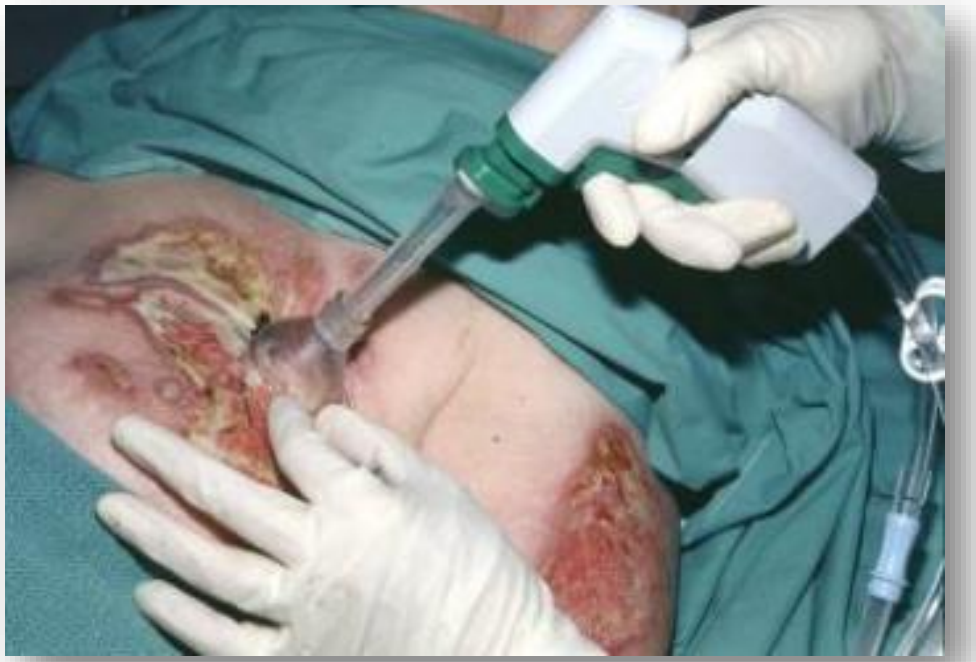




PLWS Outpatient Burn







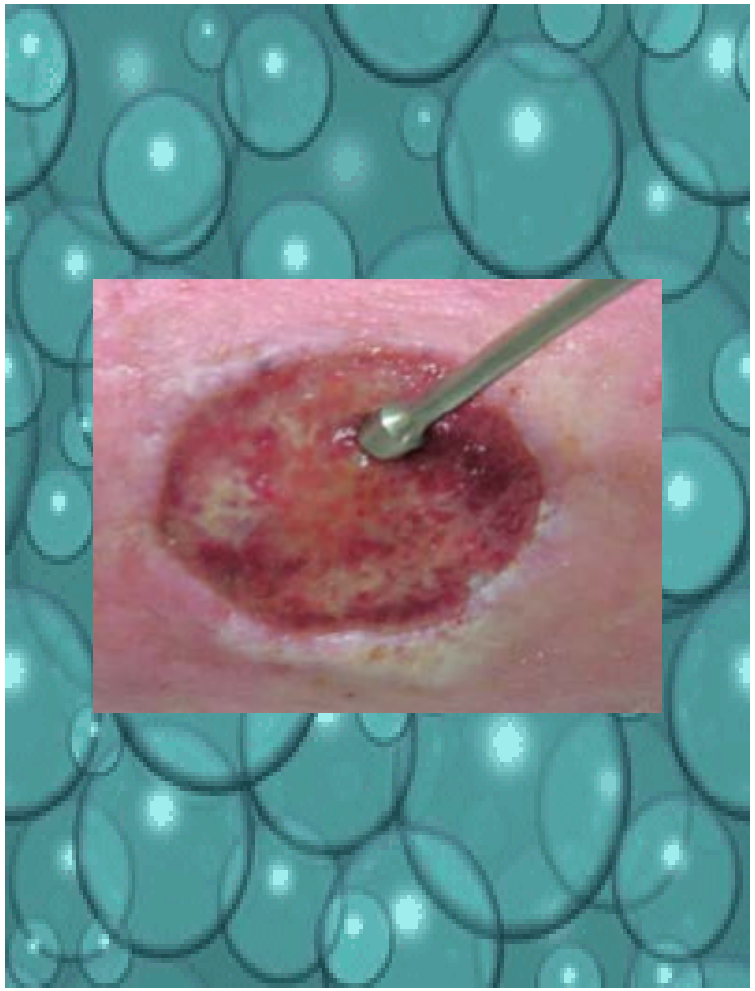
Courtesy of Harriett Loehne, DPT, CWS

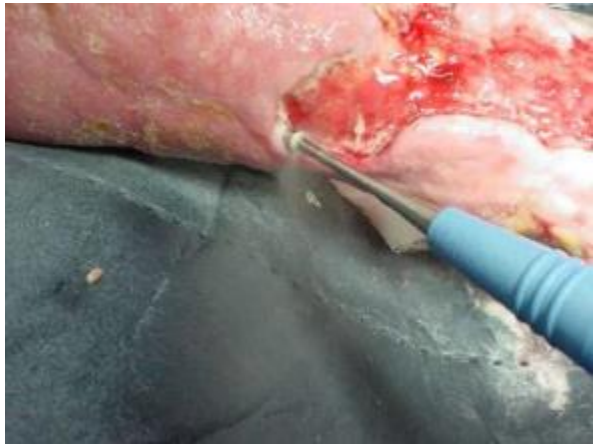
Low-frequency, Non-contact Ultrasound

Ultrasound Assisted Wound Therapy

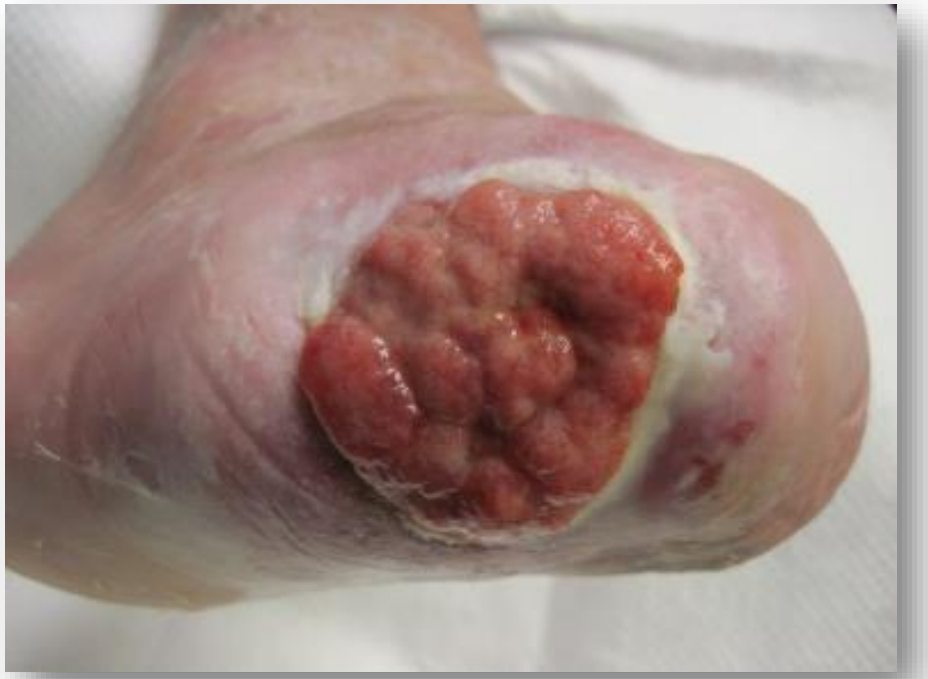
Ultrasound Assisted Wound Therapy

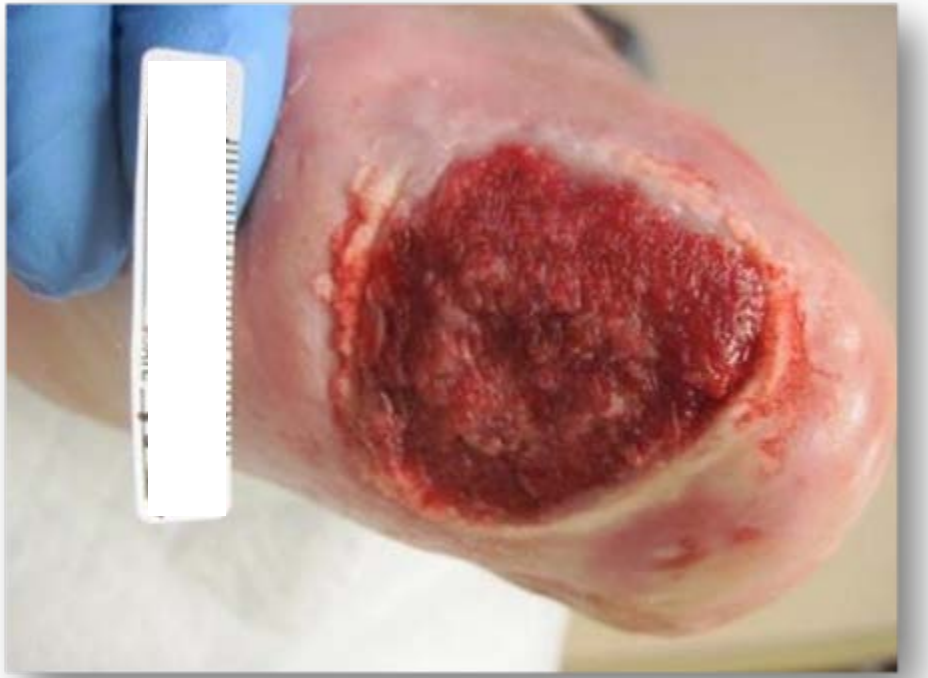
- Low frequency ultrasound
- Allows for deeper penetration of the solution
- Microcavitations cause bacterial destruction, separate devitalized tissue



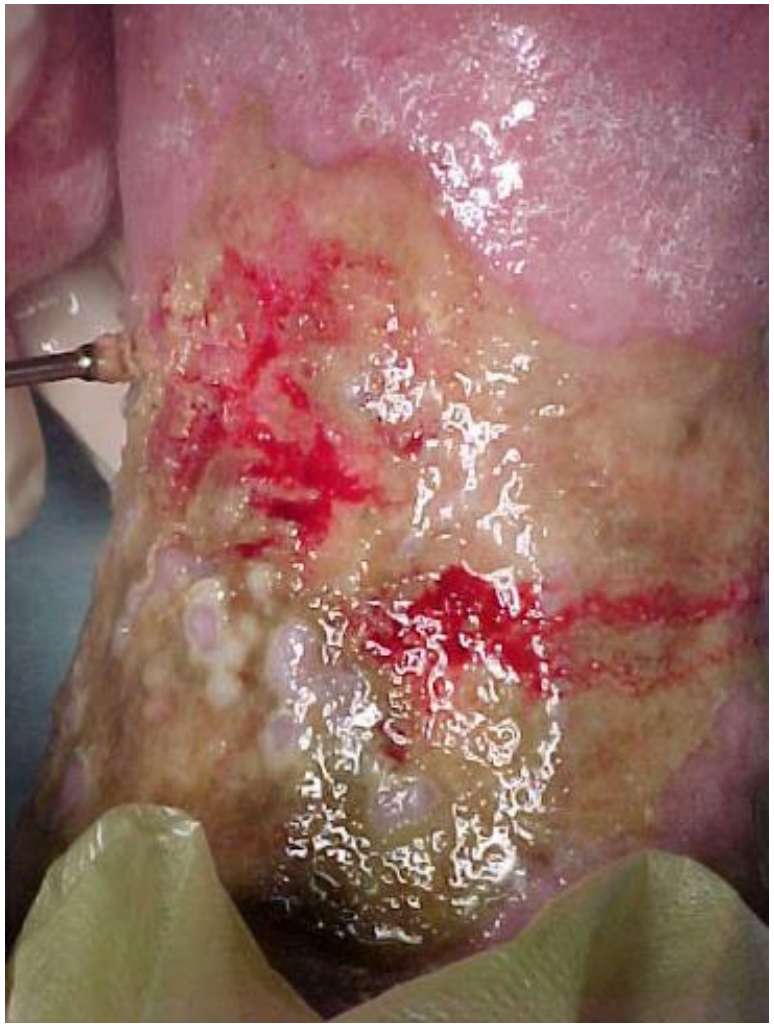


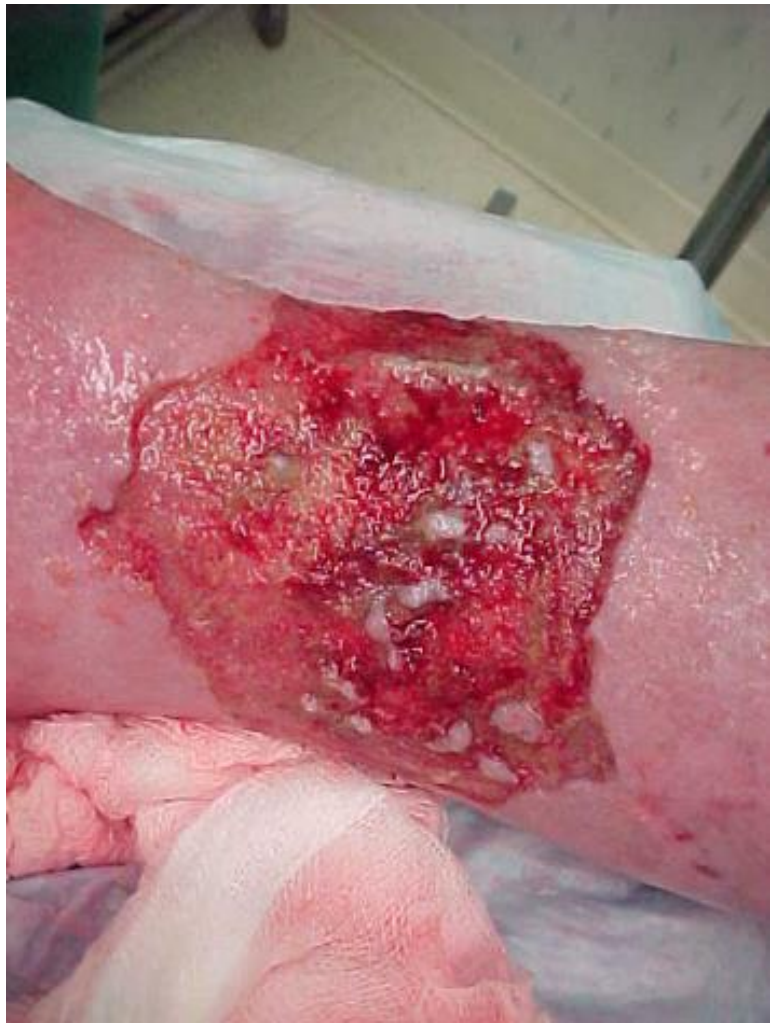
Ultrasound for Hypergranulation Tissue





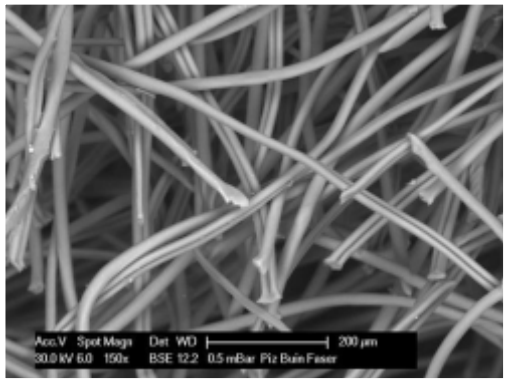
Ultrasound for Slough / Fibrinous Exudate

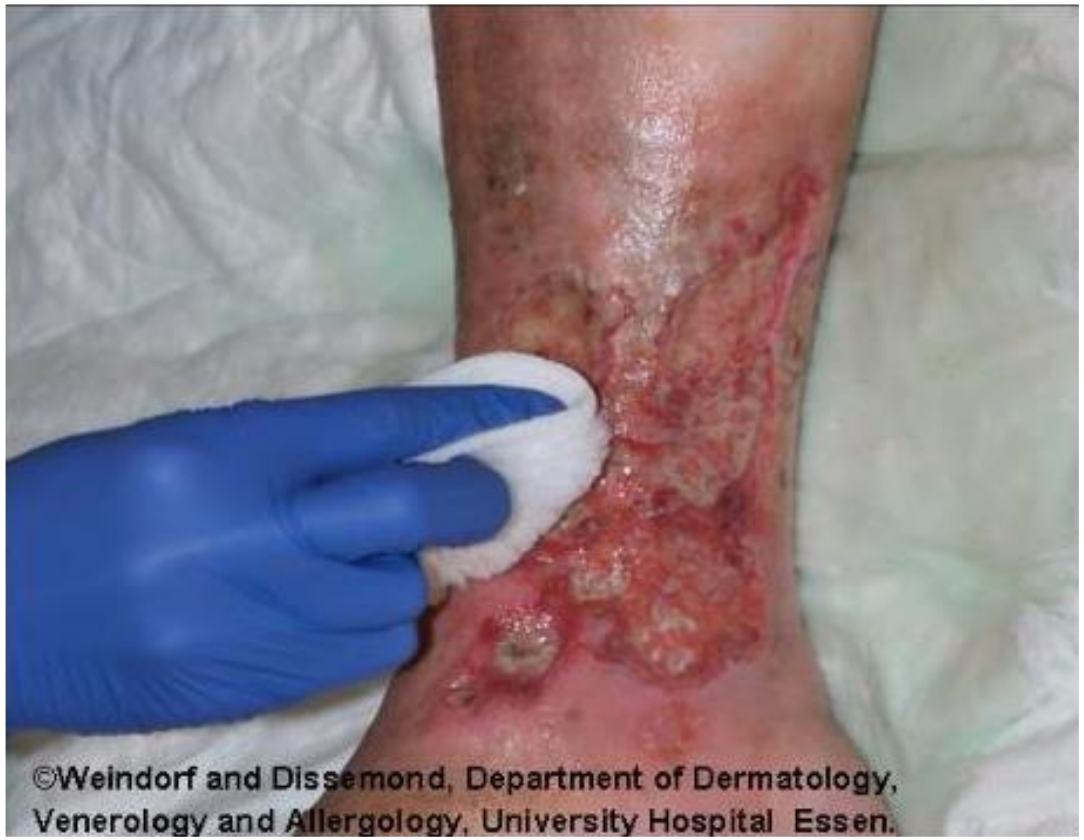




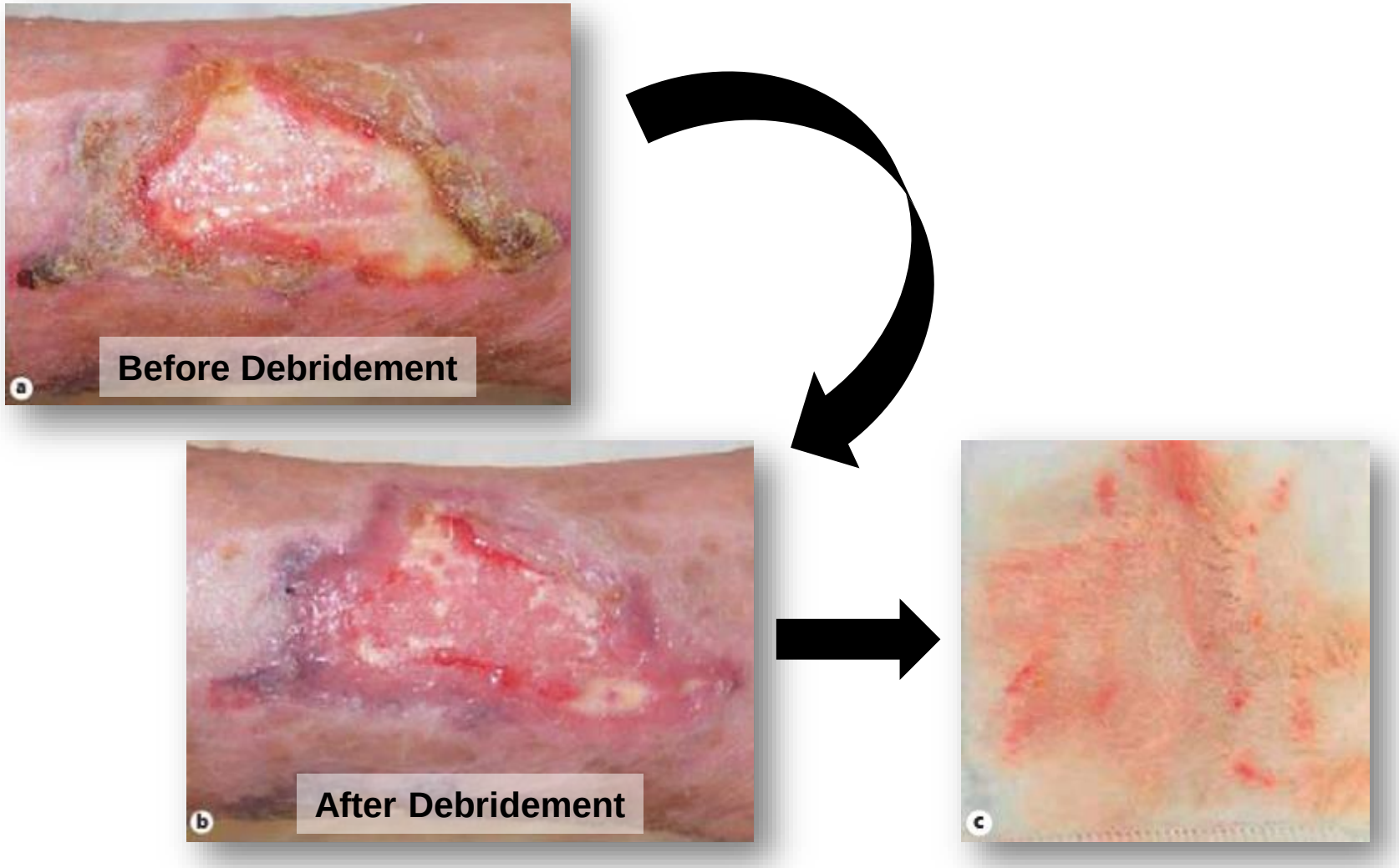
Monofilament Technology: What is It?

Device with 18 million angled, soft polyester fibers that promote wound and periwound cleansing and debridement *effectively* and relatively *painlessly*



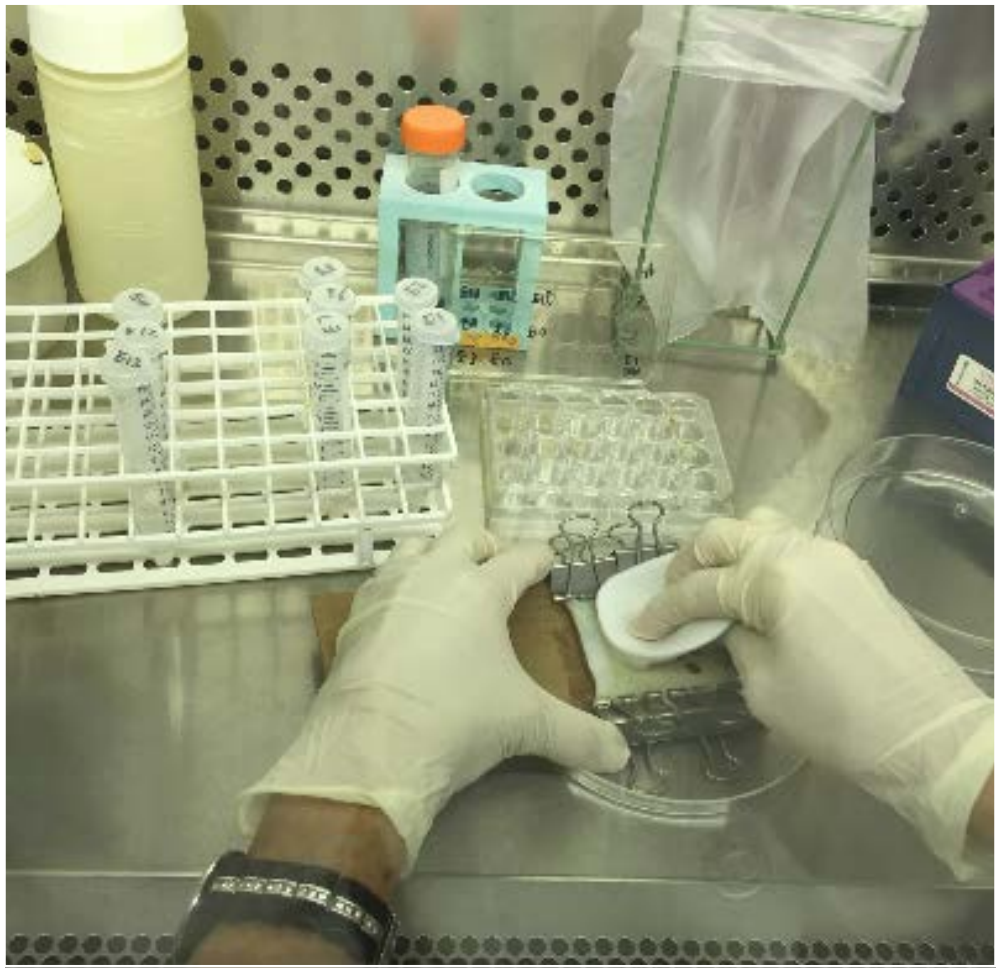


Example of Venous Leg Ulcer Patient Debrided with Monofilament Pad



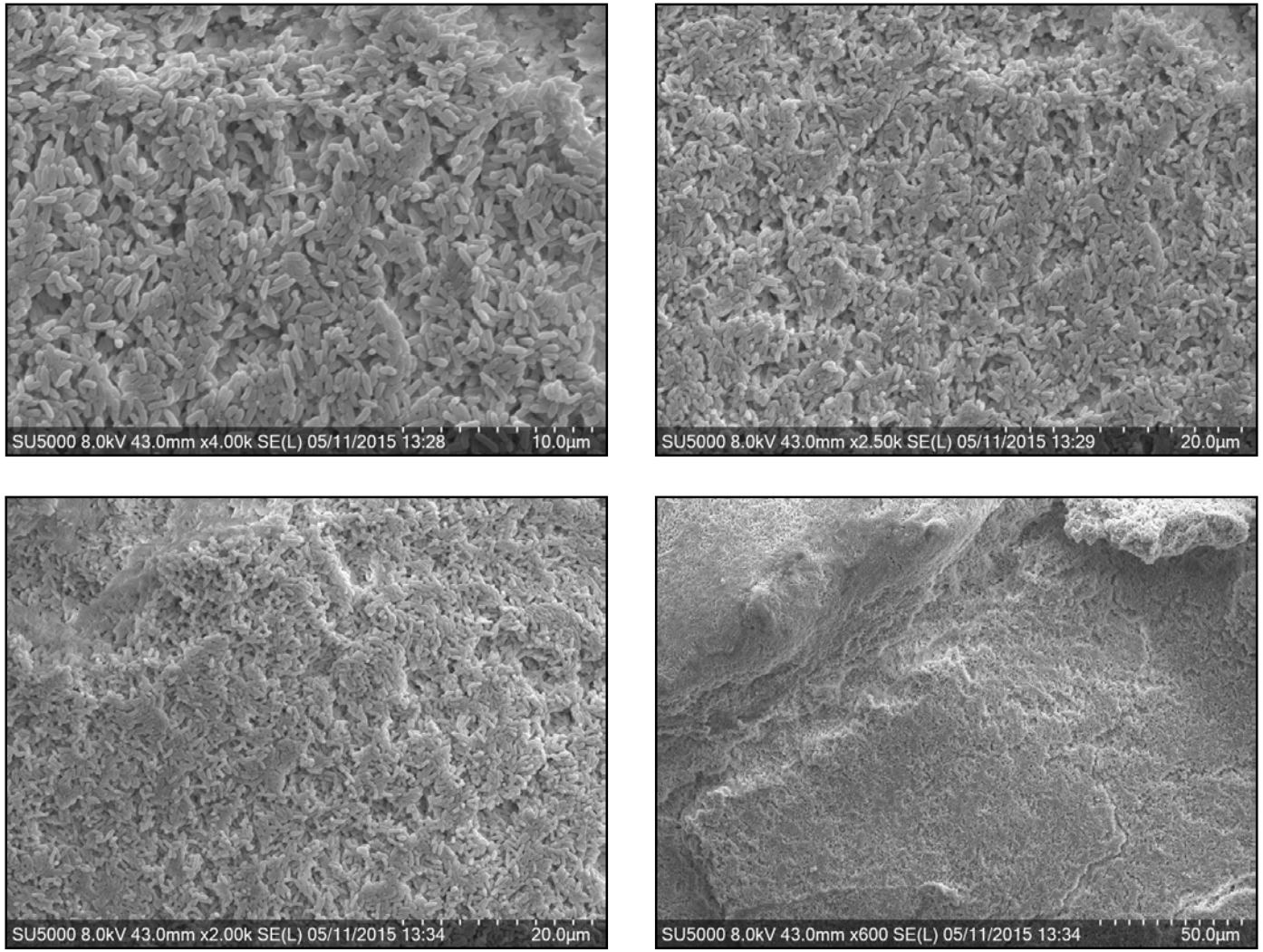
Wiegand C, et al. *Skin Pharmacol Physiol*. 2016;29(6):318-323.

Monofilament Pad Debridement of Mature *P. aeruginosa* Biofilm Grown on Pig Skin Explant



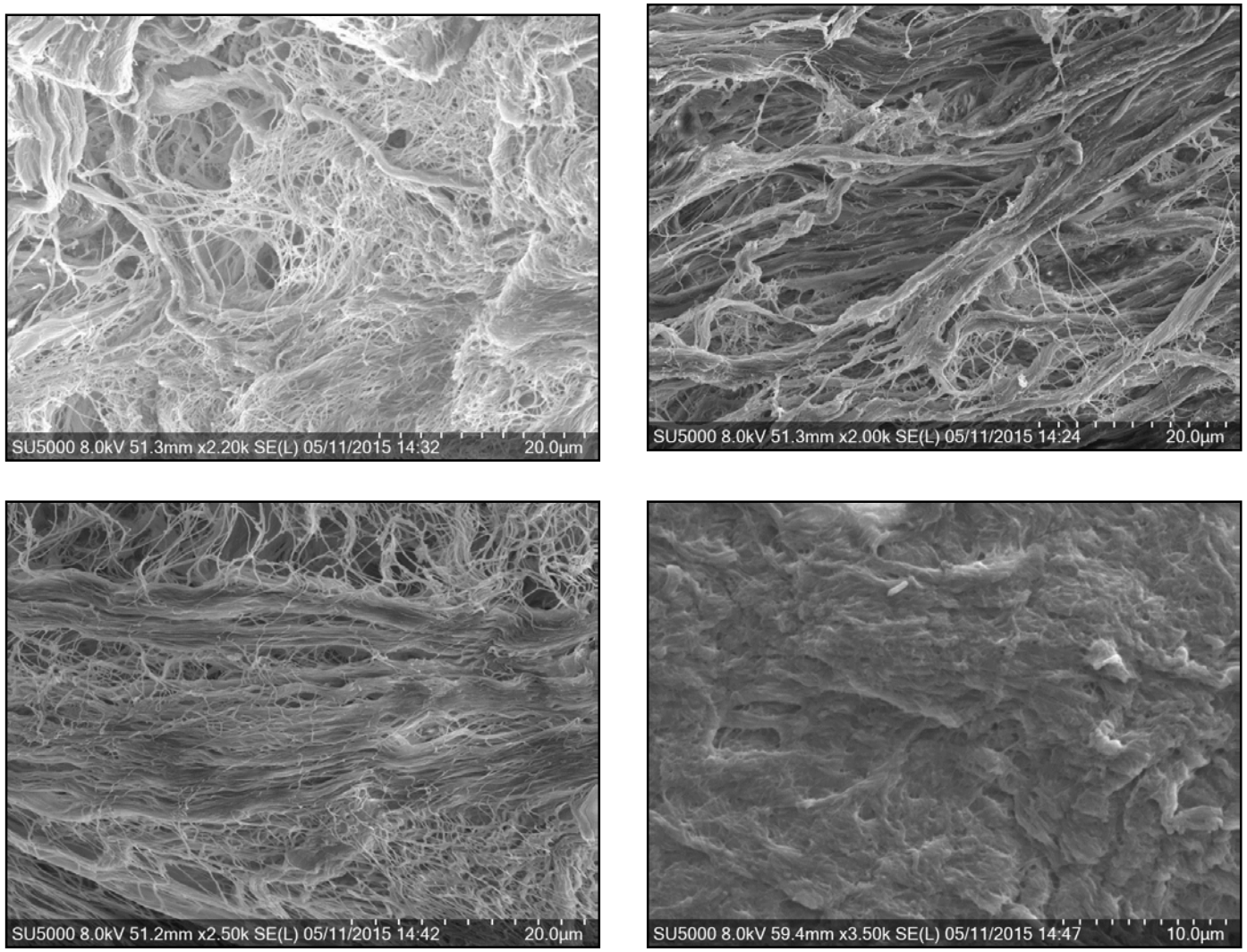
Yang Q, et al. *Microfilament pad debridement of biofilms on pig skin explants and clinical cases.* Submitted.

SEM of Mature *P. aeruginosa* Biofilm on Pig Skin Explants before Monofilament Debridement



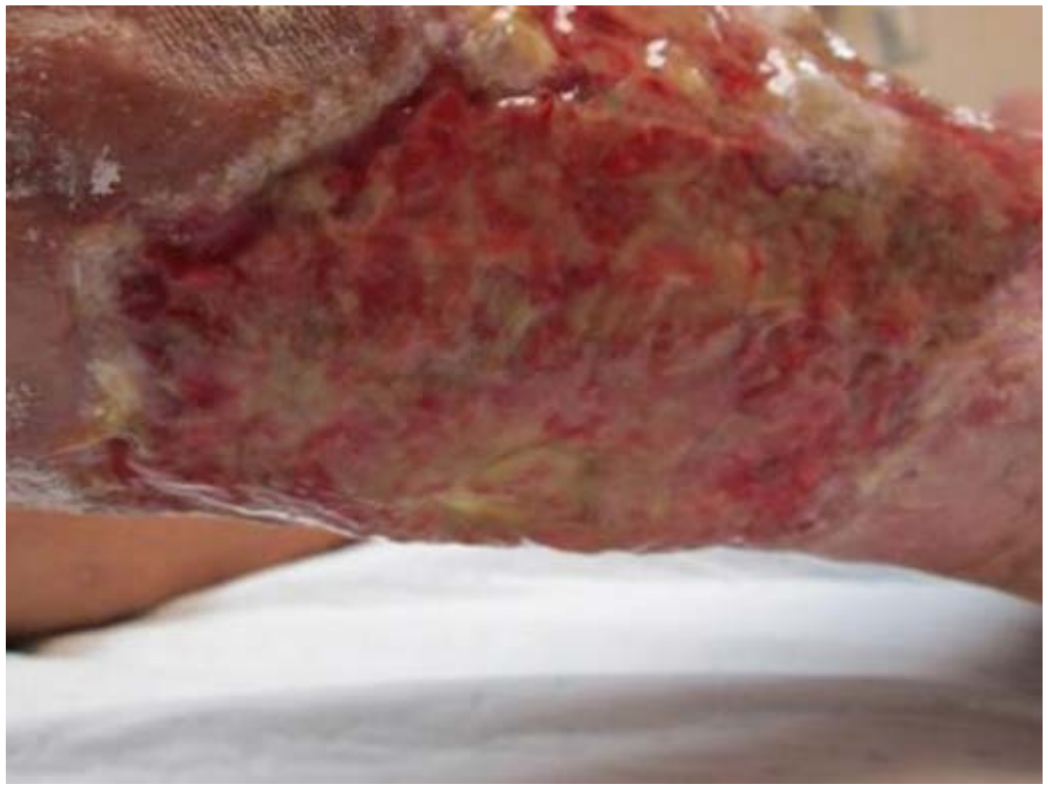
- SEM = scanning electron microscopy.
- Yang Q, et al. *Microfilament pad debridement of biofilms on pig skin explants and clinical cases.* Submitted.

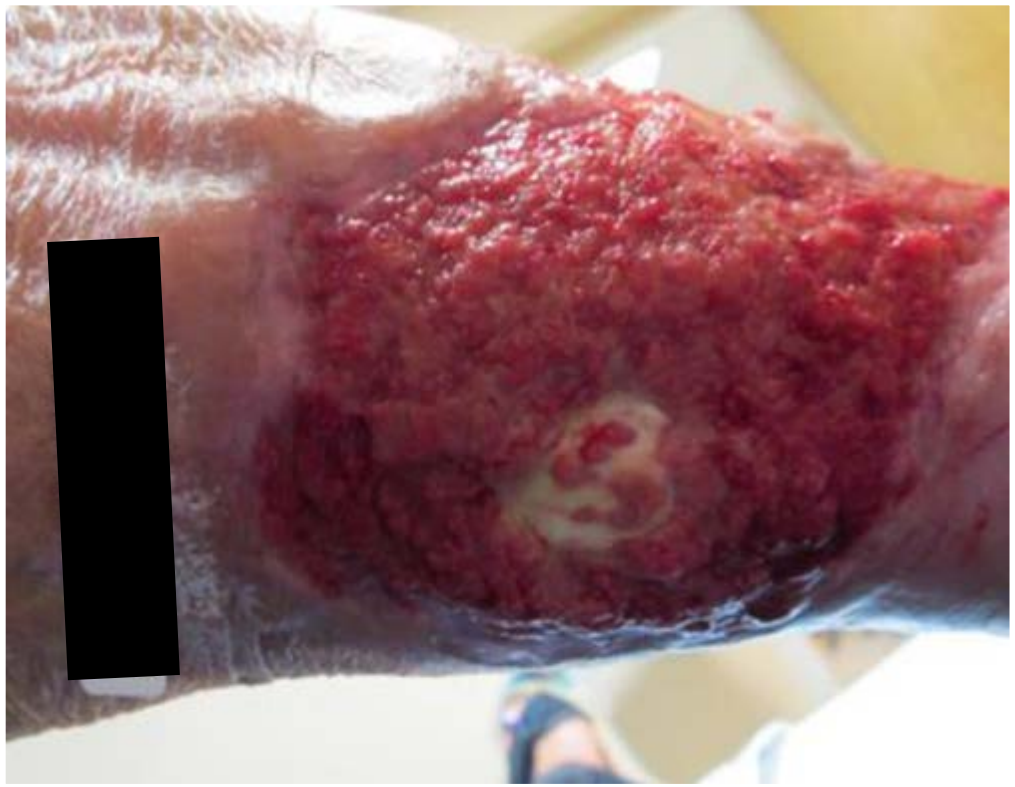
SEM of Mature *P. aeruginosa* Biofilm on PigSkin Explants after Monofilament Debridement



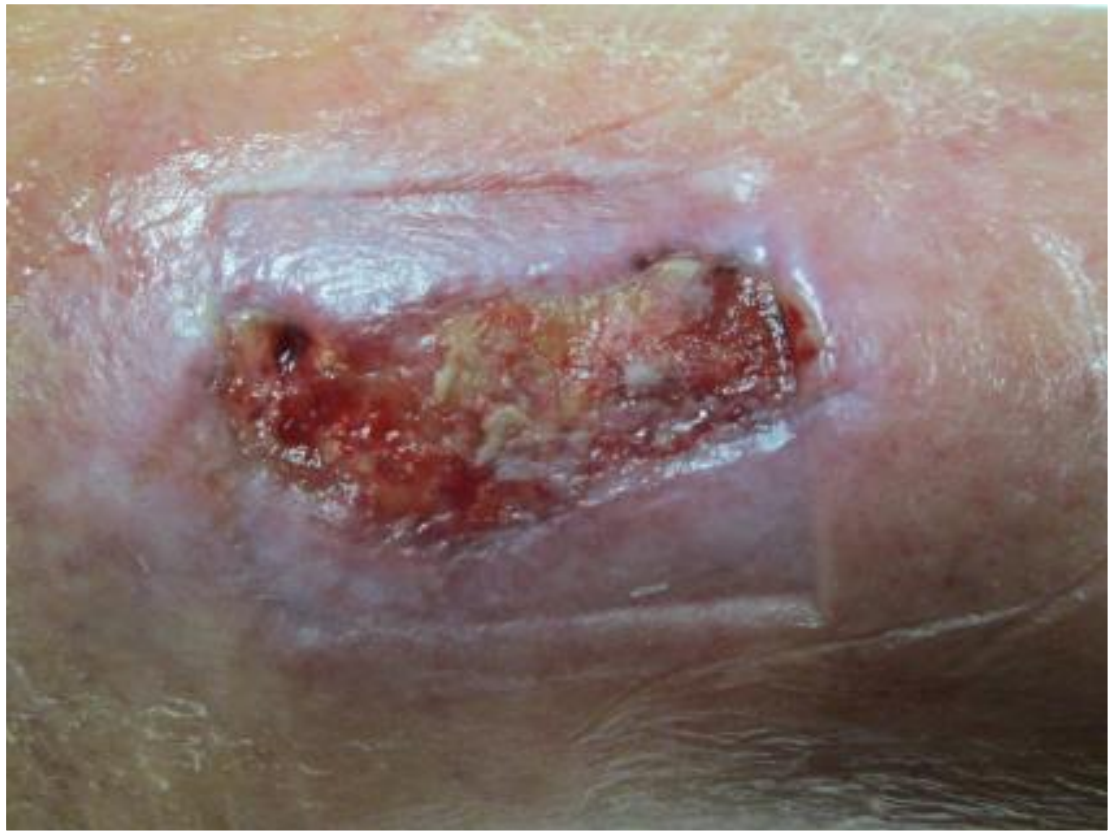
Yang Q, et al. *Microfilament pad debridement of biofilms on pig skin explants and clinical cases.* Submitted.

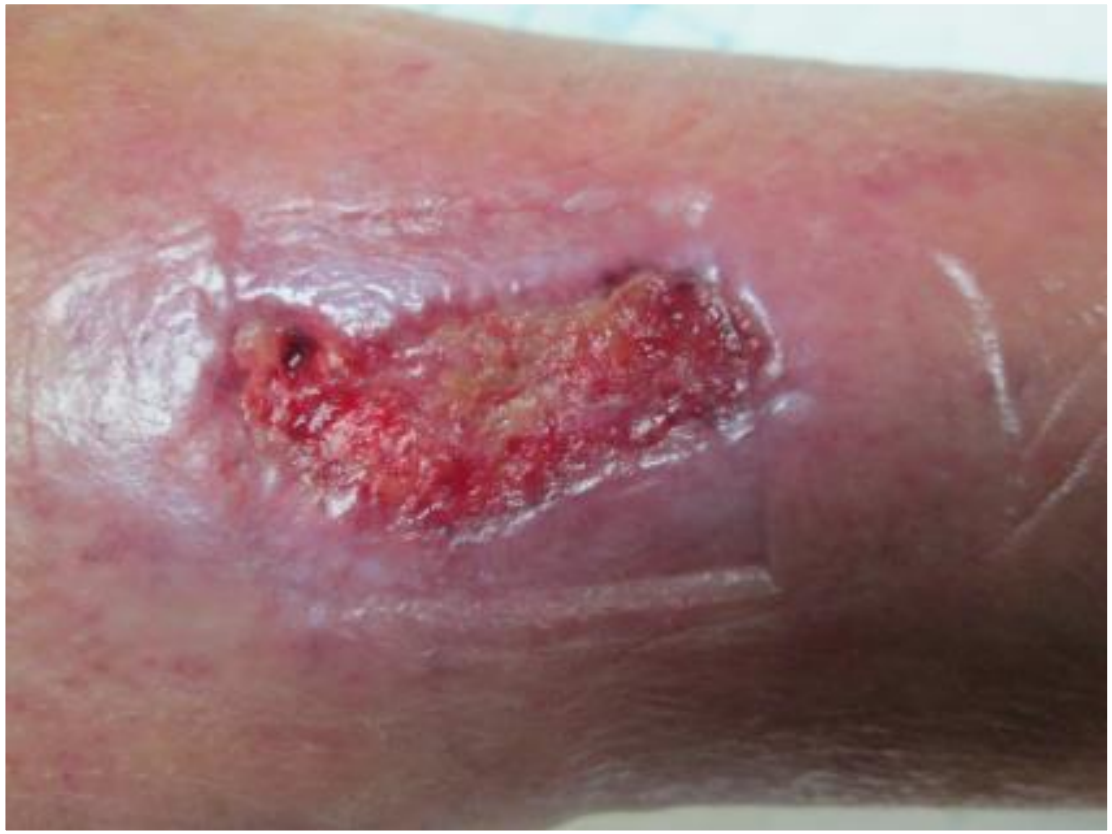
Large Venous Ulcer



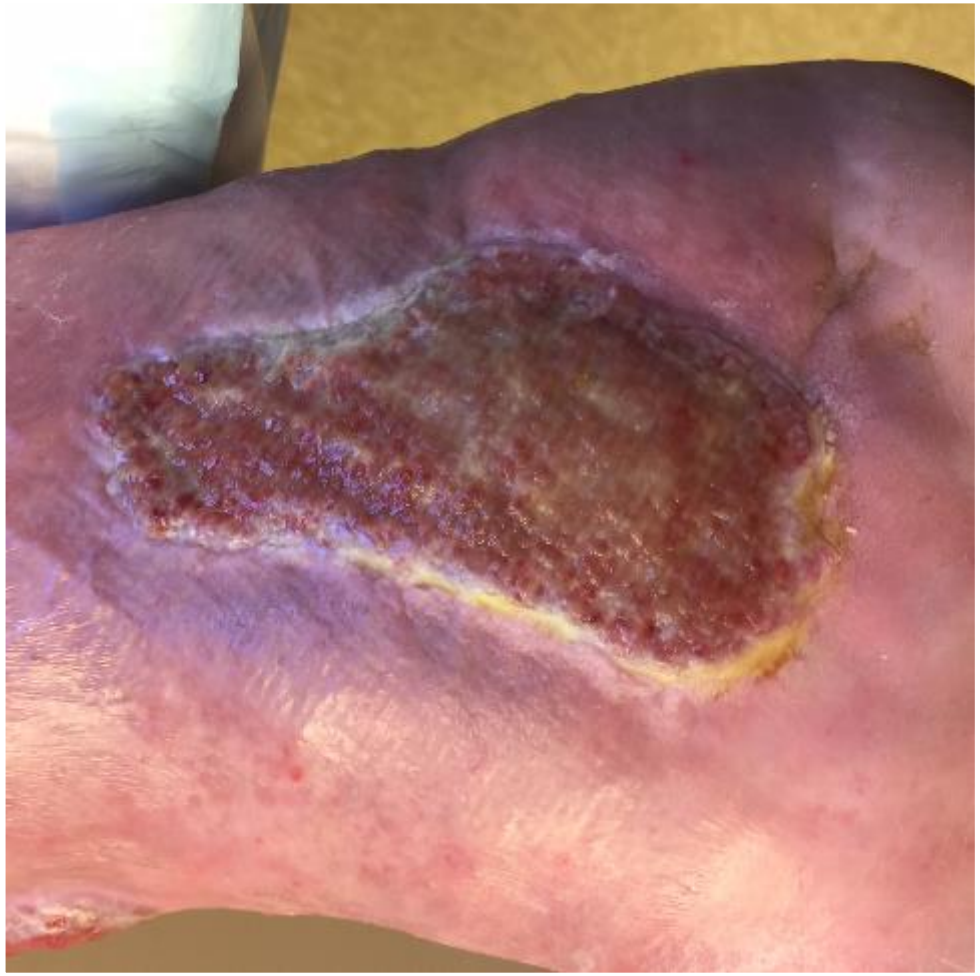


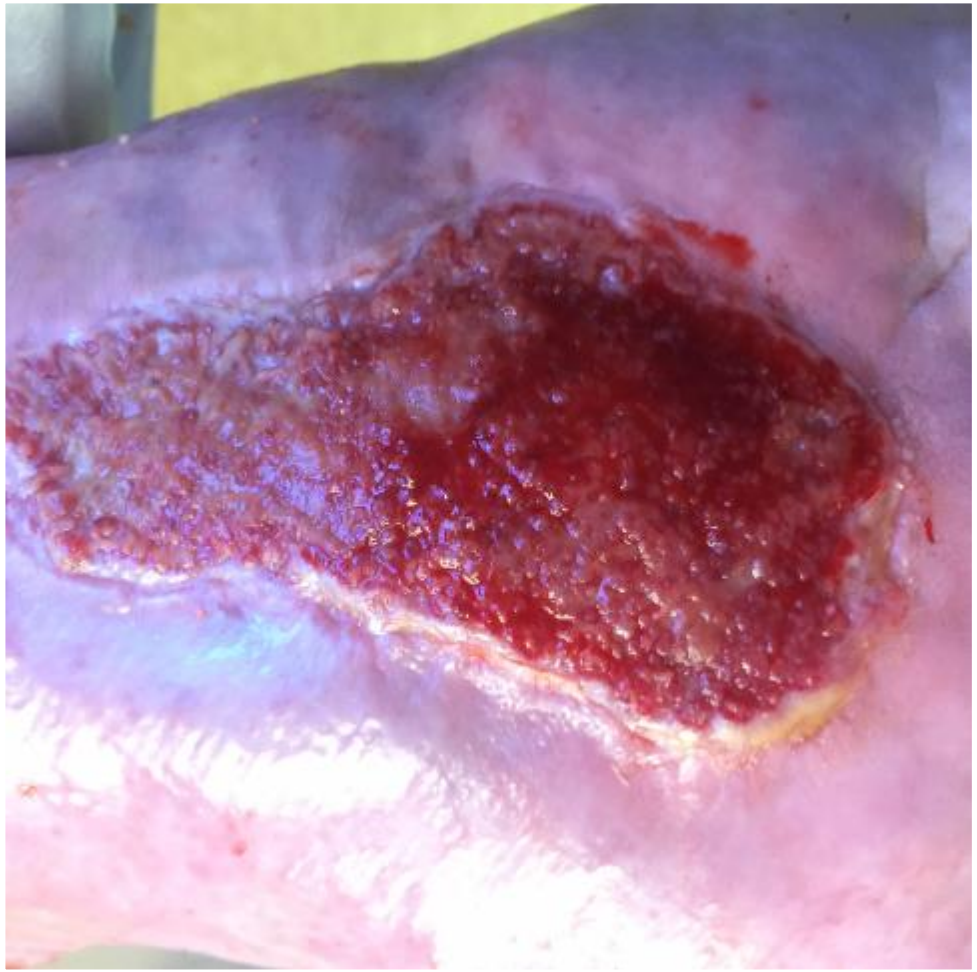
Loosened Debris



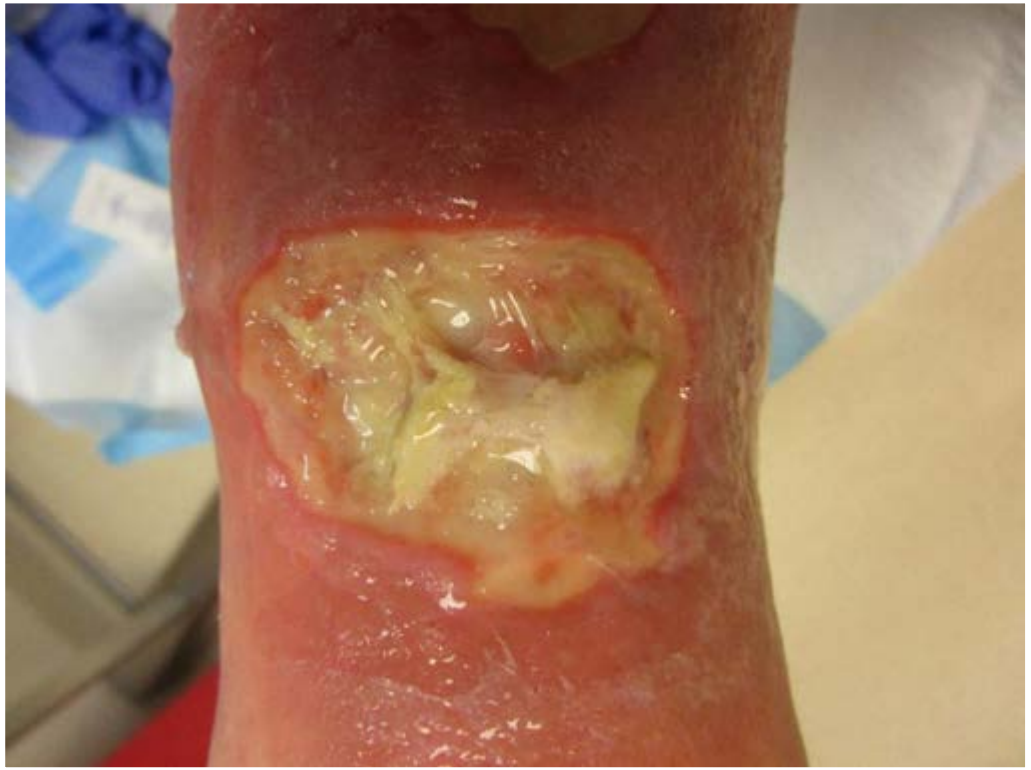


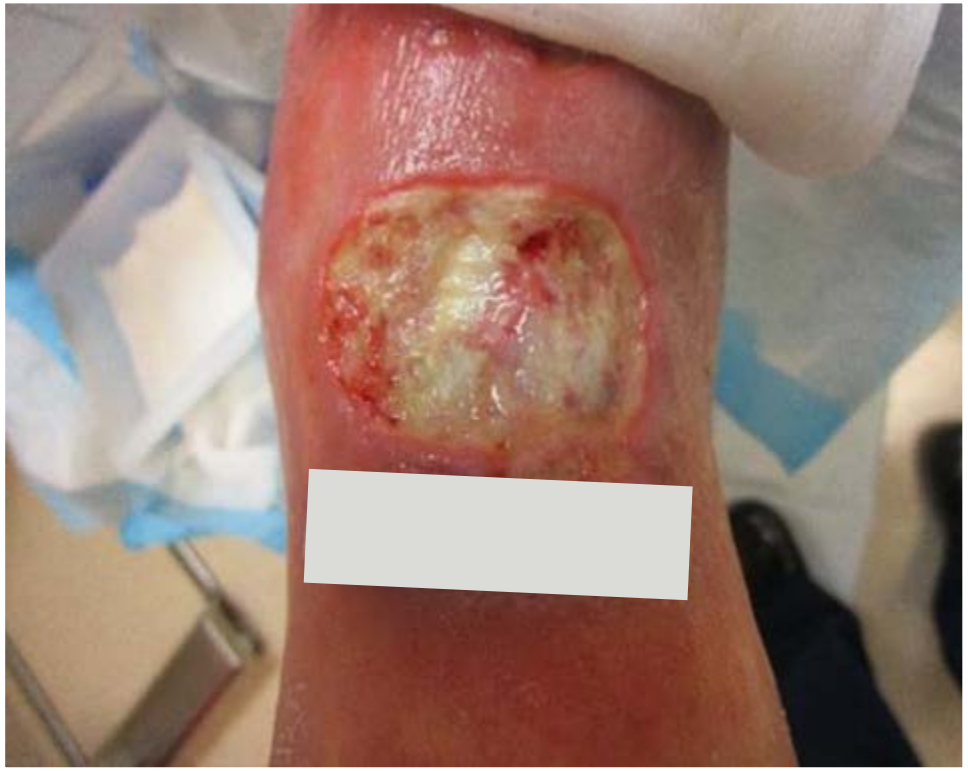
Venous Leg Ulcers



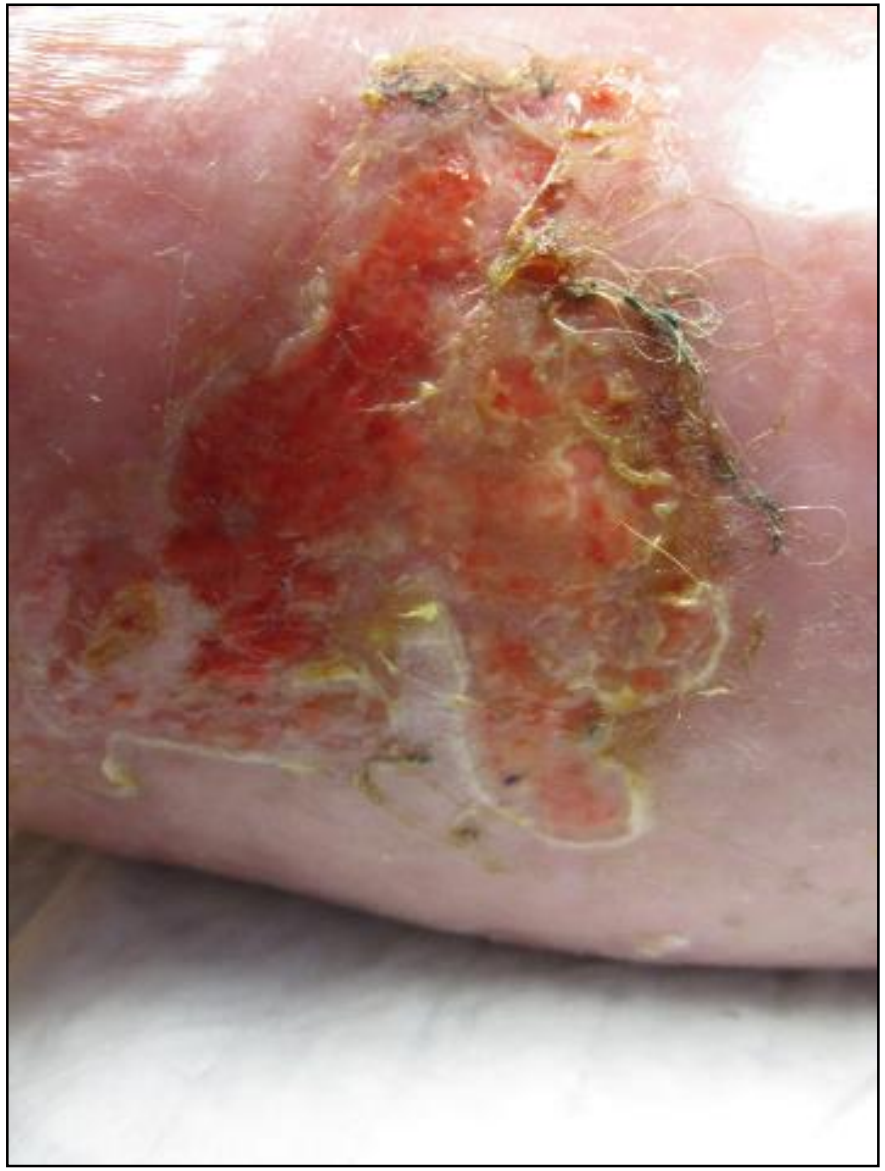


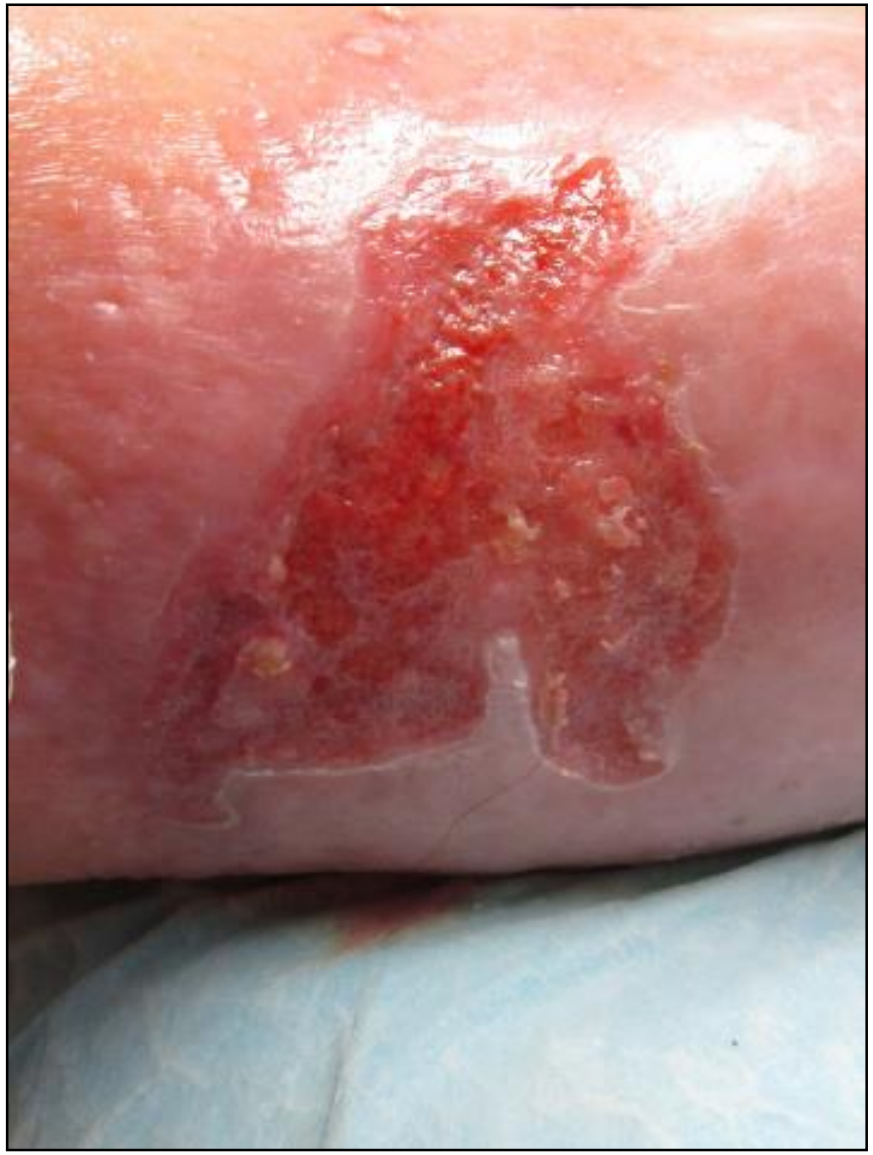
Loose Slough



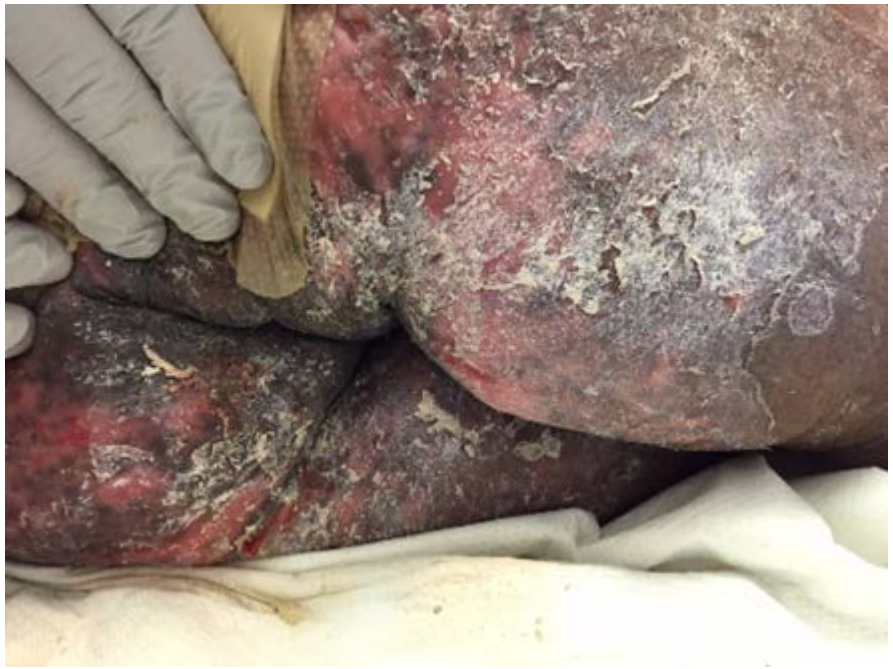


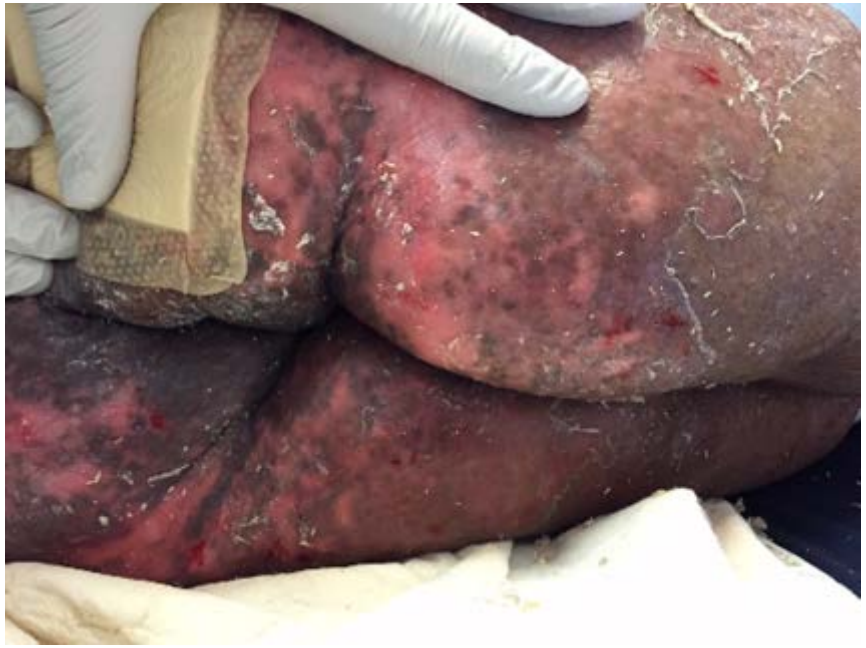
“Gifts” from home

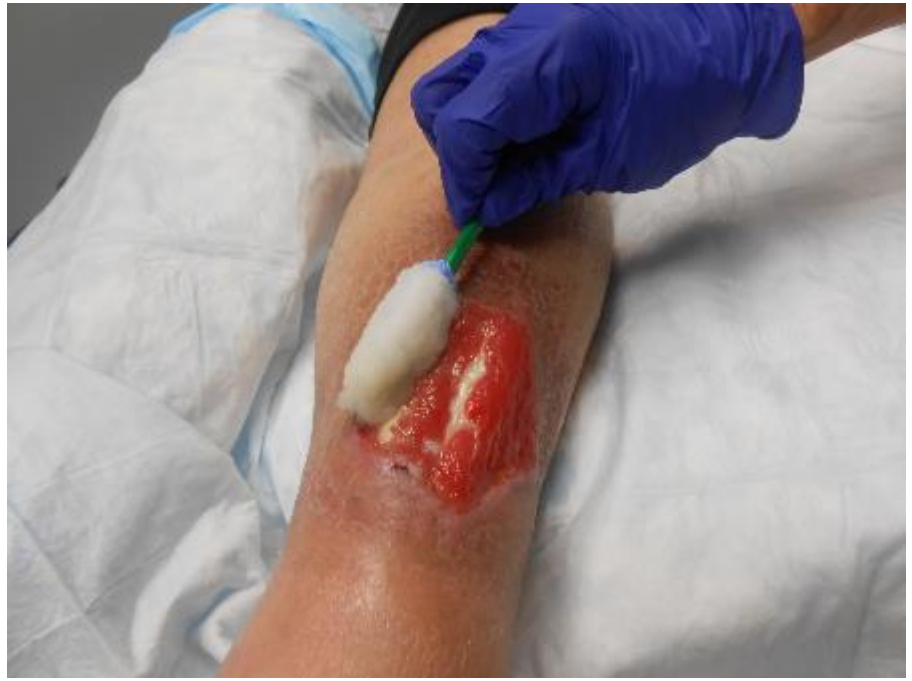


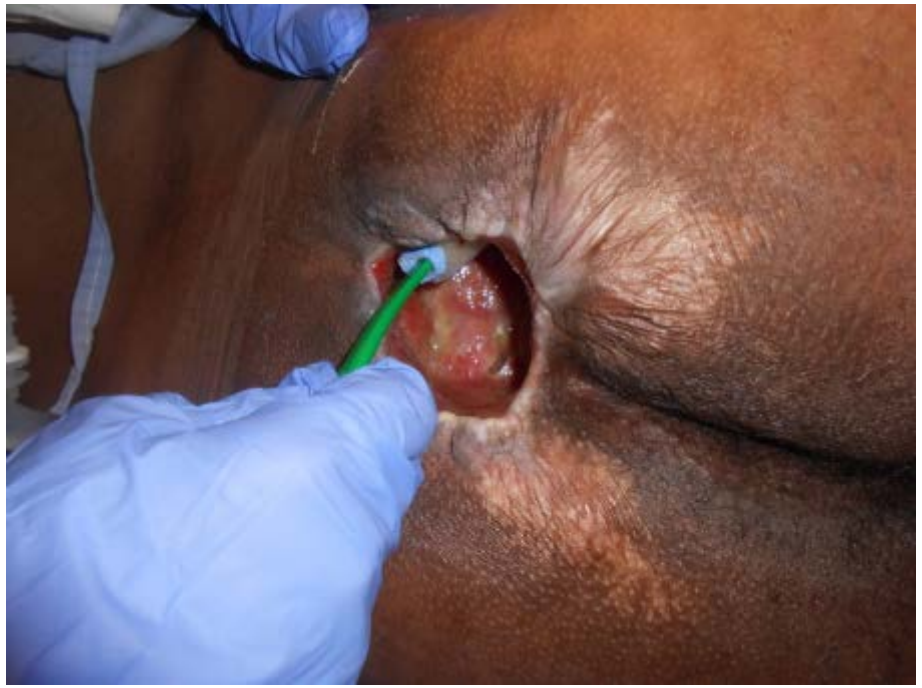


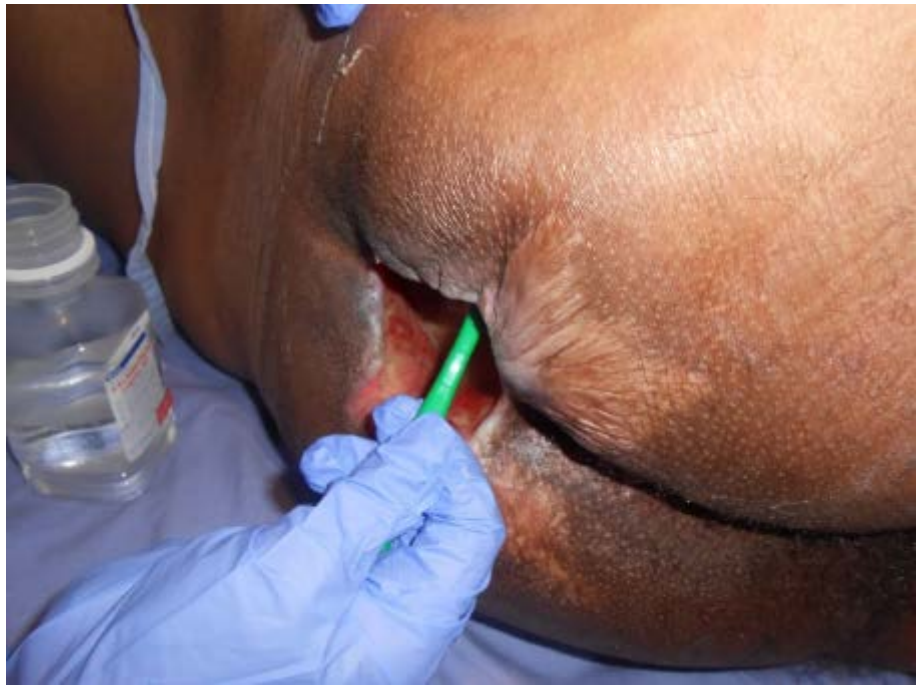
Non-Traumatic Skin Cleansing











Summary

- Effectively Removes and Manages Necrotic Tissue:
  - Can stand alone or used in conjunction with other debridement modalities
  - Puts debridement in the hands of the bedside nurse
  - May decrease time enzymes needed by increasing their effectiveness
  - Removes loose debris and necrotic tissue
- Manages Pain during Cleansing/Debridement
  - Shown to be effective alternative when patient not a candidate for other debridement modalities because of pain issues
- Manages Wound Bioburden/Biofilm
  - Physically disrupts biofilm to allow increased effectiveness of antimicrobials/antibiofilm agents
  - Decreases wound bioburden
- Assists with debridement/cleansing of undermined/tunneled areas
- Manages/debrides periwound hyperkeratosis/venous dermatitis

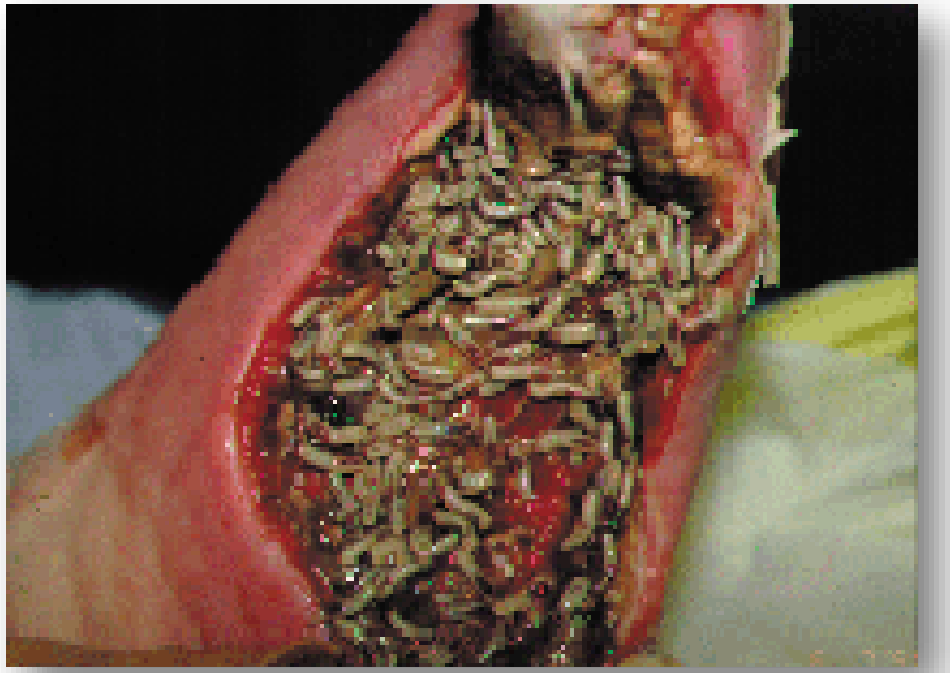
Biosurgical

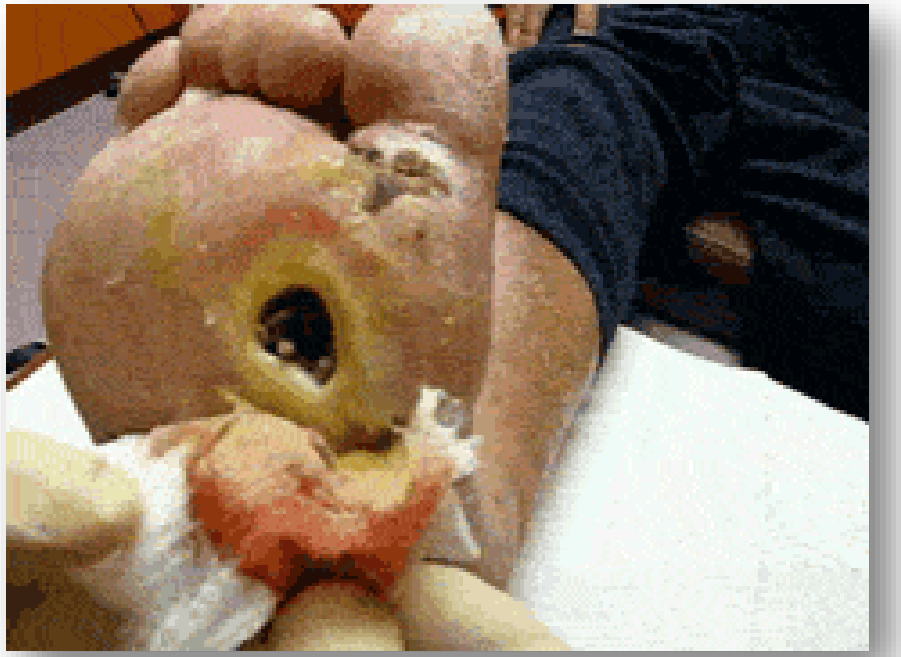
- Biologic debridement is the application of sterile, medical grade larvae (maggots) into the wound for the purpose of removing devitalized tissue, disinfection, and promotion of wound healing.
- Debridement occurs as larvae introduce proteolytic enzymes to promote rapid removal of devitalized tissue.

Wound, Ostomy and Continence Nurses Society. (2015). Methods of Wound Debridement: Best Practice for Clinicians. Mt. Laurel: NJ

Mechanisms of Action

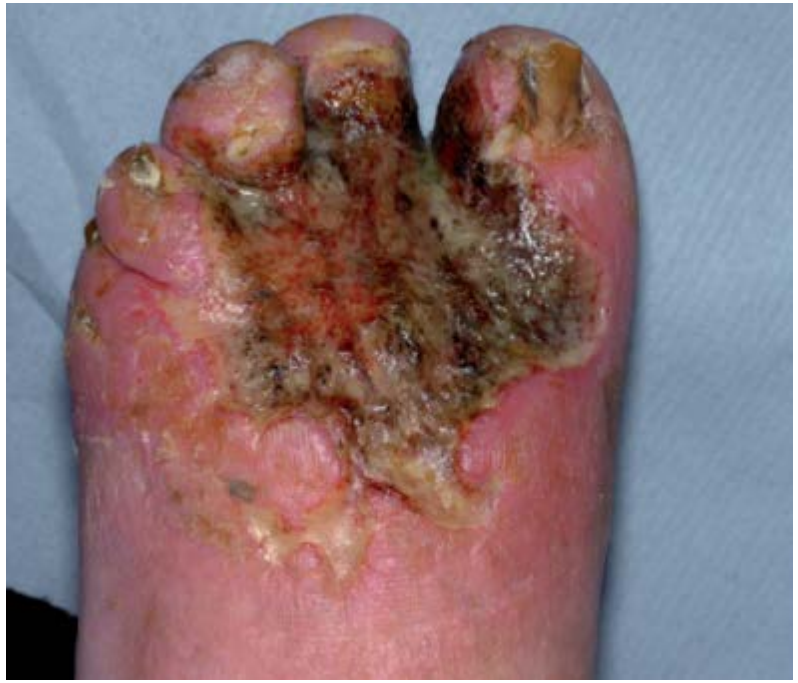
- Larvae secrete proteolytic enzymes which liquefy necrotic tissue
- Movement around surface of wound with “teeth”
- Actual ingestion of the tissue by the larvae
- Bacteria are destroyed in the alimentary tract due to antibacterial substance after ingestion of resident bacteria





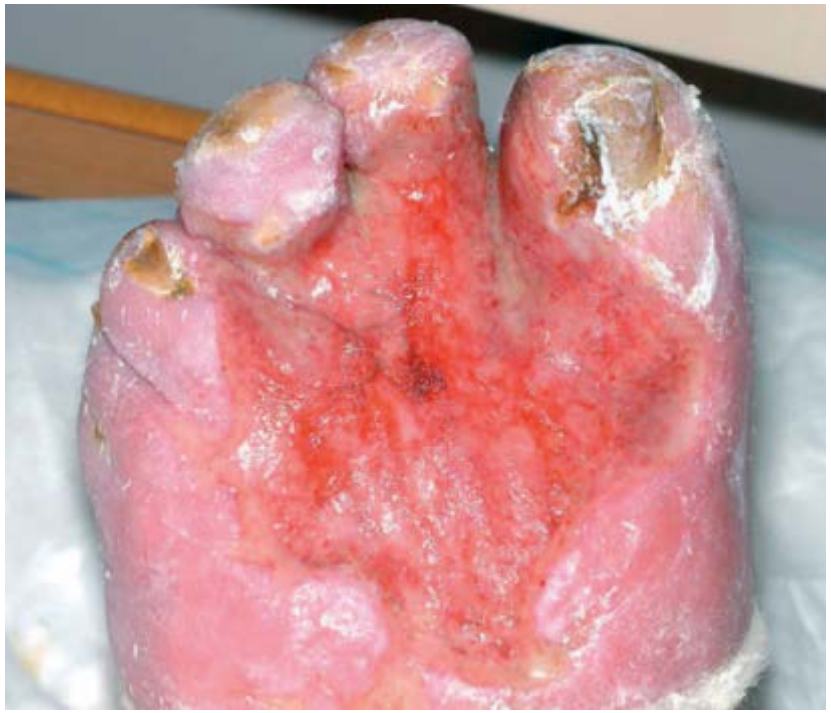
Bio-Bags

- Heat-sealed mesh pouch
- Contains maggots and hydrophobic foam spacer
- Allows for easy examination of wound
- Easy to apply
- 4-day treatment time



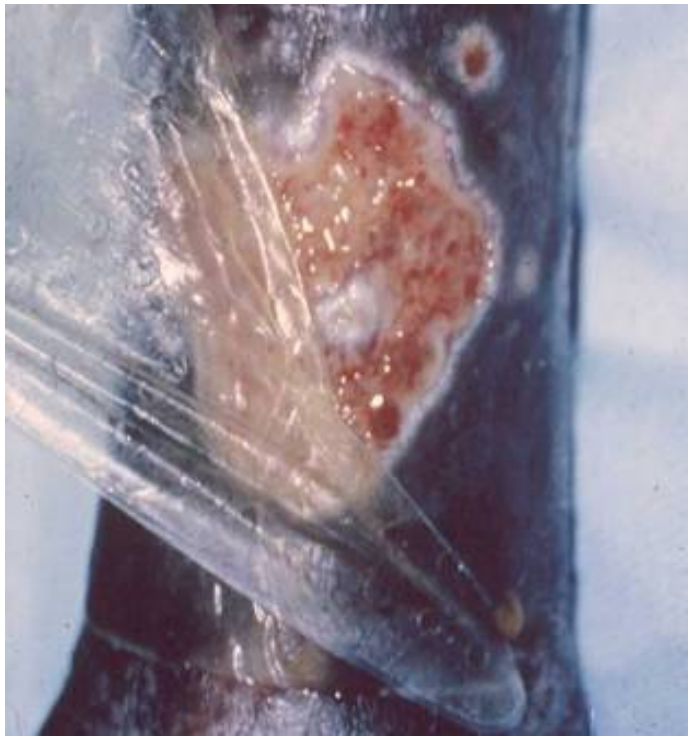
Baseline

After 1 treatment

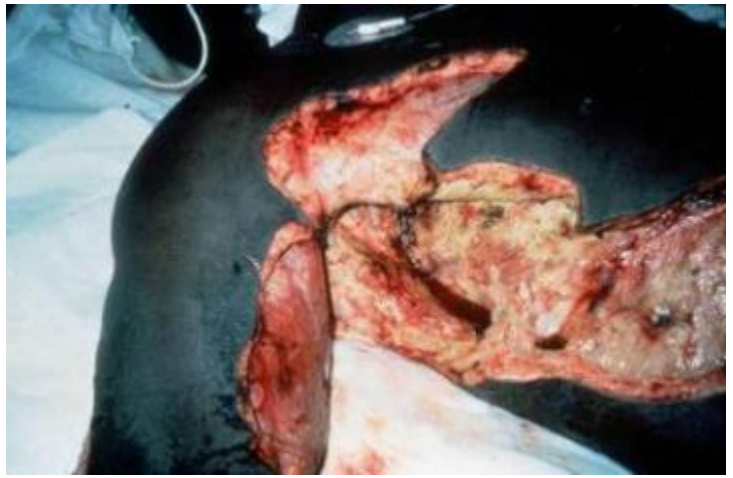


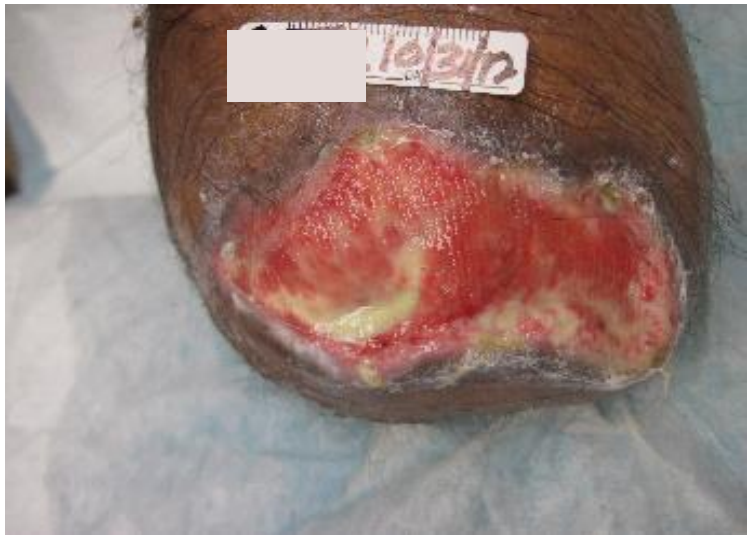
Autolytic Debridement

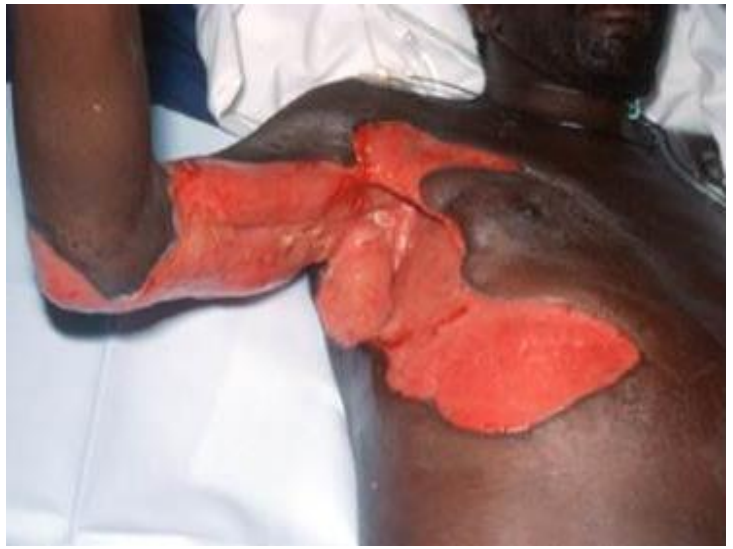
- Natural degradation of devitalized tissue utilizing proteolytic enzymes
- Achieved through use of moisture retentive dressings

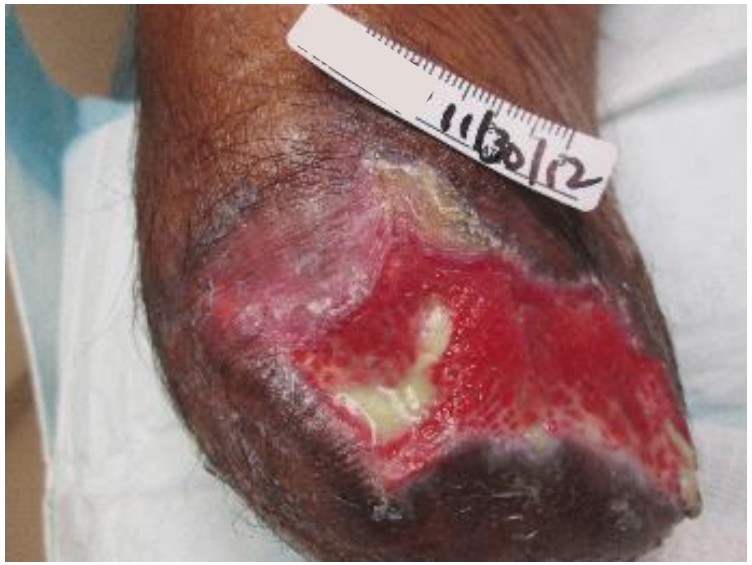


Autolytic - Hydrogel

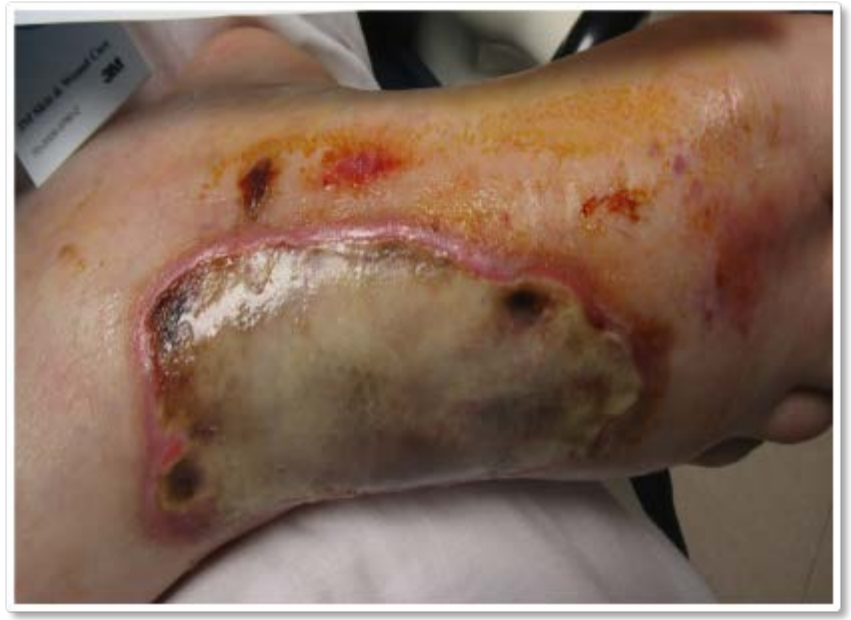




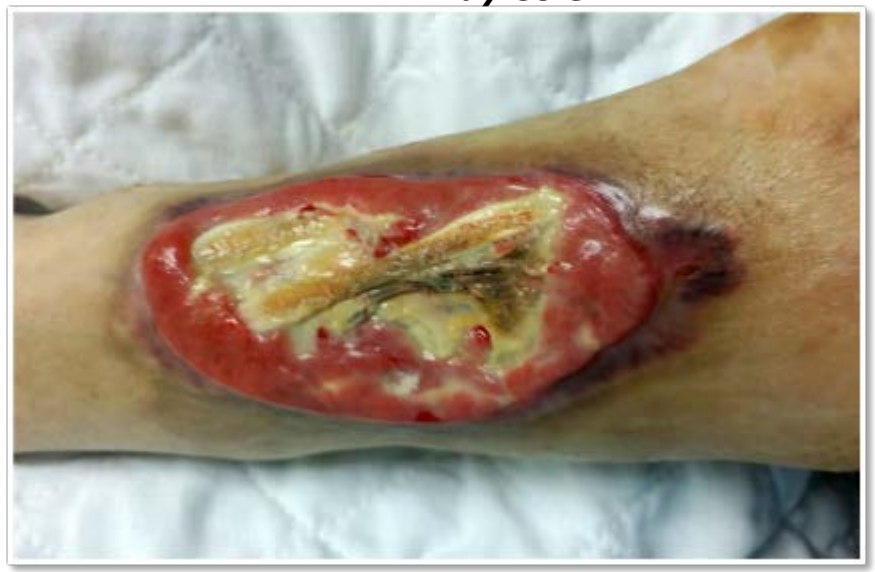


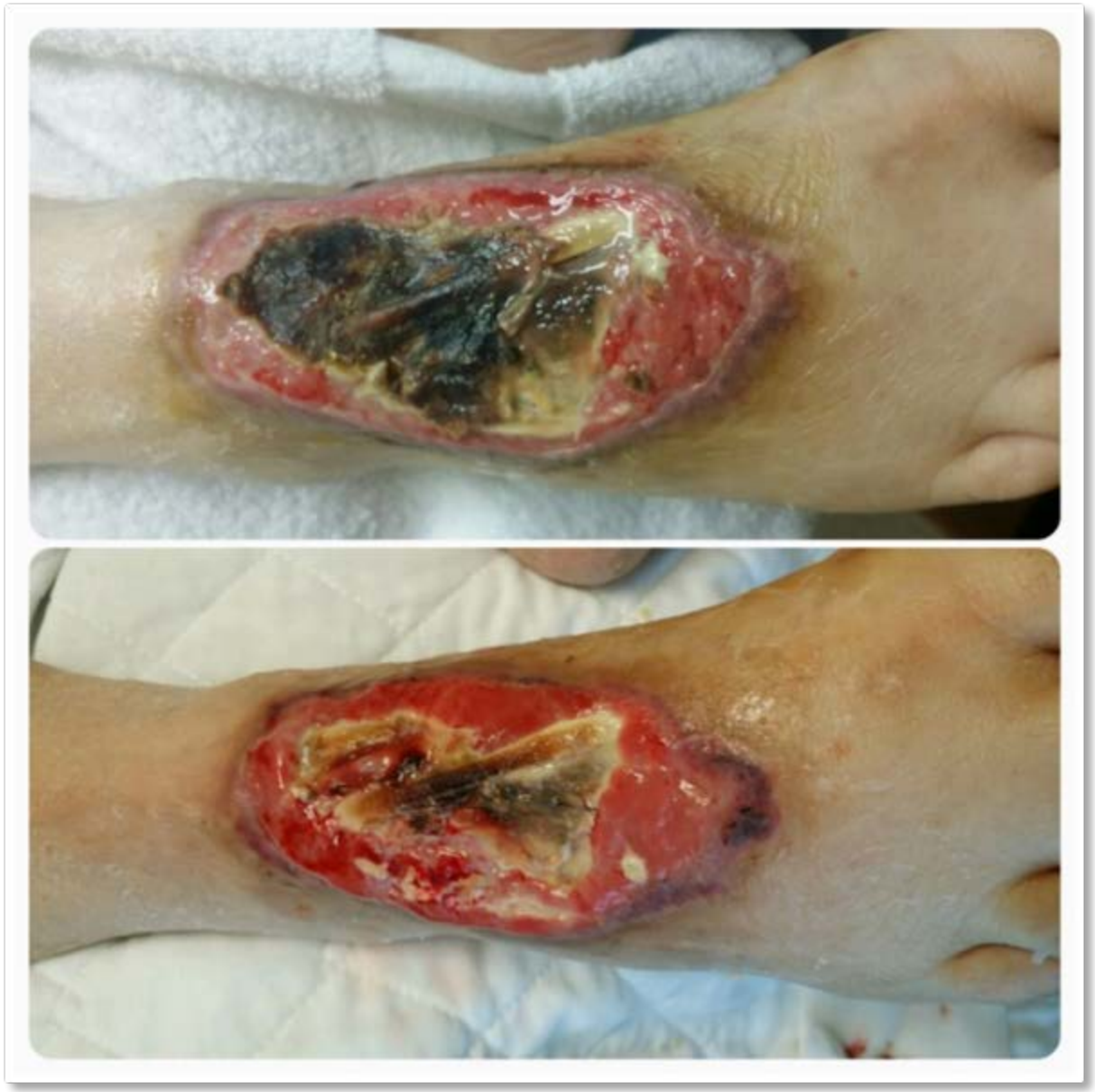


Autolytic - Surfactant



Dr. Windy Cole

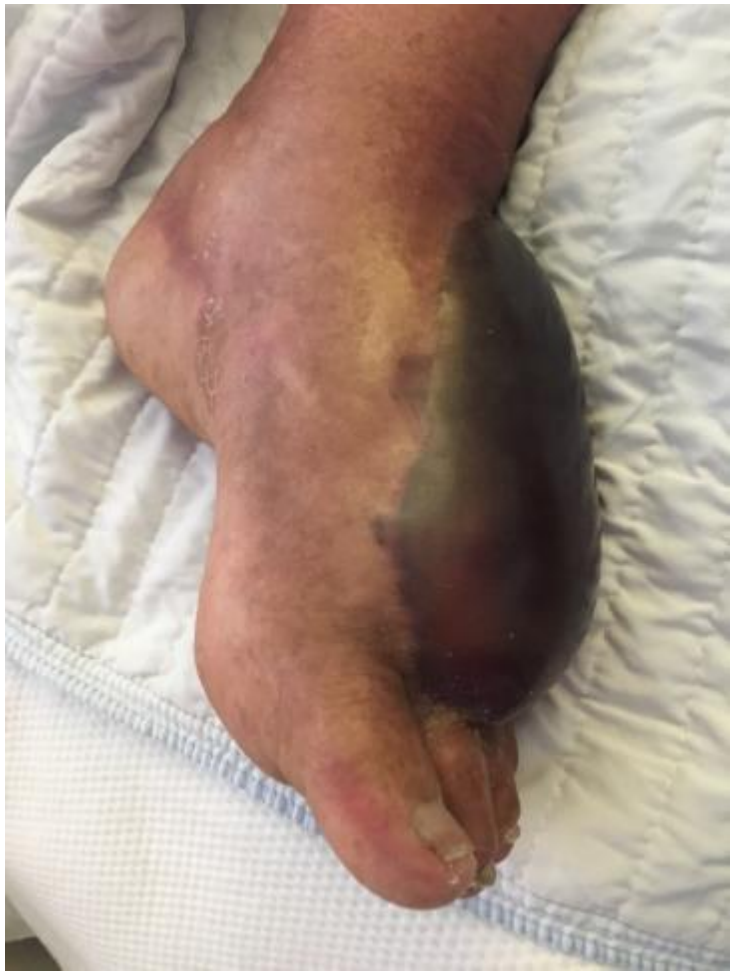


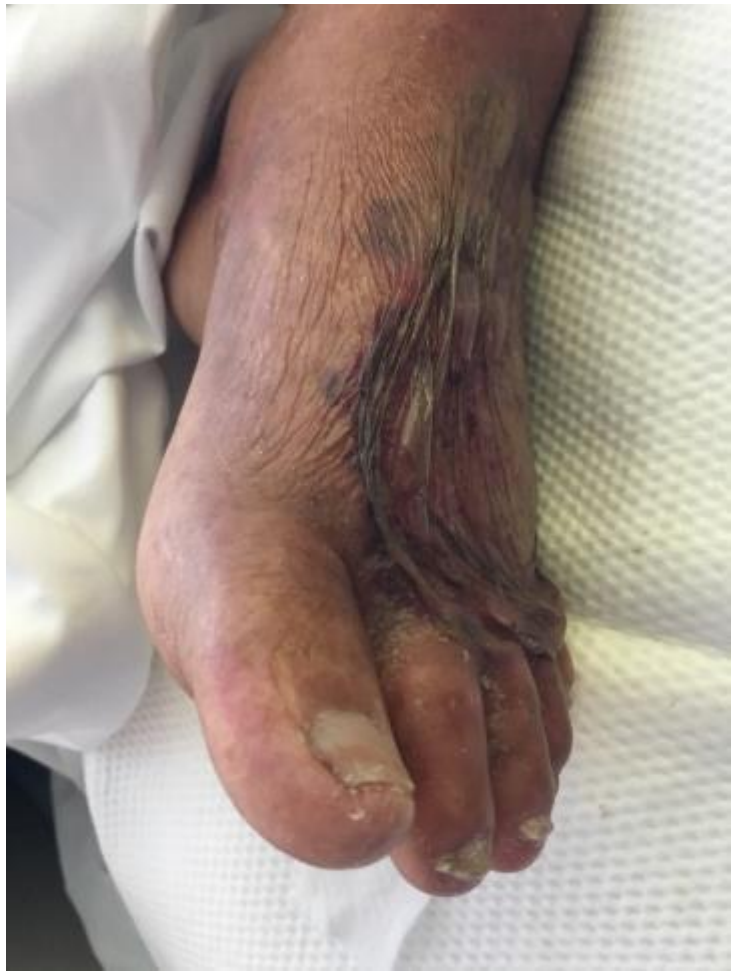


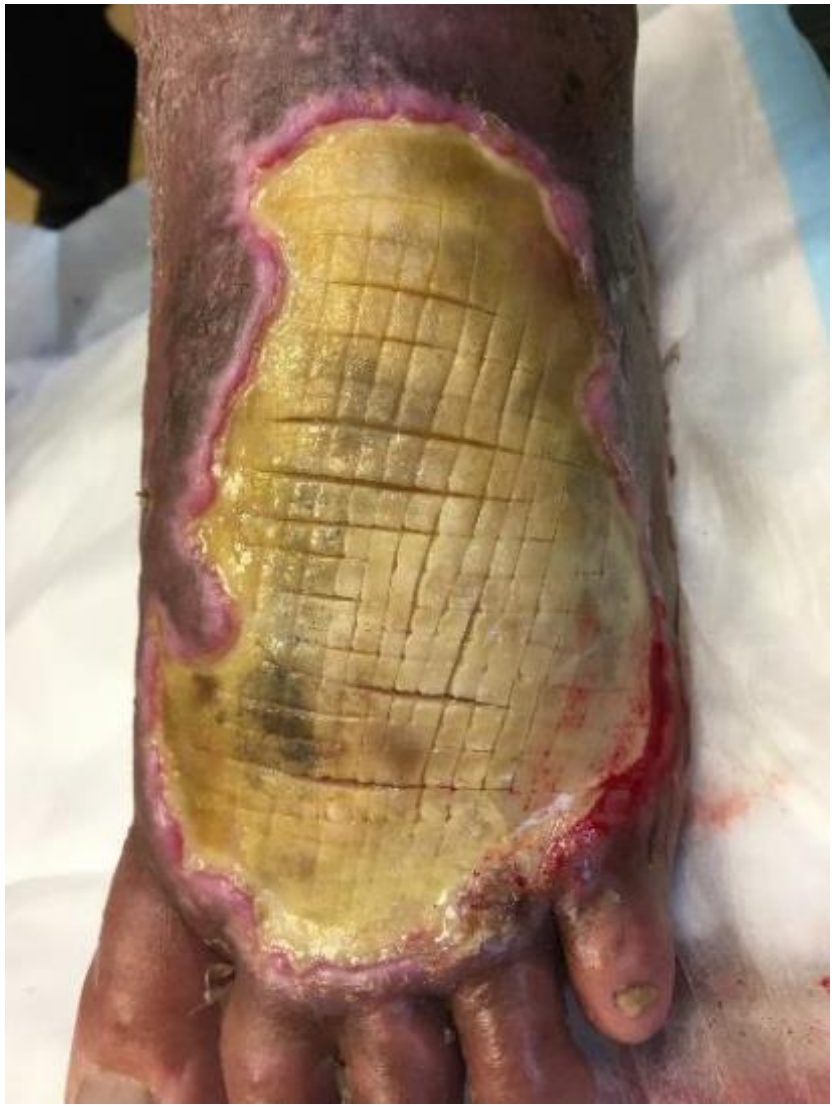
Enzymatic

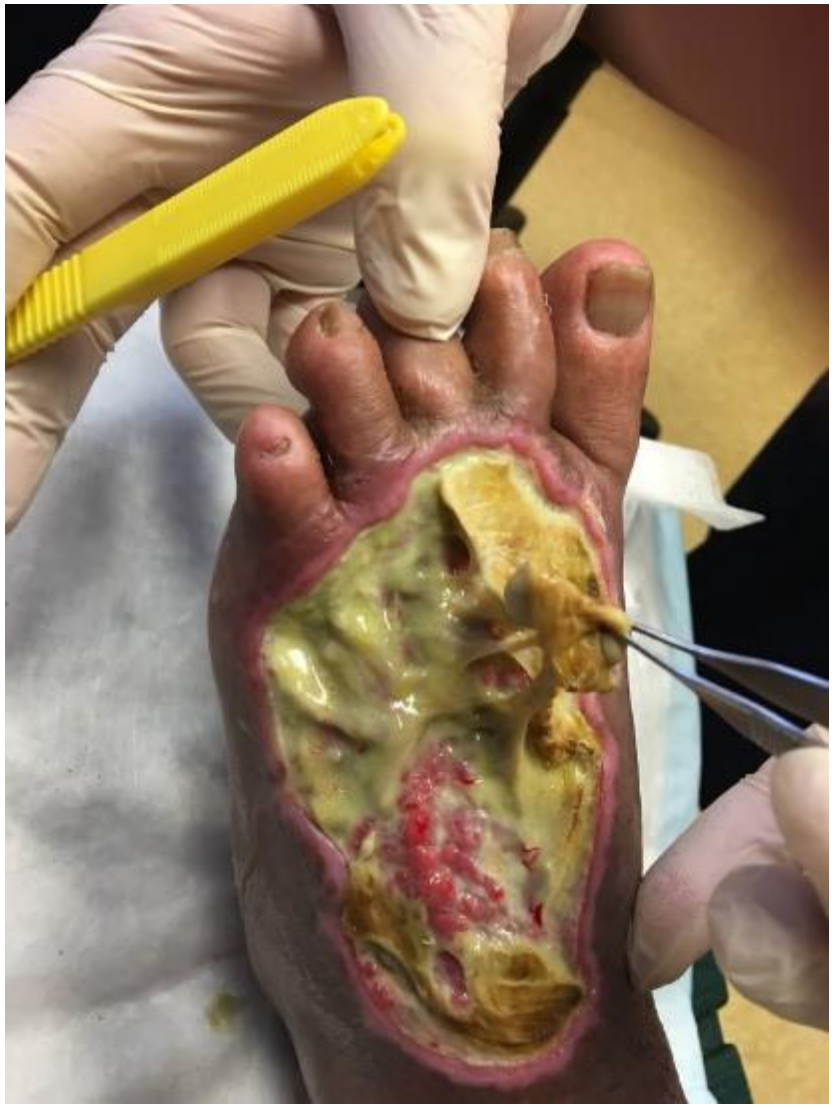
- Use of exogenously applied agent to work directly on the devitalized tissue or indirectly by dissolving the collagen that attaches the devitalized tissue to the wound bed, but have little to no effect on healthy tissue
- Collagenase is the only enzymatic agent approved by the Food and Drug Administration in the United States

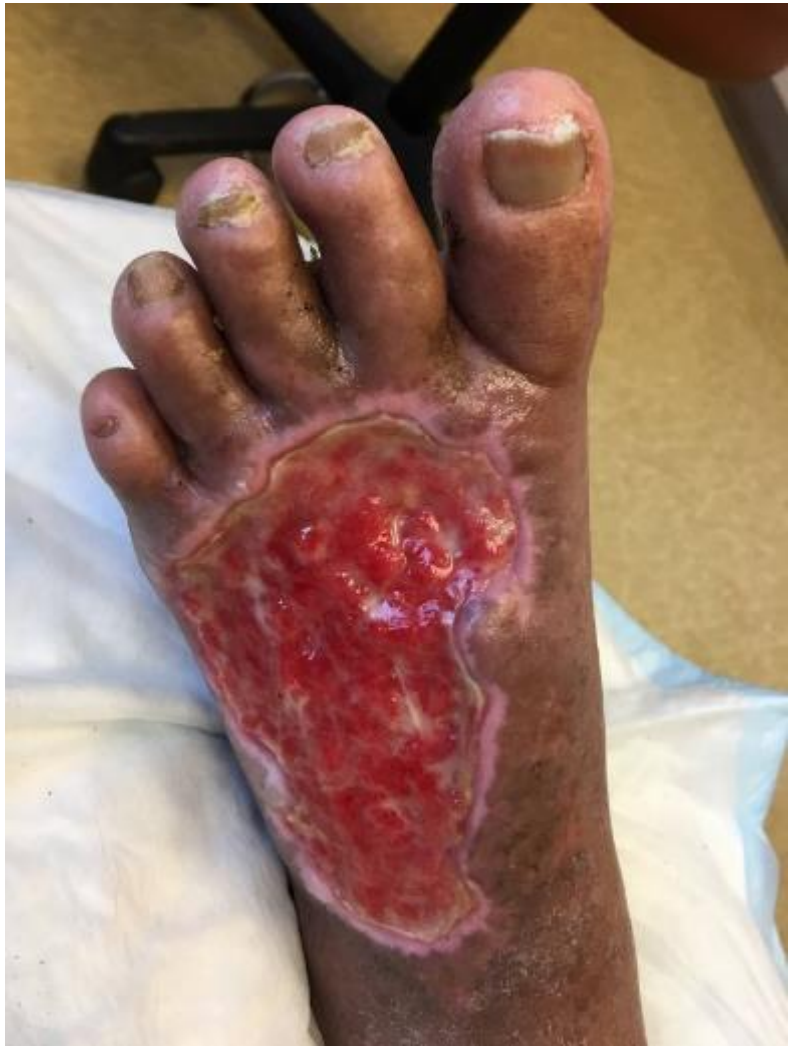
Huge Bulla Unknown Etiology

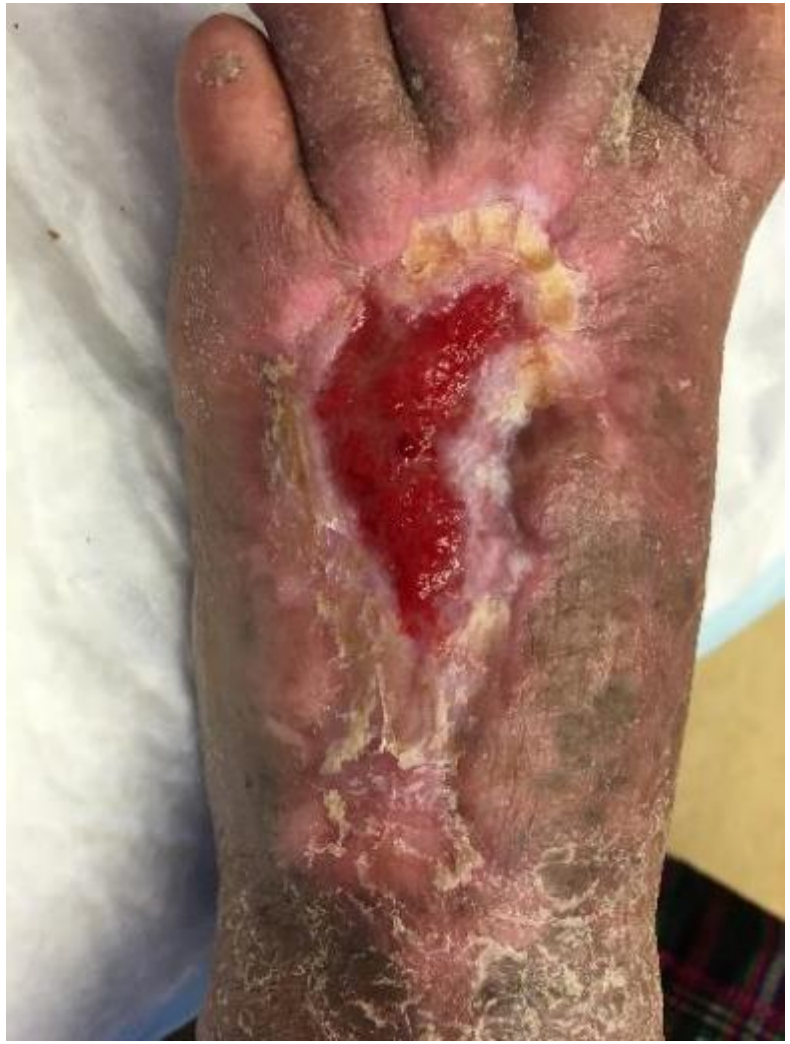












"T" – Tissue Debridement

Remove Non-Viable or Deficient Tissue

Goals are to remove necrotic tissue, microdebris, reduce bacterial burden

Strategy	Description	Examples
Surgical (Excisional/Sharp)	Removal by surgical instrument	Scalpel, scissors, hydrosurgery, lasers, curettes,
Mechanical	Removal of necrotic tissue by mechanical means	Wet- to dry dressings, hydrotherapy, ultrasound, abrasion
Biosurgical	Sterile larvae selectively digest necrotic tissue and bacteria	Sterile blowfly or housefly larvae
Autolytic	Uses the body's own enzymes to dissolve necrotic tissue; assisted with moisture-retentive dressings	Moisture retentive dressings
Enzymatic	Topical application of enzymes to liquefy necrotic tissue	Collagenase

The Wound Healing Society. Chronic Wound Care Guidelines. 2007. http://woundheal.org/documents/final_pocket_guide_treatment.aspx. Accessed March 29, 2017.

Summary

1. Biofilms are communities of bacteria encased in a self-produced matrix of polysaccharides, protein and DNA that provides high levels of tolerance to antibodies, antibiotics and antiseptics

2. Biofilms are present in a high percentage of chronic wounds and they impair healing by stimulating chronic inflammation, leading to elevated levels of proteases and ROS that degrade proteins that are essential for healing

3. Debridement with a monofilament pad reduces mature *Pseudomonas aeruginosa* biofilm on pig skin explants ~3-logs in ~6-log total CFUs

4. Biofilm Based Wound Care is part of Wound Bed Preparation (TIME) and emphasizes effective debridement

Wound Bed Preparation

- *Biofilms – impacted both "T" & "I" components*
- *Negative Pressure Wound Therapy (NPWT) – impacted "T" "I" and "M" components*
- *New topical dressings – impacted "I" component*
  - *Silver*
  - *Cadexomer Iodine*
  - *PHMB*
- *DNA-based identification of bacteria – impacted "I"*
- *Diagnostics for proteases – impacted "E"*
- *Leaper et al Int Wound J 9(sup 2) 1-19, 2012*

Extending the TIME concept: what have we learned in the past 10 years?*

David J Leaper, Gregory Schultz, Keryln Carville, Jacqueline Fletcher, Theresa Swanson, Rebecca Drake

Leaper DJ, Schultz G, Carville K, Fletcher J, Swanson T, Drake R. Extending the TIME concept: what have we learned in the past 10 years? Int Wound J 2012; 9 (Suppl. 2):1–19

ABSTRACT

The TIME acronym (tissue, infection/inflammation, moisture balance and edge of wound) was first developed more than 10 years ago, by an international group of wound healing experts, to provide a framework for a structured approach to wound bed preparation, a basis for optimising the management of open chronic wounds healing by secondary intention. However, it should be recognised that the TIME principles are only a part of the systematic and holistic evaluation of each patient at every wound assessment. This review, prepared by the International Wound Infection Institute, examines how new data and evidence generated in the intervening decade affects the original concepts of TIME, and how it is translated into current best practice. Four developments stand out: recognition of the importance of biofilms (and the need for a simple diagnostic), use of negative pressure wound therapy (NPWT), evolution of topical antiseptic therapy as dressings and for wound lavage (notably, silver and polyhexamethylene biguanide) and expanded insight of the role of molecular biological processes in chronic wounds (with emerging diagnostics and theranostics). *Tissue*: a major advance has been the recognition of the value of repetitive and maintenance debridement and wound cleansing, both in time-honoured and novel methods (notably using NPWT and hydrosurgery). *Infection/inflammation*: clinical recognition of infection (and non infective causes of persisting inflammation) is critical. The concept of a bacterial continuum through contamination, colonisation and infection is now widely accepted, together with the understanding of biofilm presence. There has been a return to topical antiseptics to control bioburden in wounds, emphasised by the awareness of increasing antibiotic resistance. *Moisture*: the relevance of excessive or insufficient wound exudate and its molecular components has led to the development and use of a wider range of dressings to regulate moisture balance, and to protect peri wound skin, and optimise healing. *Edge of wound*: several treatment modalities are being investigated and introduced to improve epithelial advancement, which can be regarded as the clearest sign of wound healing. The TIME principle remains relevant 10 years on, with continuing important developments that incorporate new evidence for wound care.

Key words: Chronic wounds • Debridement • Infection • Inflammation • Moisture balance • TIME • Wound bed preparation

INTRODUCTION

The TIME acronym was first developed more than 10 years ago, by an international group of wound healing experts, to provide a framework for a structured approach to wound bed preparation (1). This concept was adopted from a principle used in plastic surgery to ensure optimal preparation of a recipient

Authors: DJ Leaper, MD, CVM, FRCS, FACS, FLS, Section of Wound Healing, Institute for Translation, Innovation, Methodology and Engagement, Cardiff University, Cardiff, UK; G Schultz, PhD, Department of Obstetrics and Gynecology, Institute for Wound Research, University of Florida, Gainesville, FL, USA; K Carville, RN, STN(Cnsl), PhD, Silver Chain Nursing Association & Curtin University, Osborne Park, Western Australia; J Fletcher, MSc, BSc, PGCE, RN, FHEA, Institute for Translation, Innovation, Methodology and Engagement, Cardiff University, Cardiff, UK; T Swanson, RN, NPWM, AAWCo Nursing (USA), CC (WMDM), PGCE(Postgr), PGDip HSc (Nursing), MedSci HSc (Nursing), International Wound Infection Institute, South West Healthcare, Warrnambool, Victoria, Australia; R Drake, BSc, London, UK

Address for correspondence: Prof. David J Leaper, Section of Wound Healing, Institute for Translation, Innovation, Methodology, and Engagement, Cardiff University, Cardiff CF14 4XN, UK

E-mail: profdavidleaper@doctors.co.uk

*Sponsored by Smith & Nephew Wound Management.

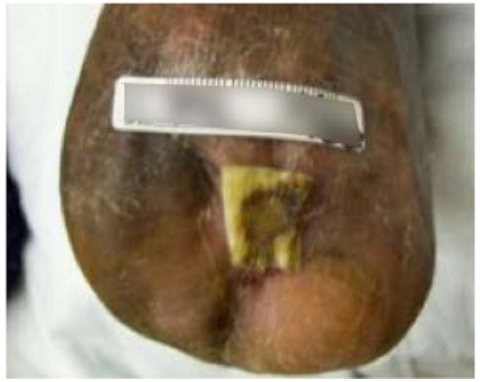
Principles of Biofilm-Based Wound Care

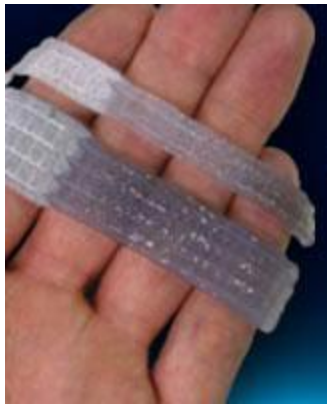
1. **Frequent debridement** of wounds to physically remove biofilm communities
2. Use an **effective microbicidal dressing** after debridement to prevent reformation of biofilms
3. **Alter topical and systemic antimicrobial treatments** to prevent emergence of dominant bacteria from polymicrobial populations; utilize DNA bacterial identification techniques
4. **Biofilm-Based Wound Care** is part of **Wound Bed Preparation (TIME)**

Wolcott RD, et al. *J Wound Care.* 2008;17(4):145-155

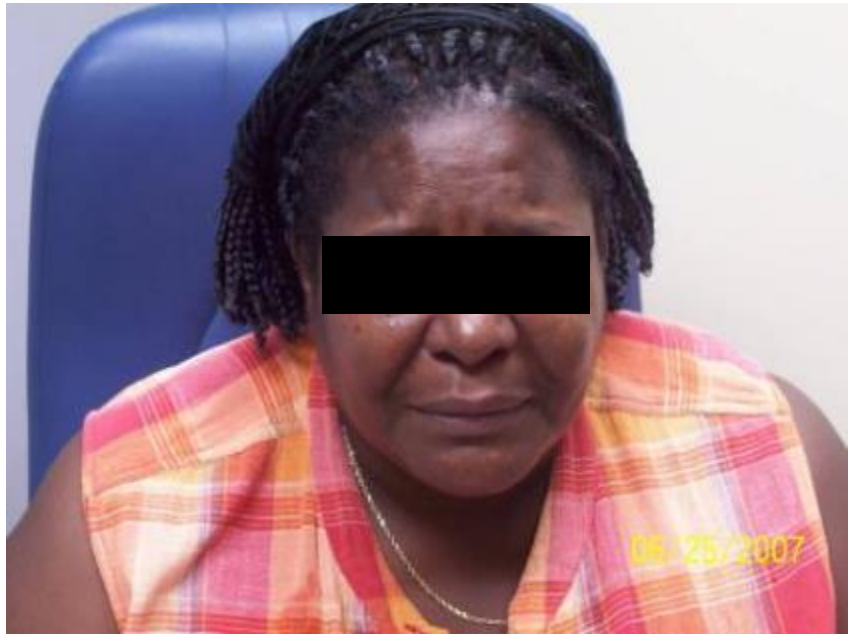
Bacterial Burden

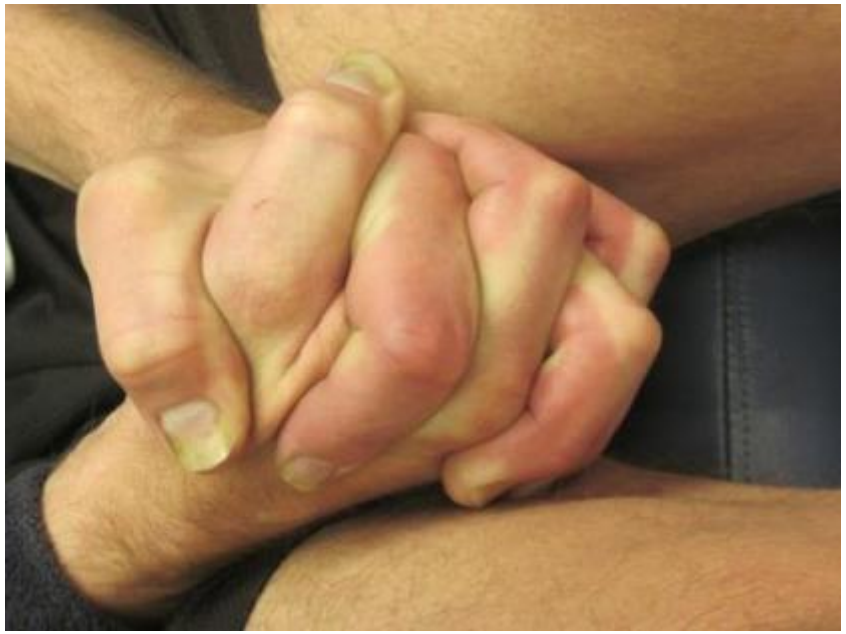
- Silver (all dressing categories come with Ag option)
- Cadexomer Iodine
- Pigmented Foam
- PHMB (Polyhexamethylene Biguanide)
- Honey
- DACC





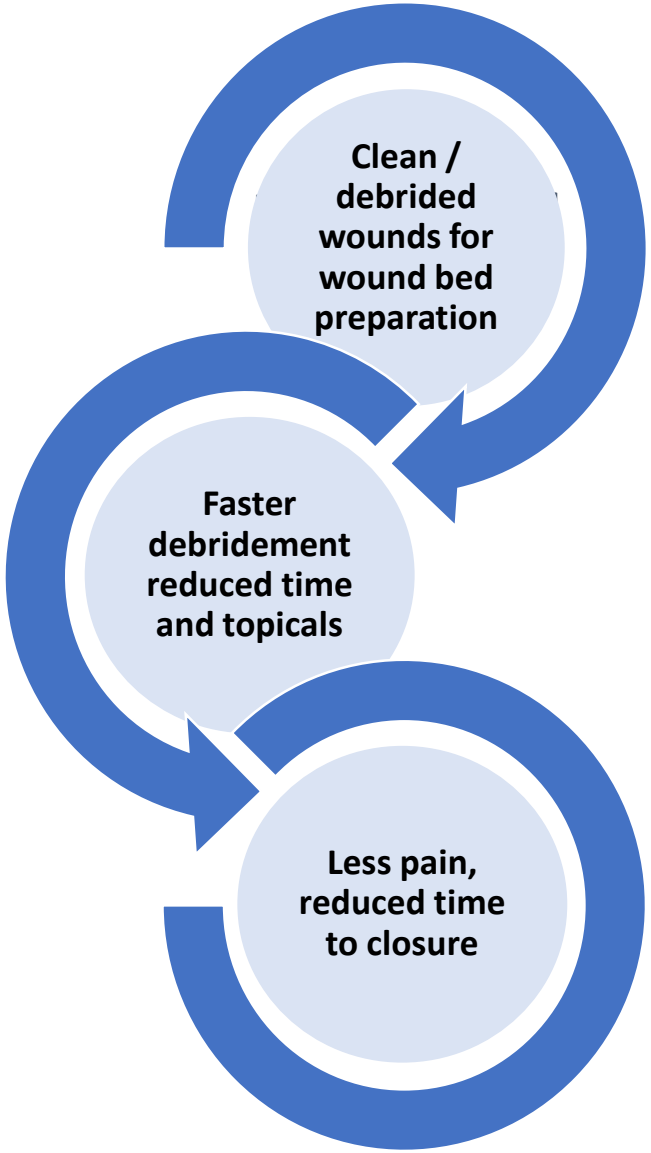
Address and Manage Pain





Healthcare Reform

Triple Aim



- Institute for Healthcare Improvement. www.ihi.org/engage/initiatives/TripleAIM/Pages/default.aspx. Accessed March 28, 2017.

Thank You!!!