



**Wound
bed prep
ROCKS!**

**We're dying
to hear about
DIMES!**

Principles of Wound Care: ***DIMES and Wound Bed Preparation***

Cynthia A. Fleck, MBA, BSN, RN, ET/WOCN, CWS, DNC, DAPWCA, FACCWS

President and Chairman of the Board, American Academy of Wound Management (AAWM)

Board of Directors, The Association for the Advancement of Wound Care (AAWC)

Medical Advisory Board, American Professional Wound Care Association (APWCA)

PRESENT Diabetes, Past Nursing Editor

Advances in Skin and Wound Care columnist

Advisory Board: ECPN, Long-Term Care Management, Home Health Product News

President, CAF Clinical Consultant

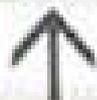
Vice President, Clinical Marketing, Medline Advanced Skin and Wound Care

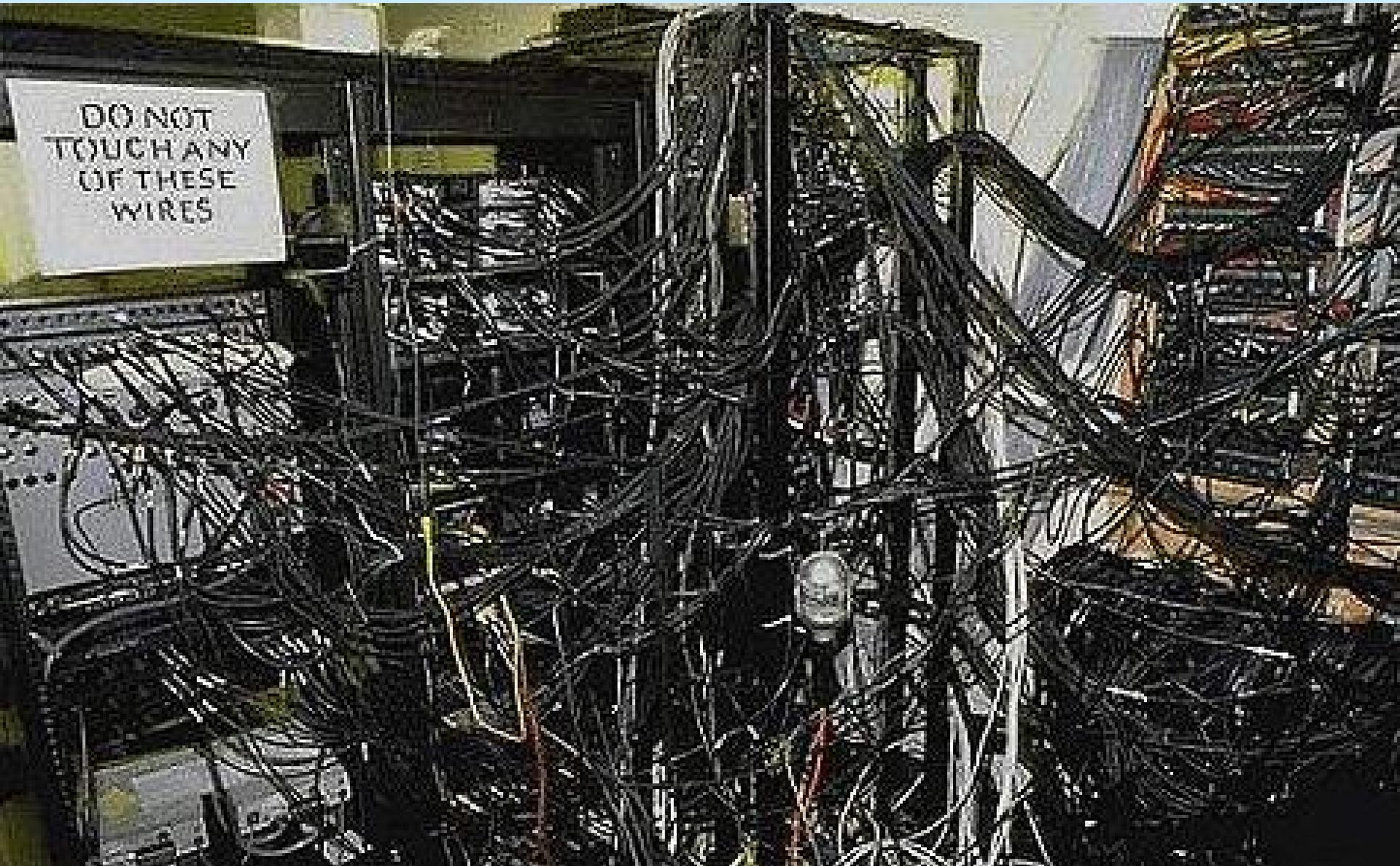


化粧室は後方へ

For Restrooms,
Go back toward your behind.

14





DO NOT
TOUCH ANY
OF THESE
WIRES

STOP

**NO
STOPPING
ANY
TIME**



LEADED GASOLINE	1.46
UNLEADED PLUS GASOLINE	1.55
PREMIUM UNLEADED GASOLINE	1.63
DIESEL #2 CASH PRICE	1.34
DIESEL #2 CREDIT PRICE	1.37



**FLY
TRIP**

oker
RESTAURANT & BUFFET



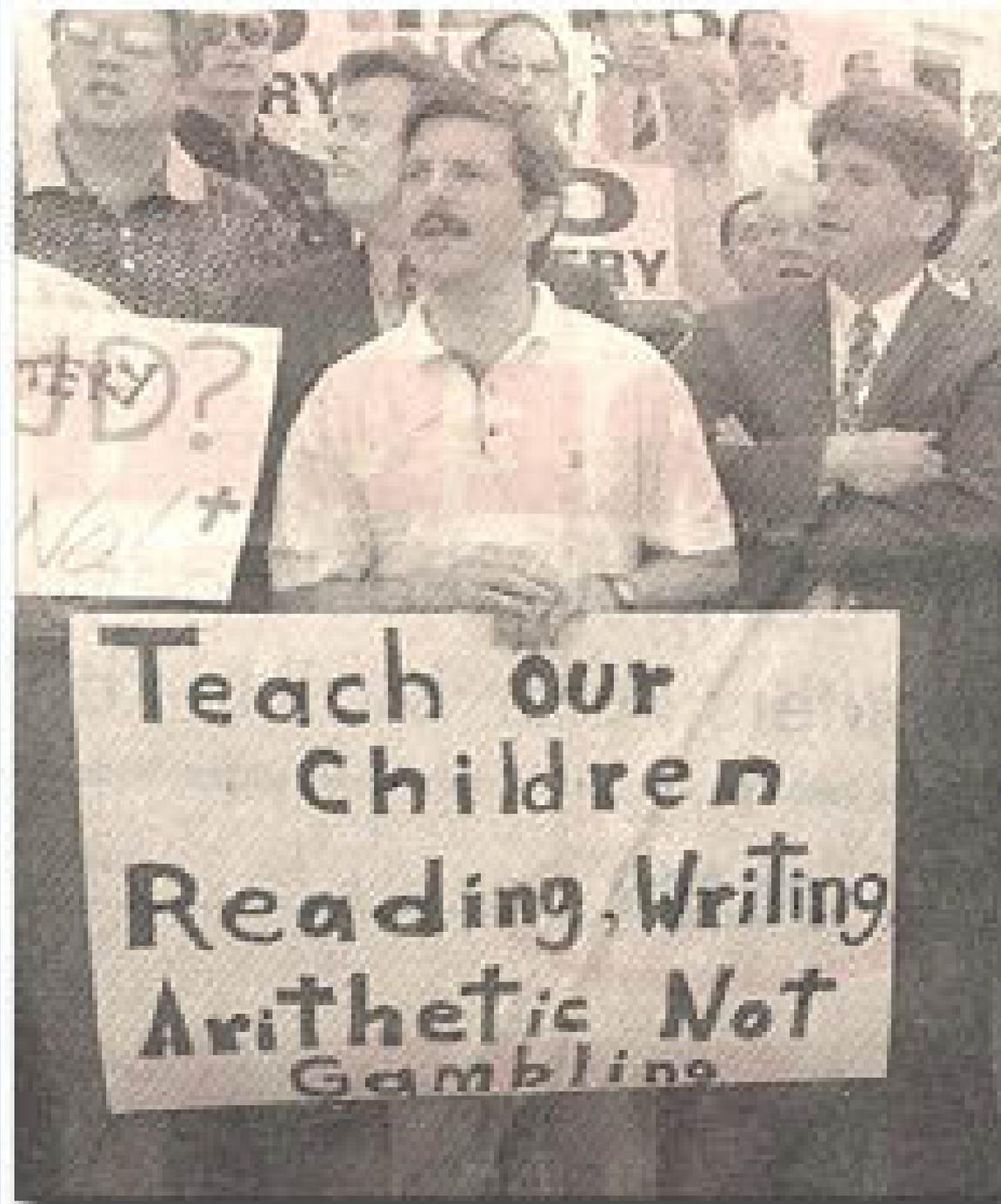


SPEED
LIMIT
50

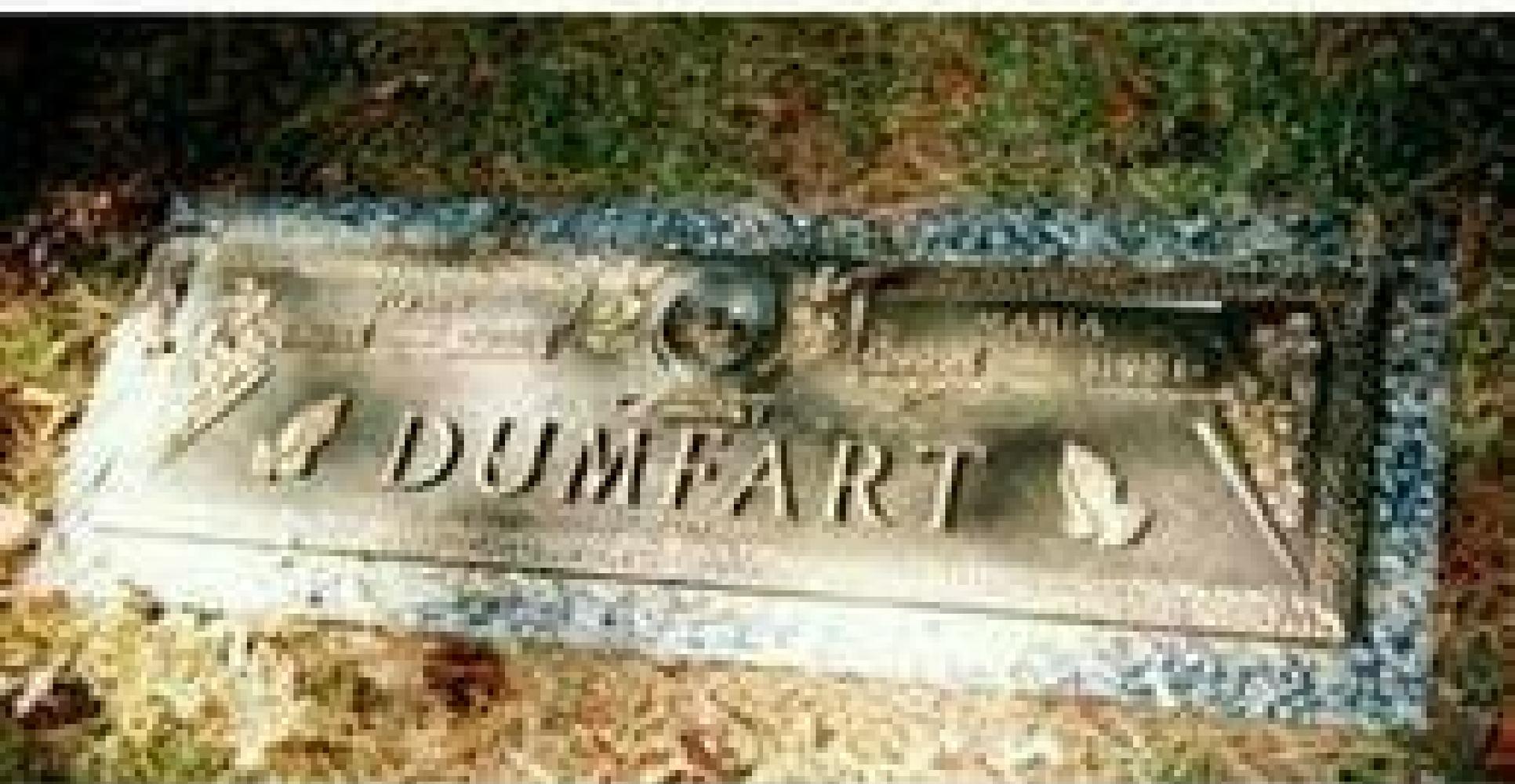
YOUR SPEED

167

POLICE



Teach our
Children
Reading, Writing
Arithmetic Not
Gambling

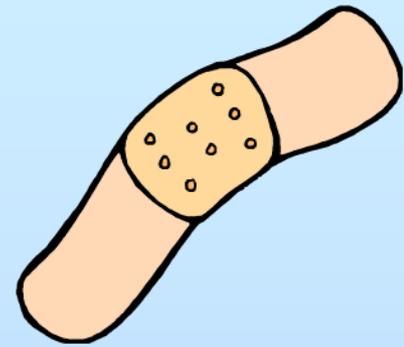


DUMFART





Chronic Wounds



- An insult or injury that has failed to proceed through an orderly and timely repair process
- Thought to be “stuck” in the inflammatory or proliferative phase.
- Key cells become senescent or go to “sleep”
- Patient with a chronic wound generally presents with a host of factors that impede the healing process
- Understanding and correcting the barriers to healing will help kick-start the healing process again.

Expect to see progress toward wound healing within 2 - 4 weeks of initiation of treatment.

Van Rijswijk L, Braden B. (1999) Pressure ulcer patient and wound assessment: an AHCPR clinical practice guideline update. *Ostomy and Wound Management* vol. 45 #1A Supplement.



Predictors of healing

- Flanagan noted that 20% to 40% reduction in 2 and 4 weeks is likely to be a reliable predictor of healing¹.
- Sheehan, et al found that a 50% reduction at week 12 is a good predictor for persons with diabetic foot ulcers².
- Margolis, et al found that if the wound is not 30% smaller by week 4, it will not heal by week 12³.



1. Flanagan M. Wound measurement: can it help us to monitor progression to healing? *J wound Care* 2003;12:189-94.
2. Sheehan ZP, Jones P, Caselli A, et al. Percent change in wound area of diabetic foot ulcers over a 4-week period is a robust predictor of complete healing in a 12-week prospective trial. *Diabetes Care* 2003;26:1879-82.
3. Margolis DJ, Verlin JA, Strom BL. Risk factors associated with the failure of a venous leg ulcer to heal. *Arch Dermatol.*1993;28(3):418-421.

Stalled healing



Dow G. Ostomy/Wound Management 49(5A):8-13, May 2003.

- A chronic wound with an increased bacterial load can delay healing
- Microorganisms responsible for wound infection:
 - prolong wound progress
 - destroy cells by competing for O₂
 - release toxins and metabolic products that damage tissue, causing necrosis and ultimately, poor wound healing.

What's happening at a cellular level?

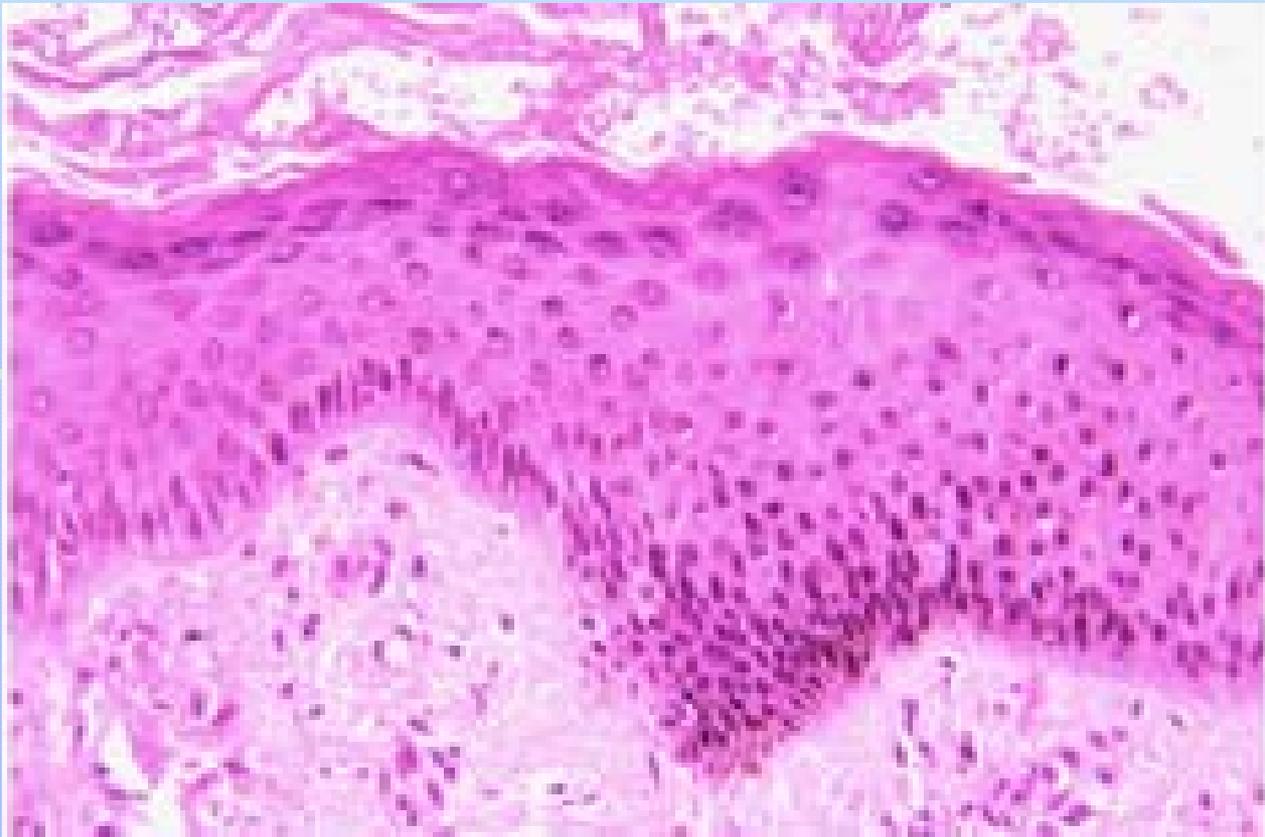


Photograph courtesy of R. Gary Sibbald, MD

- Increased bacterial burden triggers the release of excess vascular endothelial growth factor (VEGF), producing excessive but abnormally weak vascular endothelial buds.
- The subsequent granulation tissue can easily be digested by MMPs and appears bright red and “friable”.

Biopsy

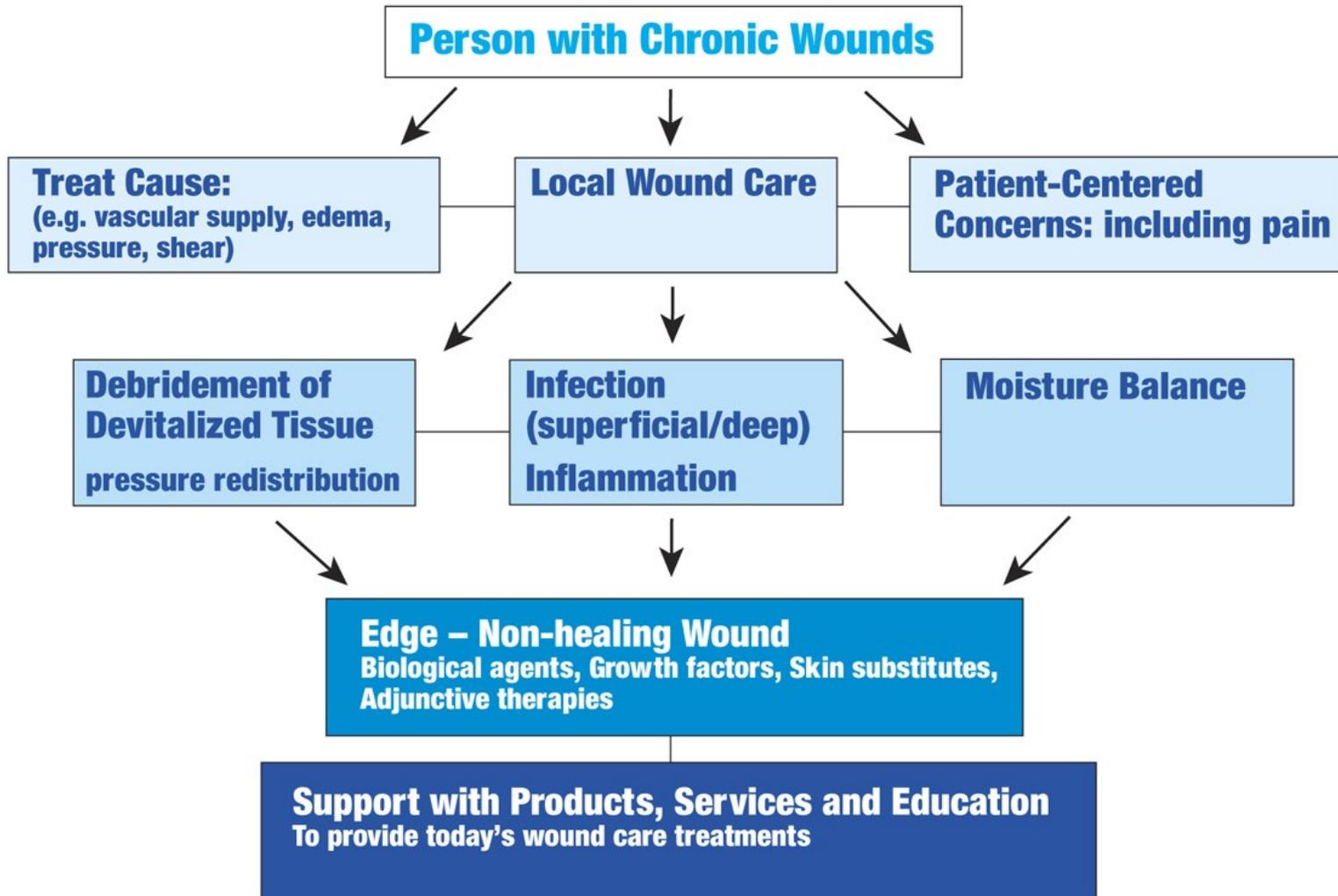
- If no healing in 12 weeks, biopsy wound and edge.





D.I.M.E.S.

Wound Bed Preparation Paradigm



What is necessary for a wound to heal “normally”?

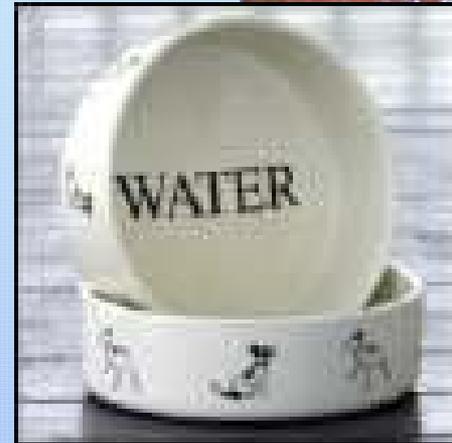


- Early appropriate treatment
- Functioning immune system
- Adequate blood flow to area
- Chronic disease under control
- Understanding of expected timing of healing for individual and wound
- ***Control of bacterial bioburden, MMPs and Elastase***

“I” - Infection

Biofilm

- Surface associated microbial community
- Matrix of assorted types of bacteria
 - tooth plaque, pet's water bowl, wounds
- Form polysaccharide coating on top
 - With nucleic acids and proteins
- Most bacteria in moist environments are found in Biofilm (deep flora)
- **Biofilm accounts for 80% of human infections¹**



1. NIH Guide. Research on Biofilm. Available from <http://grants.nih.gov/grants/guide/pa-files/PA-98-070.html>.

2. Fleck CA. Differentiating MMPs, Biofilm, Endotoxins, Exotoxins and Cytokines. *Advances in Skin and Wound Care*, March 2006;19(2):77-81.

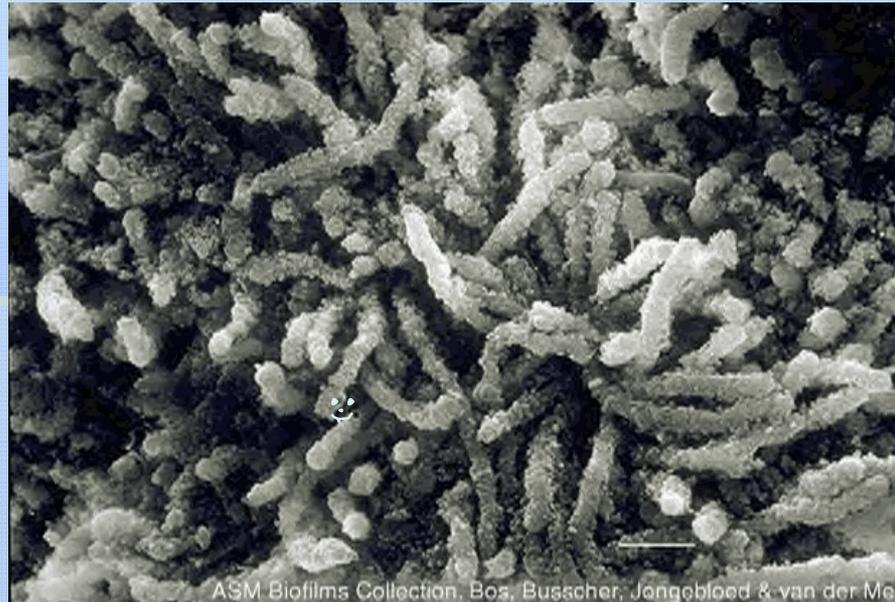
Why is Biofilm such a big problem?

- Impermeable to topical and systemic antimicrobial/antibiotic therapy
 - Demonstrates increased resistance to cellular and chemical attack
- Bacteria under protection of polysaccharide coating exchanges genetic antibiotic resistance info.
- Rapid emergence of antibiotic resistance to even new Rx's



Biofilm

- Macrophages, phagocytes and WBC's do not recognize biofilm as bacteria.
- Fundamental cause of chronic wound infections¹



ASM Biofilms Collection. Bos, Busscher, Jongbloed & van der Mei

1. Percival SL, Bowler P. Biofilms and Their Potential Role in Wound Healing. WOUNDS 2004;7:234-240

Wound Cleansers

- Normal saline?
- Recommended range of wound cleansing is 4-15 psi
AHCP Pressure Ulcer Treatment Guidelines, 1994
- 8 psi optimum
 - 35cc syringe and 19G angiocath
- Non-cytotoxic, non-ionic surfactant
Rodeheaver, 1999
- Antimicrobial cleansers with quaternary ammonium compounds such as BZK



Abandon Wet-to-Dry and moist gauze “therapy”!



- Archaic 20th Century wound care
- Painful
- Non-selective
- Gauze is traumatic to granulating wound beds
- Cotton fibers cause foreign body reaction²
- Advanced products are now available
- Bacteria can travel through over 64 layers of gauze¹
- Pass the word...

1. Ovington LG. Hanging wet to dry out to dry. *Home Healthcare Nurse*, 2001; 19(8):477-84.

2. Lawrence JC. Dressings and wound infection. *Am J Surg*. 1994;167(1 supp A):21S-24S.

COLLECTOR'S EDITION SERIES

BRUCE WILLIS

THE #1
THRILLER OF
ALL TIME!

THE SIXTH SENSE

DVD

“See dead people”

A close-up photograph of a human eye that has been severely injured. The eye is surrounded by a large amount of bright red blood, which is smeared across the surrounding skin. The eye itself is dark and appears to be in a state of shock or trauma. The background is a light blue gradient with a white grid pattern.

"I see dead tissue."

Maintenance debridement?

- Biofilm-embedded bacteria are found in the majority of chronic wounds (60%).
- Once maintenance debridement has begun, it should not be discontinued just because the wound “looks good”, even to an experienced practitioner.
- The problem - many enzymatic debriders no long available.

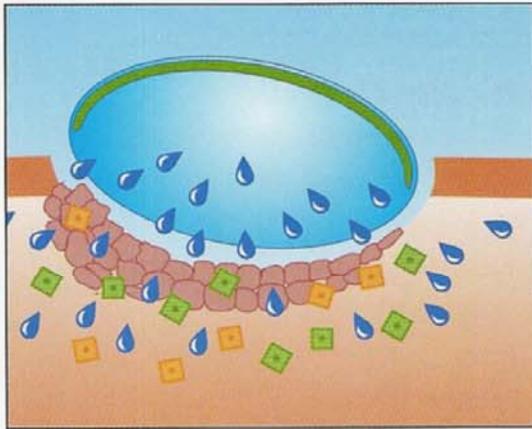


“D” – Debridement Polyacrylate Dressing

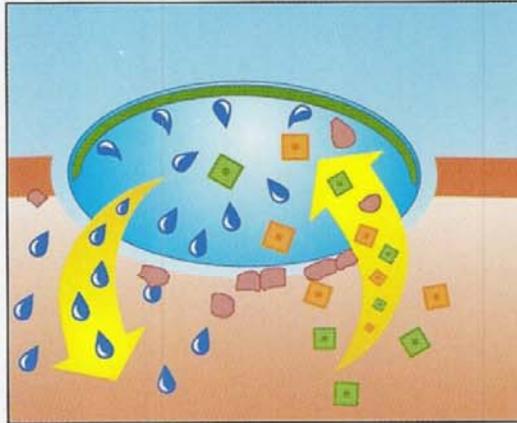
- Polymer gel encased in non-adherent polypropylene material
- Activated with Ringer’s solution
- Attracts protein molecules from wound
- Perfect alternative to “wet-to-dry”
- Appropriate for ***ALL*** wounds!



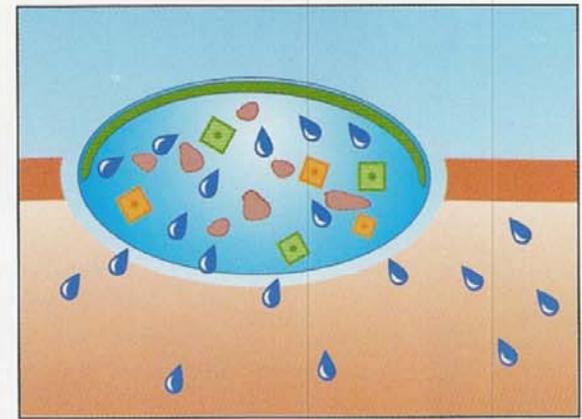
The Magic of Polyacrylate Therapy



Prior to application into the wound, the TenderWet pad is activated with Ringer's solution.



Due to the polyacrylate's higher affinity for proteins than for salts, the absorbent core simultaneously takes up and binds wound debris, necrotic tissue and microorganisms in exchange for Ringer's solution.



The 24-hour rinsing action rapidly establishes a clean wound bed, allowing for active wound healing to take place. There is tissue growth, angiogenesis and cellular migration.

			
Ringer's Solution	Wound Debris	Micro-organisms	Necrotic Tissue

Debrides

at an average rate of 38.11% per week!



Day 1

89 year-old female with severe arterial insufficiency and deep vein thrombosis. Necrotic wound on dorsal surface of left foot.



Day 16

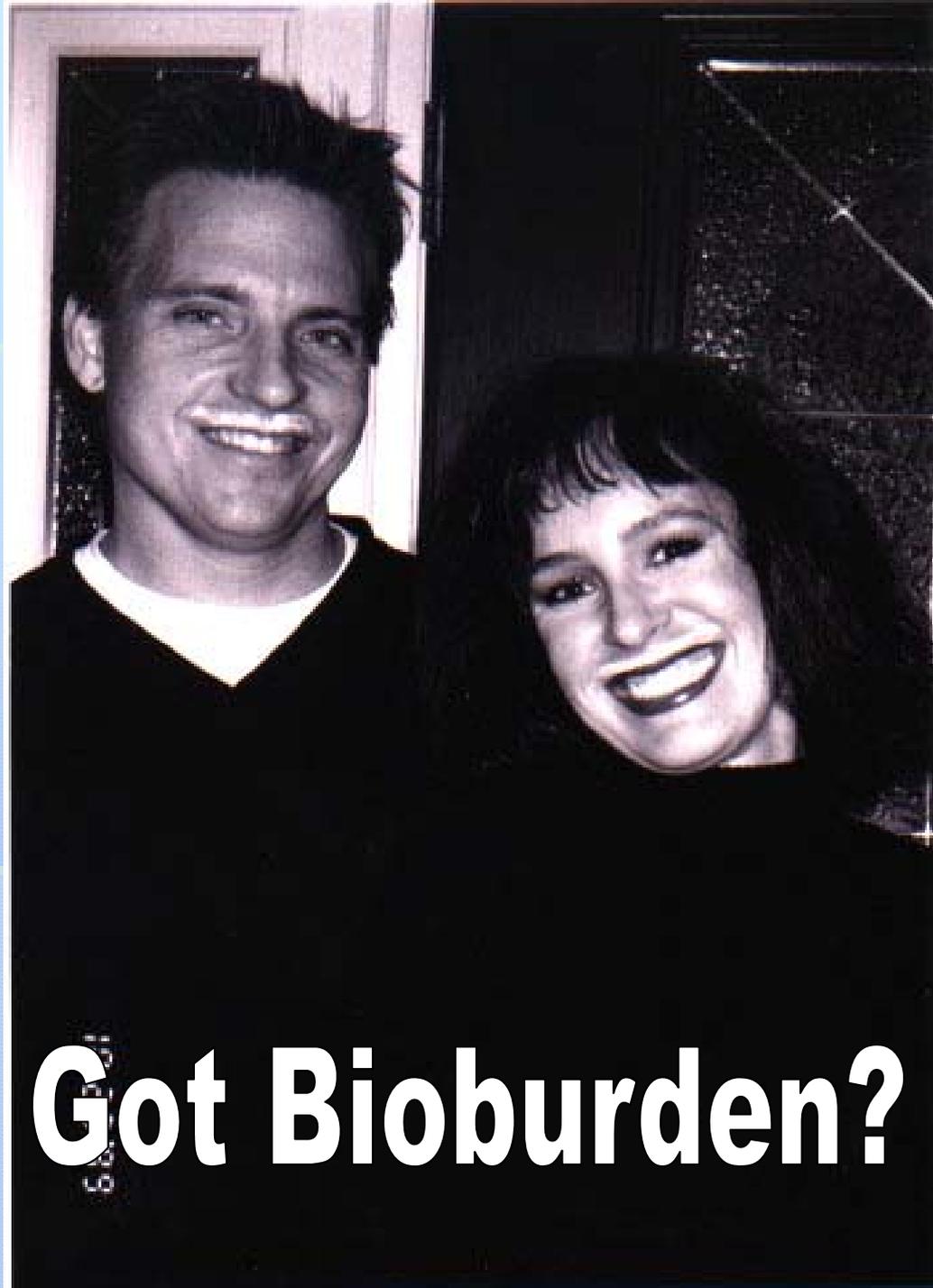
Wound base has bright red base, except for deposit of fibrin on distal-Medial wound edge. Notice the healing of wound extending to 4th toe.

Biofilm Management



- New research shows that polyacrylate gel absorbents **debride just as well as collagenase¹**.
- Recent literature has revealed that the product may be effective in **reducing wound bioburden by interfering with biofilm²**.

1. Konig, et al. Enzymatic versus autolytic debridement of chronic leg ulcers; a prospective randomized trial. *J of Wound Care*; 14(7), July 2005.
2. Bruggisser R. Bacterial and fungal absorption properties of a hydrogel dressing with a super absorbent polymer core. *J Wound Care*; 14(9), October 2005.



Got Bioburden?

Colonization

- **Acute wounds**
 - 95% are colonized with aerobes and 49% with anaerobes
- **Chronic wounds**
 - 98% are colonized with aerobes and 64% with anaerobes



Host Reaction



- When the body's resistance subsides, the wound succumbs to infection more easily
- Signs and symptoms like cellulitis and increased exudate levels may present once the bacteria overpower the body's ability to heal normally

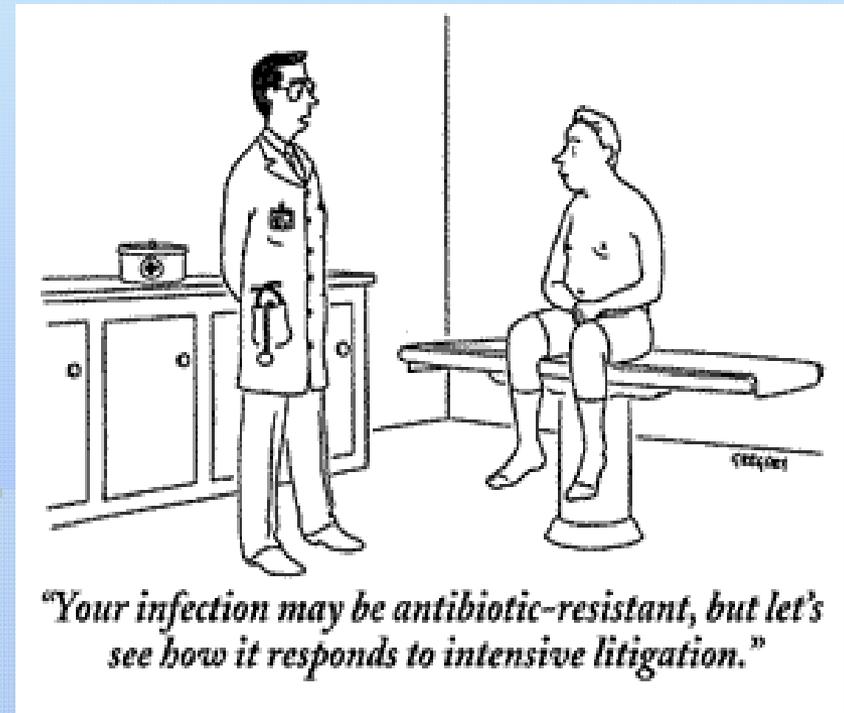
Critical Colonization

- Foul or excessive odor
- Absent or abnormal granulation tissue
- Change in wound bed color
- Delayed healing
- Friable granulation tissue
- Severe or increased pain
- Excessive or increased exudate
- Tunneling or pocketing



Systemic Antibiotics

- Should not be exclusively relied upon to control wound infection
- May not reach adequate levels in chronic granulation tissue to have an effect on bacterial levels
- **Topical antimicrobial therapy should continue** since there is often no vascular supply for delivery of systemic antibiotics to ischemic area.



Bacterial Resistance

- No recorded resistance to silver

Percival SL, Bowler PG, Russell D. Bacterial resistance to silver in wound care. *Journal of Hospital Infections* (2005) 60, 1-7.



Silver is **b-r-o-a-d** spectrum

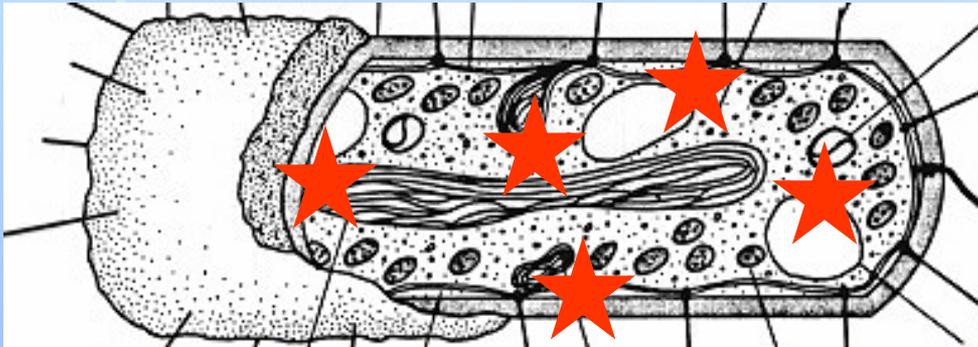
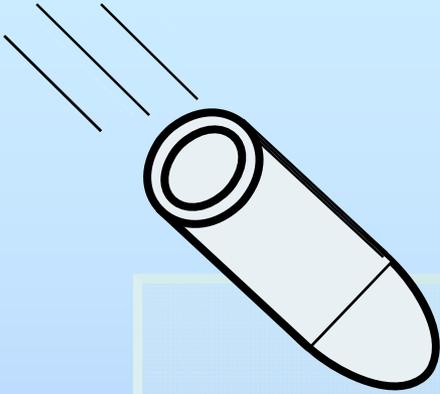
- More universally effective than antibiotics
- More broadly effective than chlorine
- Blocks the growth of:
 - Gram positive and gram negative bacteria
 - Including resistant forms of MRSA, VRE
 - Fungi
 - Viruses
 - Yeast



Broad spectrum

Strain	Bactericidal	Bactericidal
E. coli ToP10F	No growth	No growth
E. coli 8739	No growth	No growth
Kleb. pneumoniae ³³⁴⁷²	No growth	No growth
Kleb pneumoniae ³³⁴⁷⁵	No growth	No growth
Listeria monocytogenes	No growth	No growth
Staph aureus ²⁵⁹²³	No growth	No growth
Staph aureus MRSA	No growth	No growth
Staph sp Coagulase neg	No growth	No growth
Staph aureus ⁶⁵³⁸	No growth	No growth
Group A Streptococci	No growth	No growth
Proteus mirabilis	No growth	No growth
Ps. aeruginosa ²⁷⁸⁵³	No growth	No growth
Ps. aeruginosa ⁹⁰²⁷	No growth	No growth
Bacillus subtilis	No growth	No growth
Enterobacter cloacae	No growth	No growth
Enterococcus faecium	No growth	No growth
Enter. faecium VRE	No growth	No growth
Enter. faecium ENFA	No growth	No growth
Serratia marcescans	No growth	No growth
Candida albicans ¹⁰²³¹	No growth	No growth
Candida parasilosis	No growth	No growth
Candida albicans	No growth	No growth
Aspergillus niger ¹⁶⁴⁰	No growth	No growth

Modes of Action



- Cell wall
- Membrane transport
- RNA function
- DNA synthesis
- Protein function

Silver Containing Dressings

- All contain silver
 - Most important feature is how the silver is delivered
 - Continuously
 - Enough silver to kill bacteria
 - Non-cytotoxic to wound and host
 - No extra work on the part of the caregiver





**Please,
not the old silver
again!**

Out with the Old...



- **Silversulfadiazine** (Silvadene, SSD, Thermazene, Flamazine)
 - Sulfonamide that requires B.I.D. or T.I.D. application
 - Adverse reaction – hematologic neutropenia
 - Up to 5% of patients receiving
 - Contraindicated in those with sulfa allergies
 - Needs to be scrubbed out of application site
 - Can be painful
 - Only effective for about 20 minutes
 - Unsophisticated delivery system
 - Lower level of ionic silver
 - Not stable in moisture
 - Can stain skin and soft tissue permanently
 - Poor eschar penetration
 - Develops pseudo-eschar
 - Pro-inflammatory
 - Cells don't like oil

“E” – Edge

Convert Non-Advancing Edge to Advancing Edge



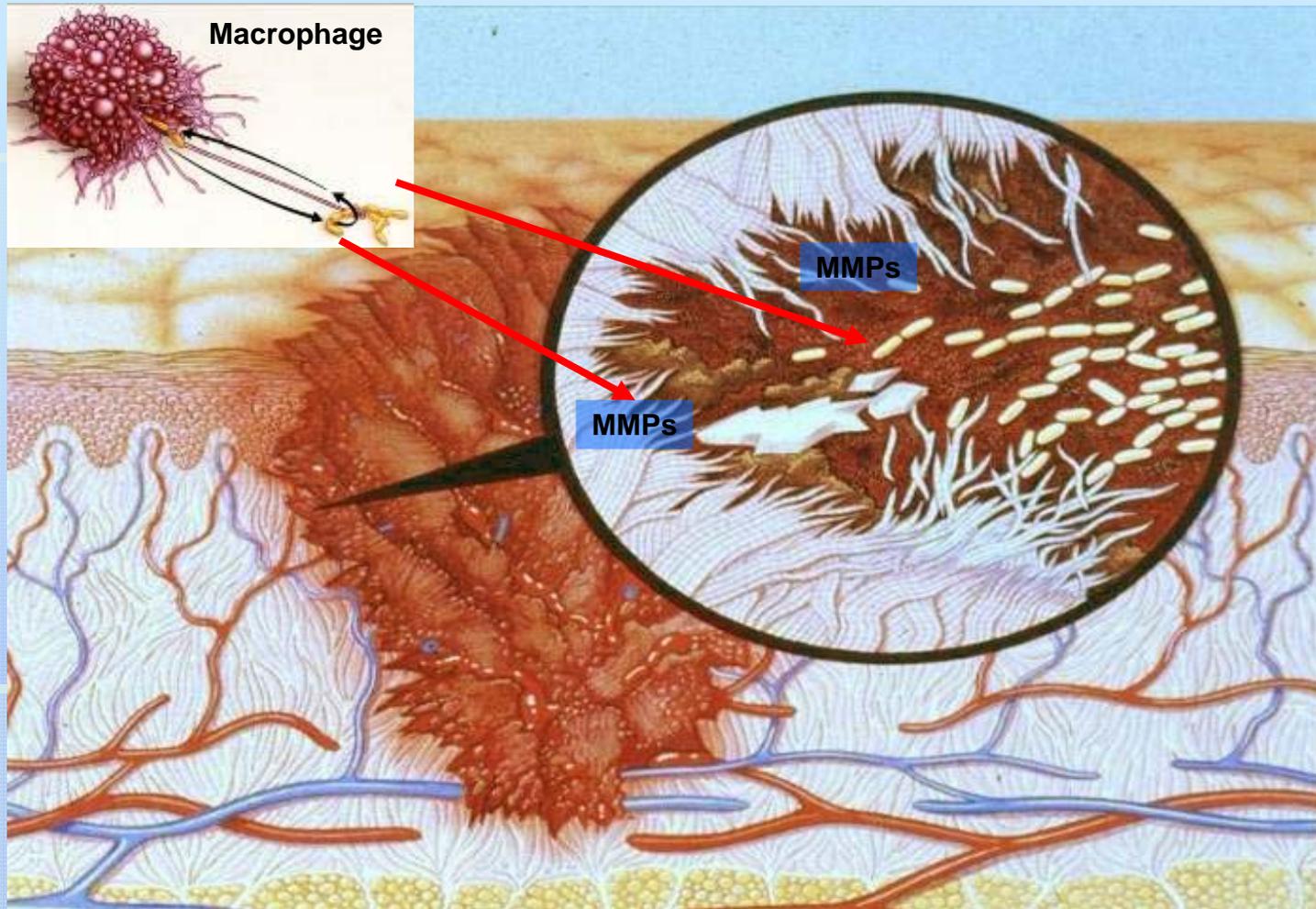
Non-advancing Edge



Advancing Edge

Failure of the epidermal edge of a chronic wound to migrate over granulation tissue is probably due to abnormal extracellular matrix components that are damaged by excess proteases and senescent cells.

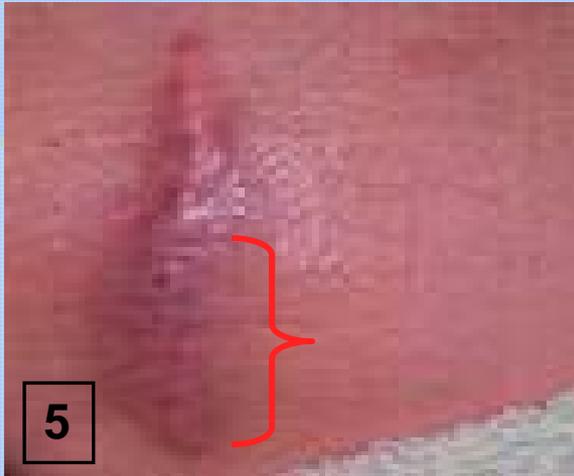
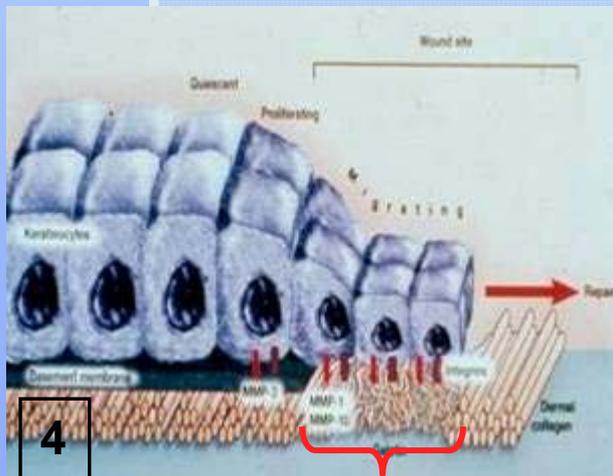
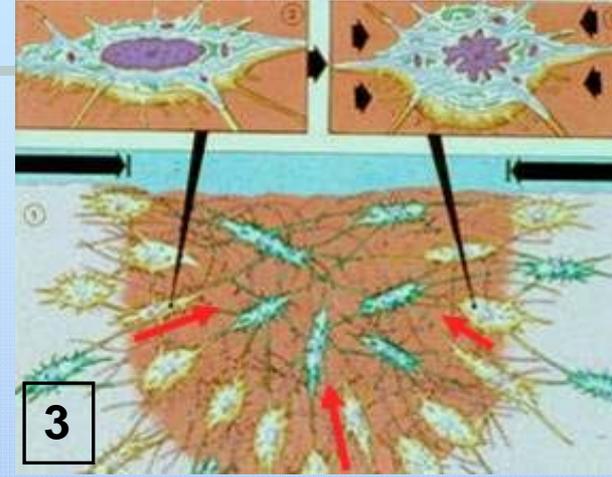
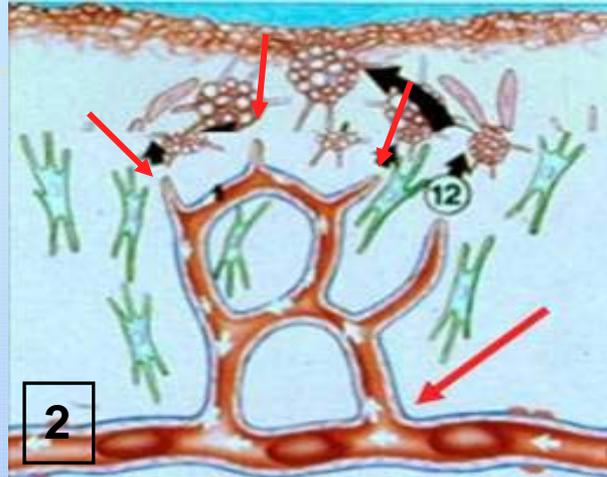
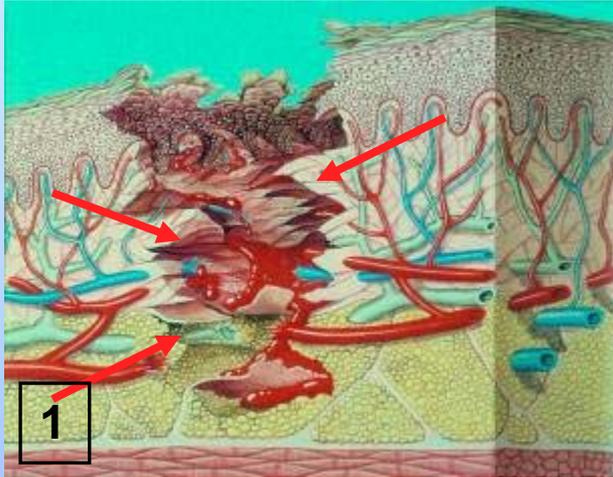
Controlled Wound Inflammation Is Beneficial



Inflammatory cells kill microorganisms and release proteases (MMPs, elastase) that remove denatured ECM components and permit wound healing to proceed. Wounds that are contaminated by bacteria and fungus must not be closed.

MMPs Are Necessary for Wound Healing

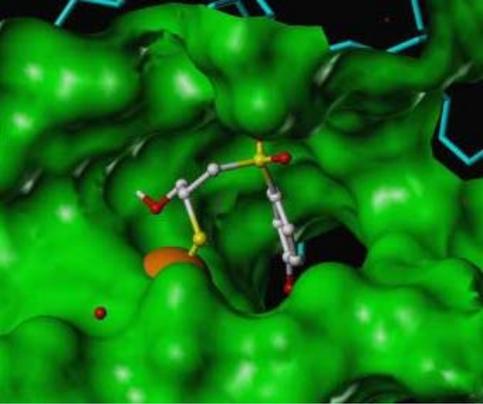
Debridement, Angiogenesis, Contraction, Epithelial Migration, Remodeling



MMPs are necessary for several key process in wound healing:

- 1) removing denatured matrix**
- 2) degrading capillary basement membrane for angiogenesis**
- 3) contraction of ECM by myofibroblasts**
- 4) migration of epidermal cells**
- 5) remodeling of scar**



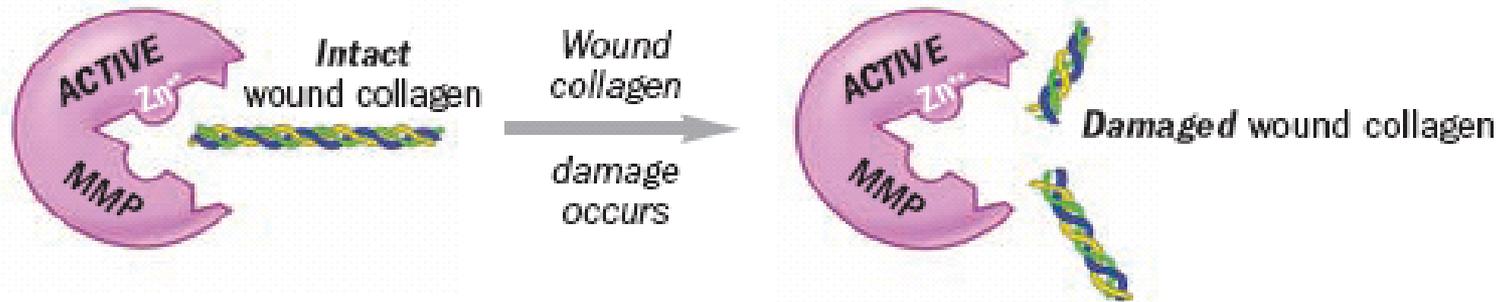


A “Good Guy” gone BAD...

- **Matrix metalloproteases (MMPs)**
 - **Structurally related protein-degrading enzymes** (proteases) that alter the ECM during wound healing process
 - **MMPs control** platelet aggregation, macrophage and neutrophil function, cell migration and proliferation, neoangiogenesis and collagen secretion and deposition.
 - They “**turn on**” or “**turn off**” matrix proteins, cytokines, growth factors and adhesion molecules.
 - **Crucial for the inflammatory phase** of wound healing are the same molecules that, when produced in surplus, hinder healing in chronic wounds.
 - **Chronic wounds** generally have **high protease levels** (MMPs) and **low protease inhibitor levels** (TIMPs) compared with acute wounds.

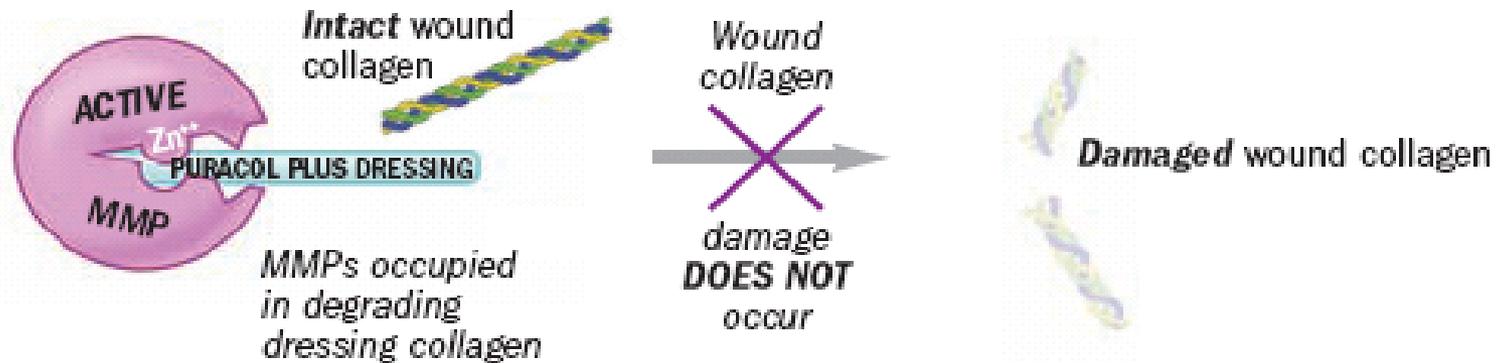
MMPs destroy collagenic structures

NO DRESSING

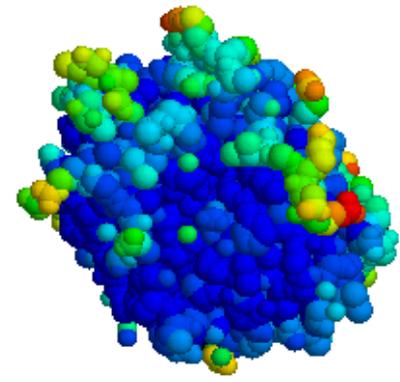


Effect of dressing on MMP activity

WITH DRESSING



Another scary enzyme that you may not have heard about...



- **Elastase** is another enzyme involved in the healing process.
 - Primary substrate is the ECM protein, elastin, which contributes to the elasticity of dermal tissue.
 - **Elastase** activity is known to be **high** in the chronic wound.
 - **Elastase** acts by converting pro-MMPs (the natural precursor of MMPs) to active MMPs.
 - Contributes heavily to the MMP load in the chronic wound.
 - **Elastase** is shown to play a key role in perpetuating the vicious chronic wound cycle.
 - **Elastase** will tend to bind to *native* collagen and degrade it.
 - Many current collagen dressings are comprised of denatured collagen.

1. Zhu Y, Liu D, Skold M, Umino T, Wang H, Spurzem J, Kohyama R, Ertl R, Rennard S. Synergistic neutrophil elastase-cytokine interaction degrades collagen in three-dimensional culture. *Am J Physiol Lung Cell Mol Physiol.* 2001; 281: L868-878.

2. Konig M, Peschen M, Vanscheidt W. Molecular biology of chronic wounds. Hafner J., Ramelet A, Schmeller W, Brunner U (eds): *Management of leg ulcers*, Curr Probl Dermatol. Basel, Karger, 1999, 29, 8-12.

3. Kafienah W, Buttle D, Burnett D, Hollander A. Cleavage of native type I collagen by human neutrophil elastase. *Biochem J.* 1998; (330) 897-902.

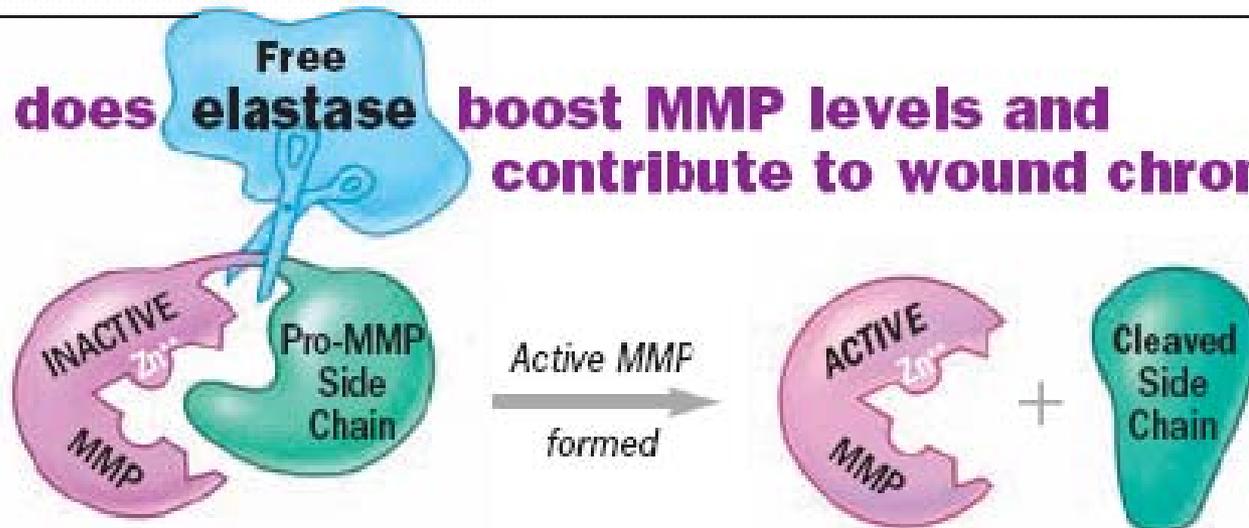
Why is Elastase a problem?



- Elastase activates MMPs
 - MMPs are problematic in the chronic wound.
 - They destroy collagen
- Elastase destroys elastin, a key component of the ECM
- Elastase destroys other useful proteins like tissue inhibitors of matrix metalloproteinases (TIMPs), “anti-MMPs”

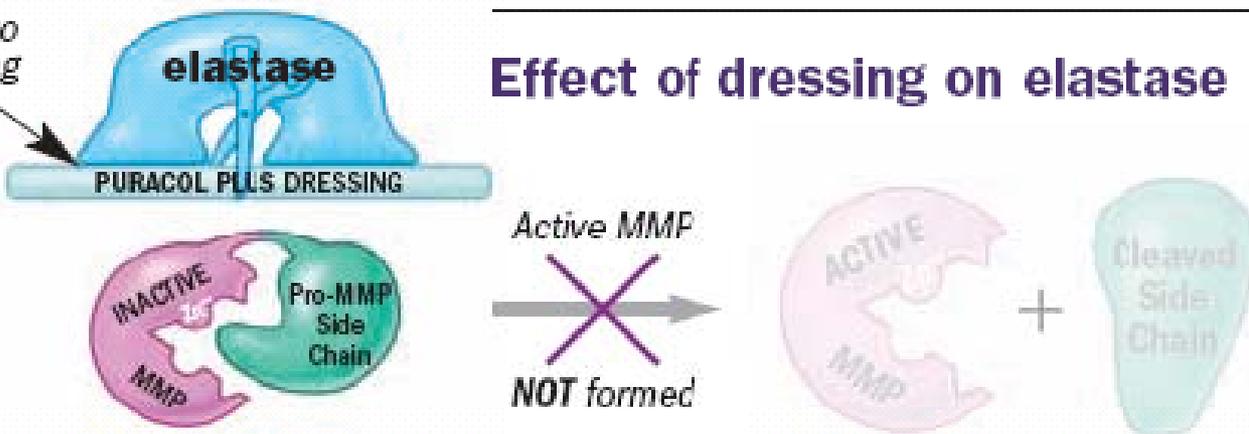
How does **Free elastase** boost MMP levels and contribute to wound chronicity?

NO DRESSING



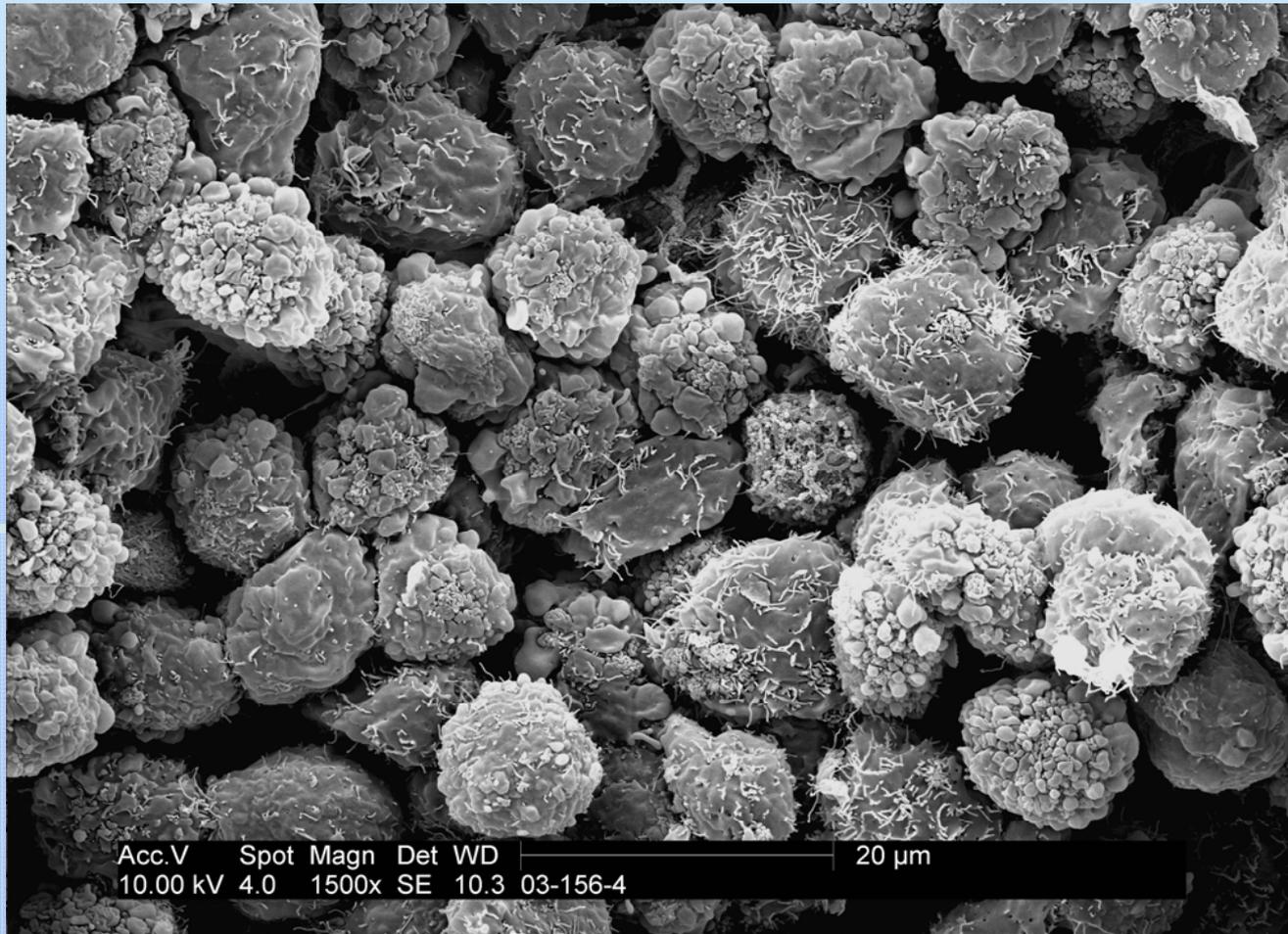
bound to dressing

WITH DRESSING

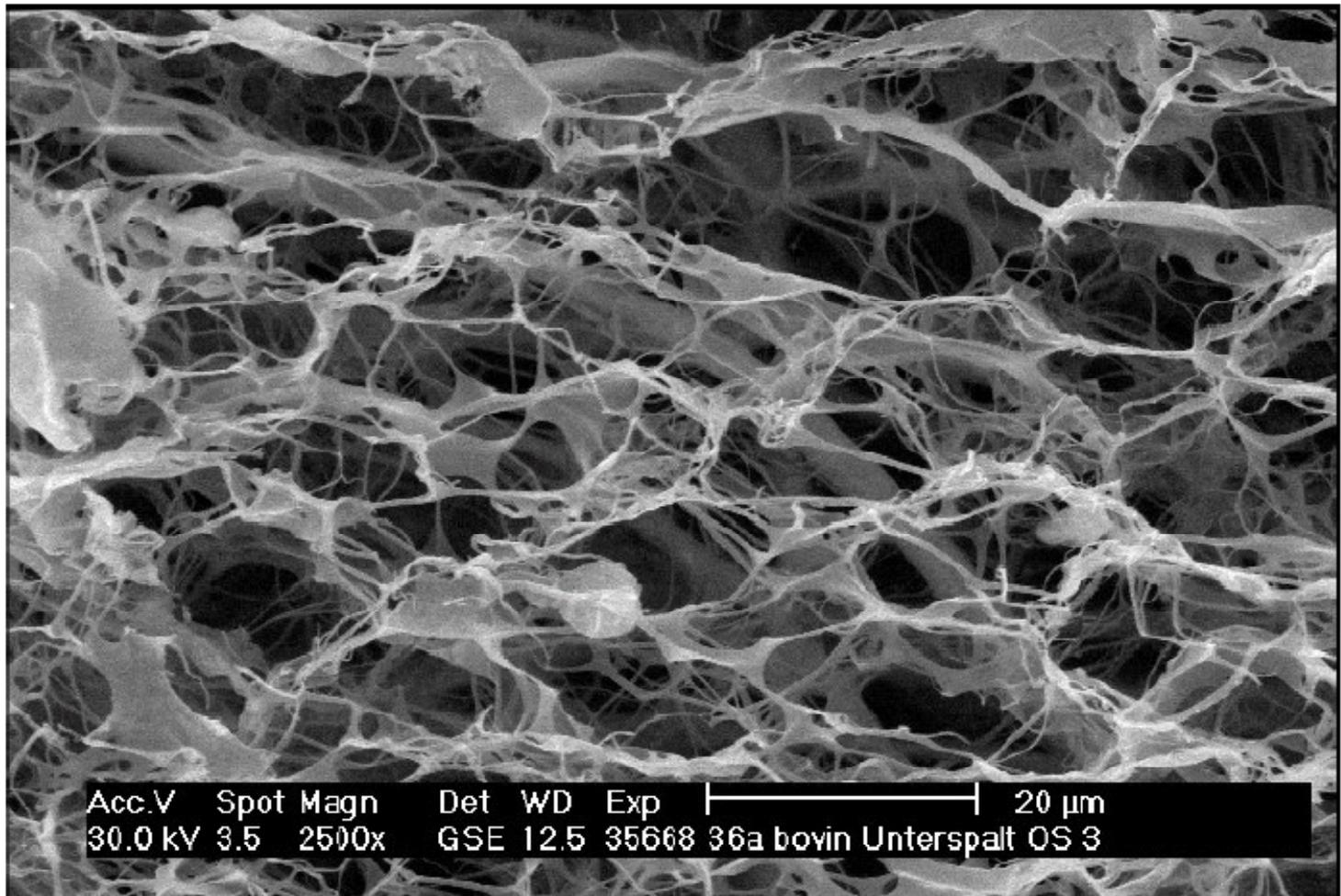




- Fibroblasts proliferating on pure collagen.
- MMP's are likely to be degrading the collagen structure in real wounds.



Second Generation Collagen Dressing Microscaffold





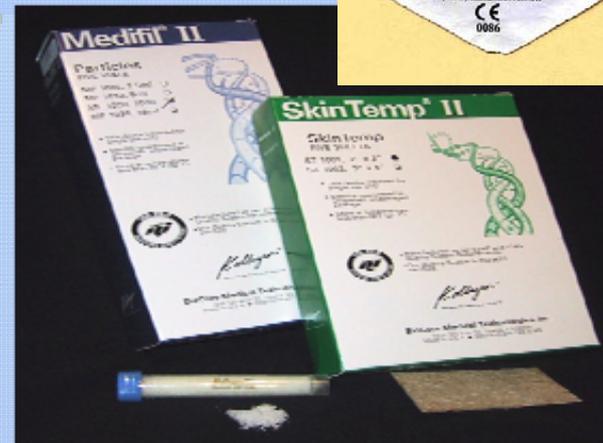
How do dressing remain “native” and not denatured?



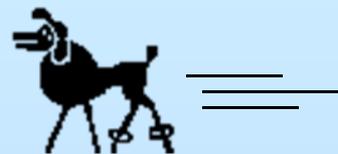
- Use mild conditions for extraction of cellular material
- Do not exceed 20° C to extract
- No harsh chemicals used
- Only use freeze drying...no oven drying
- Denatured collagen is processed chemically
 - loses it's triple helix structure (“jungle gym”)

How do advanced collagen dressings help?

- Soak up MMPs in the wound
- Shut down Elastase by binding and trapping it like a magnet
- Provide “jungle gym” for fibroblasts via microscaffold
 - Fibroblasts like 3-D structures
- Offers a sacrificial substrate as a “food” source to the destructive enzymes



Thank you for
listening to our
Mom!



Lily and William