

# The Science of Wound Repair

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Consultant / Educator

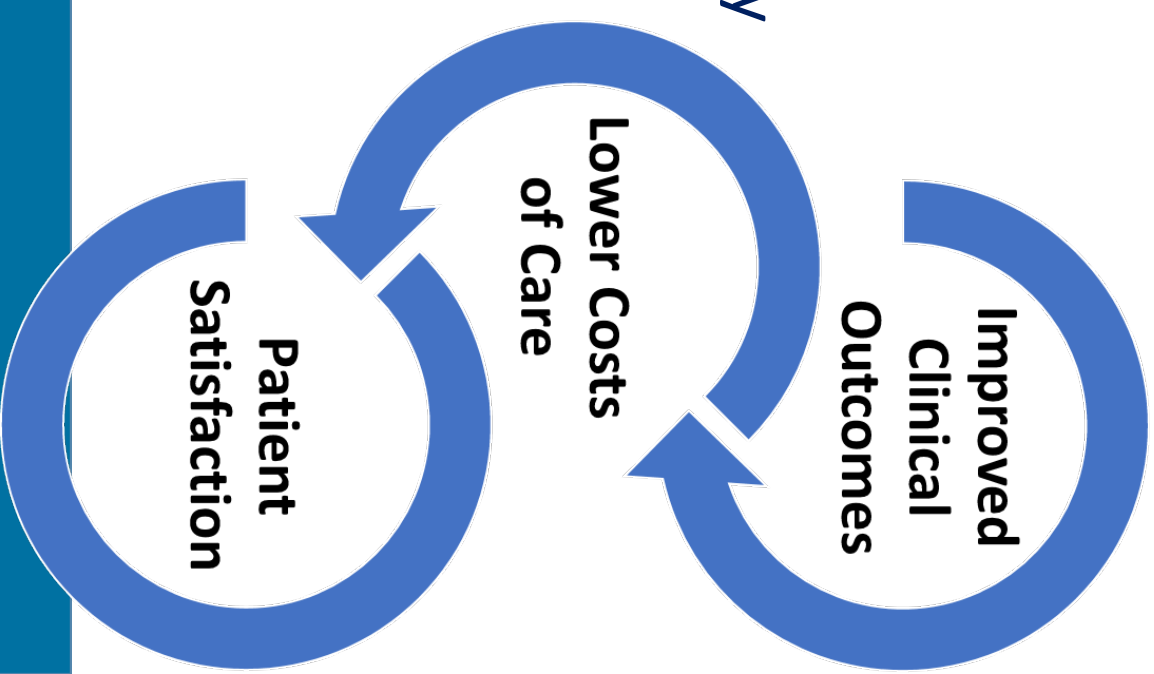
Grand Island, New York

Catholic Health Advanced Wound Healing Center

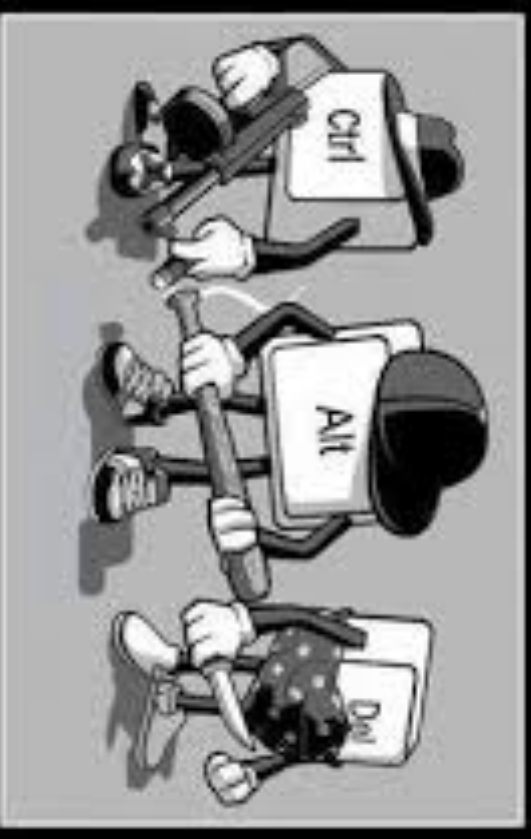
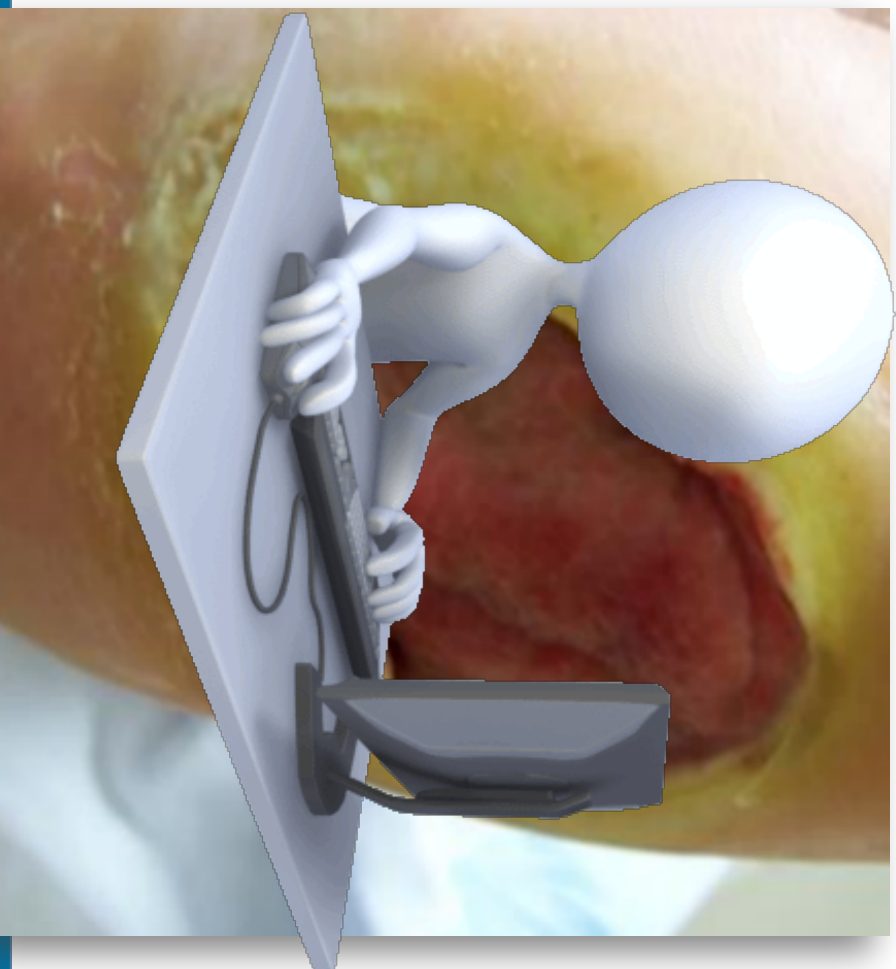
Cheektowaga, New York

# Today's Clinical Challenge

- Patients are complicated
- There's Scrutiny – financial, regulatory, quality
- Nothing is easy
- We must practice evidence based care
- Cost effectiveness is mandatory
- All care must be coordinated
- The patient is the focus...



## My History with Non-Healing Wounds



**ADMIT IT**

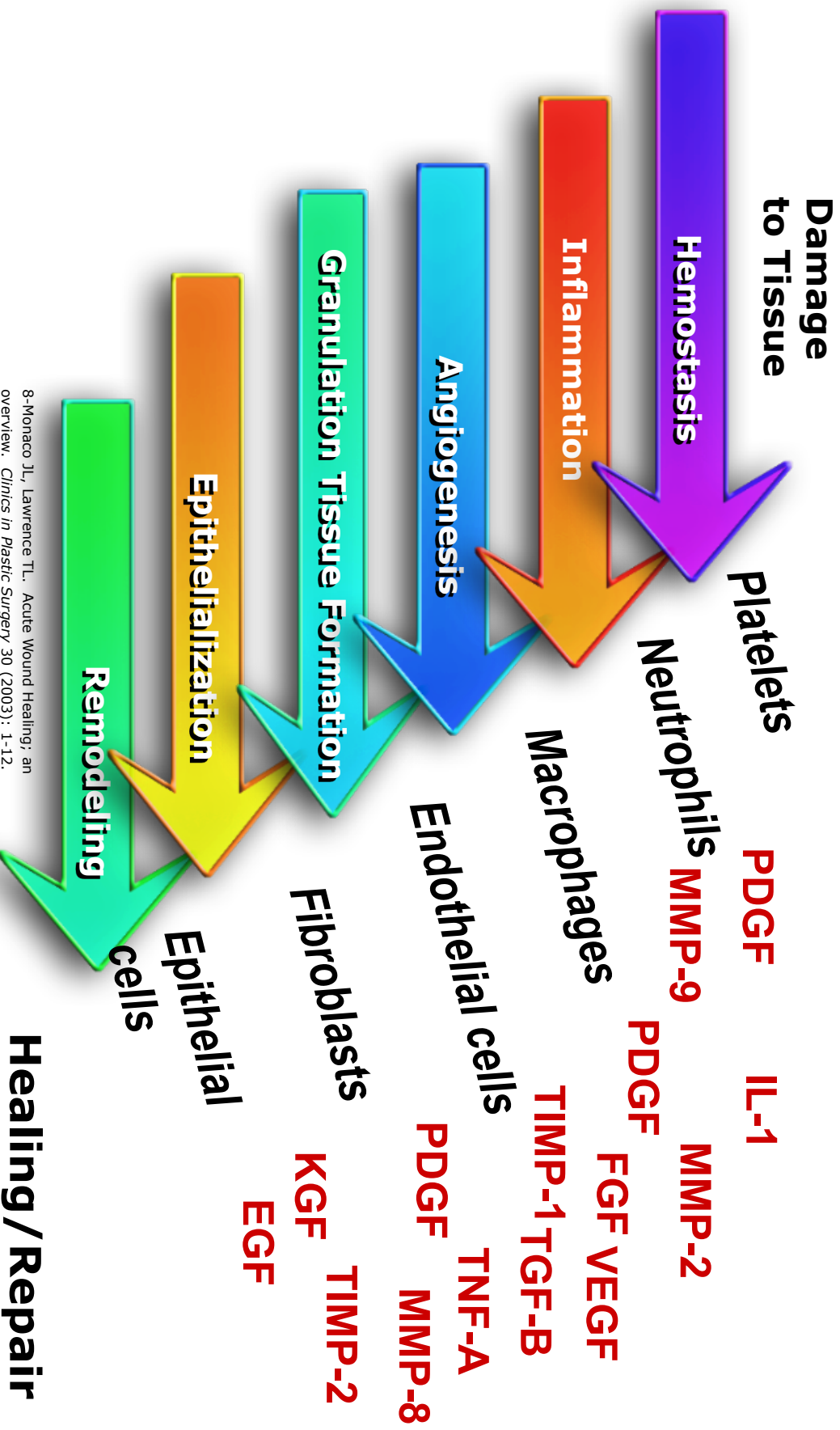
You always call them when you  
have a problem...

To Fix It.....





# Events of the Normal Healing Process



8-Monaco JL, Lawrence TL. Acute Wound Healing: an overview. *Clinics in Plastic Surgery* 30 (2003): 1-12.

# Hemostasis

- Immediately after injury
- Must occur for healing to progress
- Primary contributors
  - Vasoconstriction
  - Platelet aggregation
  - Fibrin deposition
- Clot is end product
  - Composed of aggregated platelets, fibrin mesh and embedded cells
  - Prevents further fluid & electrolyte loss
  - Prevents contamination
  - Provides provisional matrix



# Inflammation

## The clean up.....

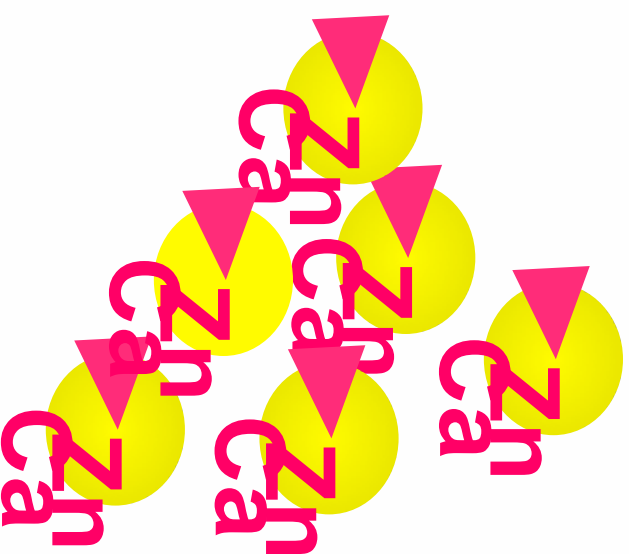


# Inflammation: Key Players

- Leukocytes: Basophils, eosinophils, neutrophils, lymphocytes, monocytes
  - Neutrophil: Small white cell, in great abundance, present after injury for initial cleanup
- Monocyte: Large circulating leukocyte; differentiates into macrophage
- Macrophage: Phagocytic cell; able to engulf large amounts; produces MMPs and growth factors; function best at acidic pH and low O<sub>2</sub> tension
- Mast Cells: Involved in the presence of subacute and chronic inflammatory disease
  - Part of the skin immune system

# Matrix Metalloproteases

- Family of protein-degrading enzymes
- Assists with wound clean-up
- Necessary for cells to move and migrate
- In great abundance will be destructive



# Tissue Inhibitors of MMPs (TIMPs)

- Reversibly bind MMPs in 1:1 ratio to inactivate
- Secreted locally by cells
- Four TIMPs known
- Each TIMP can bind more than one type of MMP

*TIMP-1*

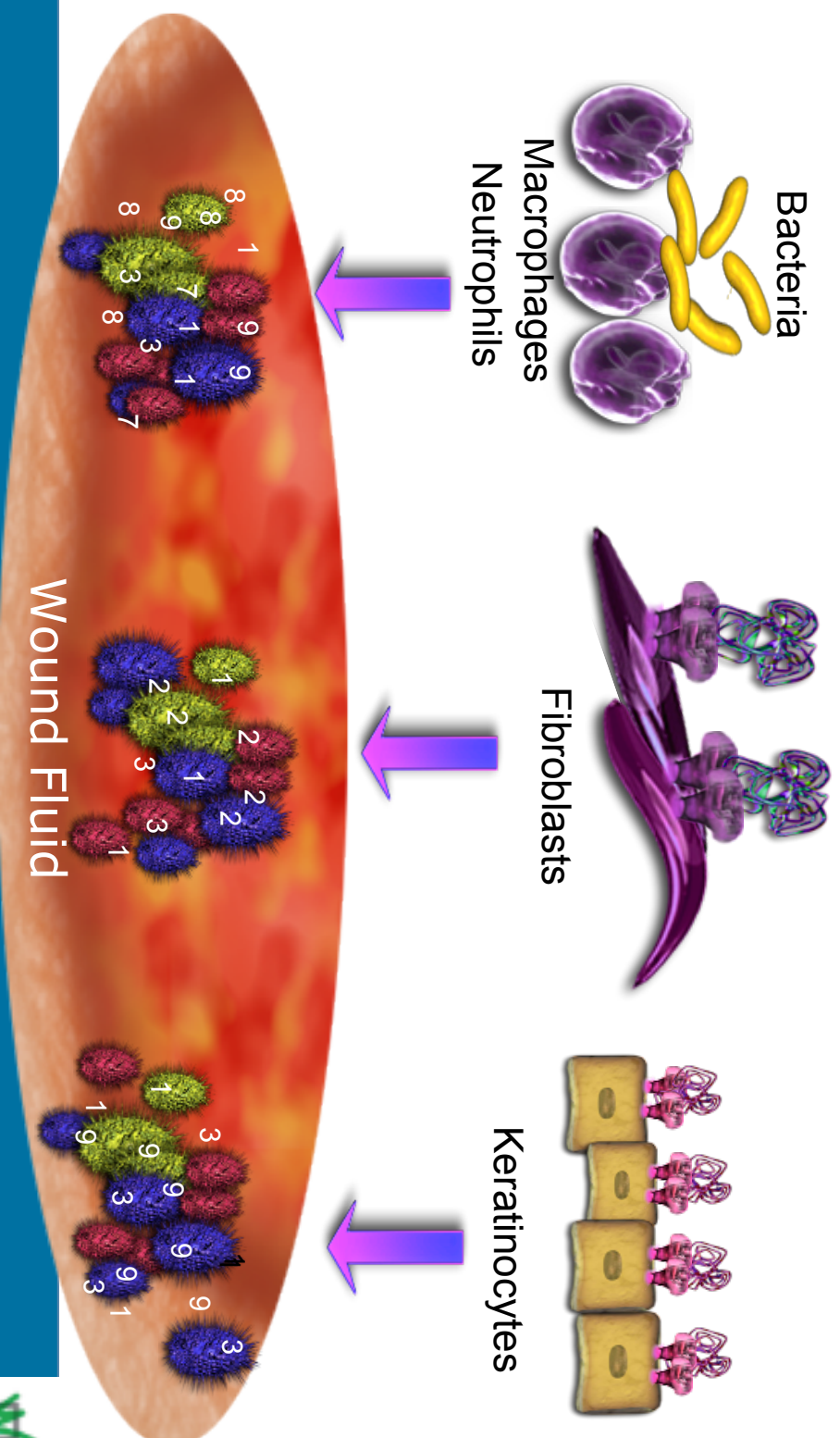
*TIMP-2*

*TIMP-3*

*TIMP-4*



# The Production of Matrix Metalloproteases (MMPs)





# Inflammation



- Characterized by erythema, edema, heat, and pain
  - White cells (neutrophils, monocytes) migrate out into wound space
- Transition from vasoconstriction to vasodilation mediated by variety of factors
  - Mast cells: histamine
- Neutrophils usually depleted after 2-3 days by the process of apoptosis
  - Replaced by tissue monocytes
- Stimulated to differentiate into macrophages by cytokines and growth factors
- Early macrophages clean up, later in inflammation, they recruit cells to begin the proliferative phase of healing

# Proliferation

The Build up.....



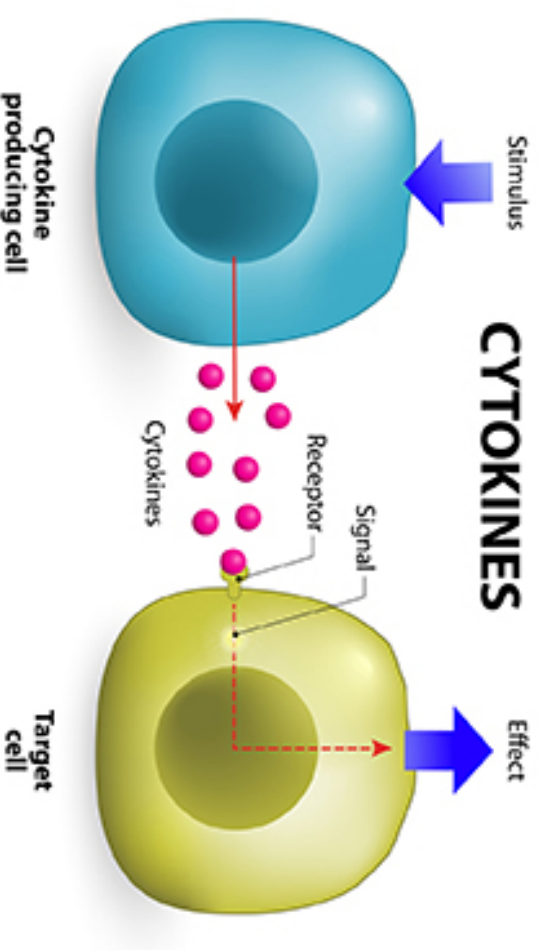
# Proliferative Phase

- Granulation Tissue Formation
  - Replace lost dermal tissue with granulation tissue
- Angiogenesis
  - New blood supply
- Epithelialization
  - Resurfacing



# Cytokines

- Cell: Activity
- Cellular or chemical mediators
  - Regulate activity
- Growth factors
- Inflammatory mediators
- Interleukins
  - Inter: “As a means of communication”
  - Leukin: Produced by and act on leukocytes
- Tumor necrosis factor
  - Involved in systemic inflammation
  - One of the cytokines making up the acute-phase reaction



<https://en.wikipedia.org/wiki/Interleukin>.

[https://en.wikipedia.org/wiki/Tumor\\_necrosis\\_factor\\_alpha](https://en.wikipedia.org/wiki/Tumor_necrosis_factor_alpha).

# What Are Growth Factors?

Complex proteins, released by cells, that stimulate:

- Cell migration (chemotaxis)
- Cell proliferation (mitosis)
- Angiogenesis
- Production and degradation of extracellular matrix
- Growth factor production by other cells
- Apoptosis (programmed cell death)

# Growth Factor Nomenclature

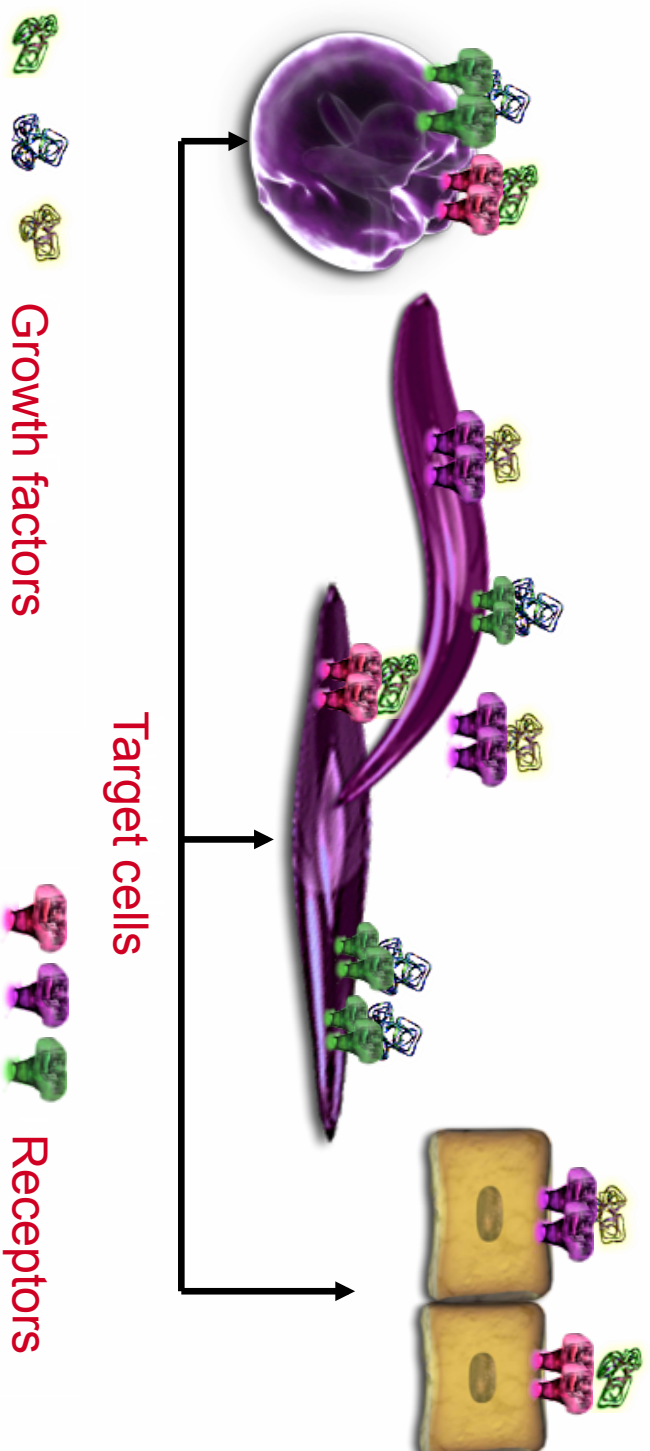
- May be named for the cell that they come from, the cell they act on, or activity first observed
  - Platelet Derived Growth Factor (PDGF)
  - Epidermal Growth Factor (EGF)
  - Keratinocyte Growth Factor (KGF)
  - Fibroblast Growth Factor (FGF)
  - Transforming Growth Factor (TGF)





# How Do Growth Factors Work?

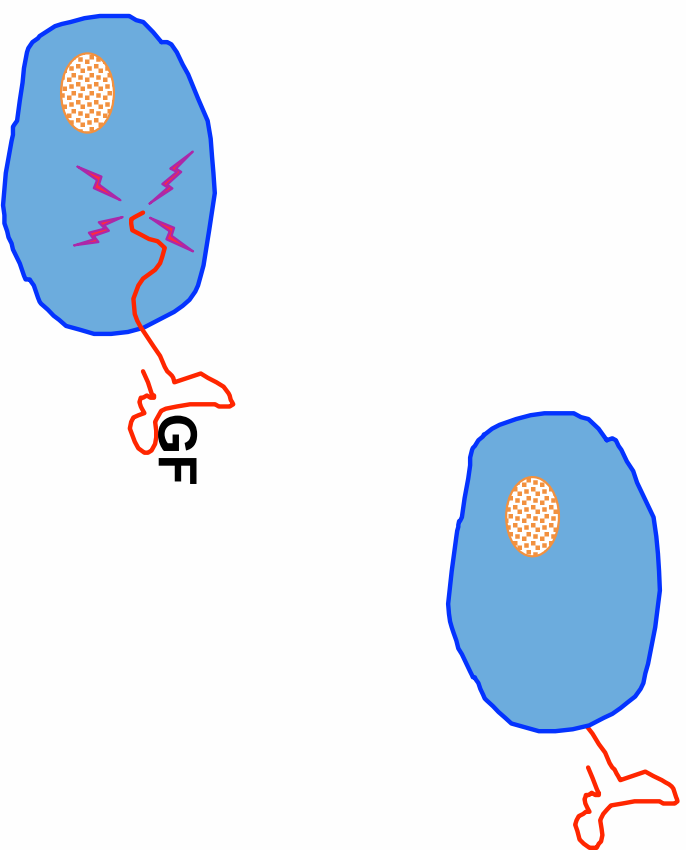
- Different cells secrete different growth factors and can express several different receptors



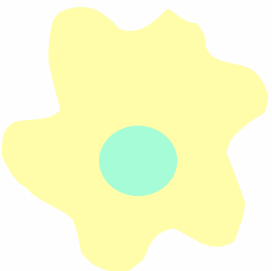


# Growth Factor Receptors

- Cell surface proteins
- Sit on the plasma membrane of the cell
- External binding of GF activates internal activity of protein
- Activates enzyme or gene

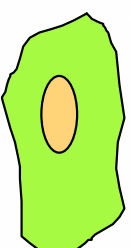


**Growth Factors are like words....**

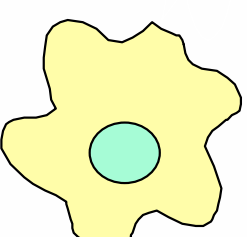


**PDGF**

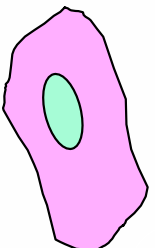
**VEGF**



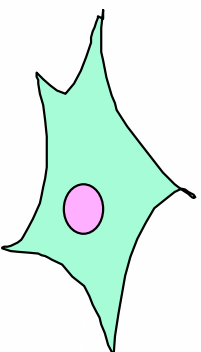
**FGF**



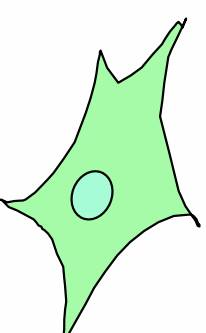
**EGF**



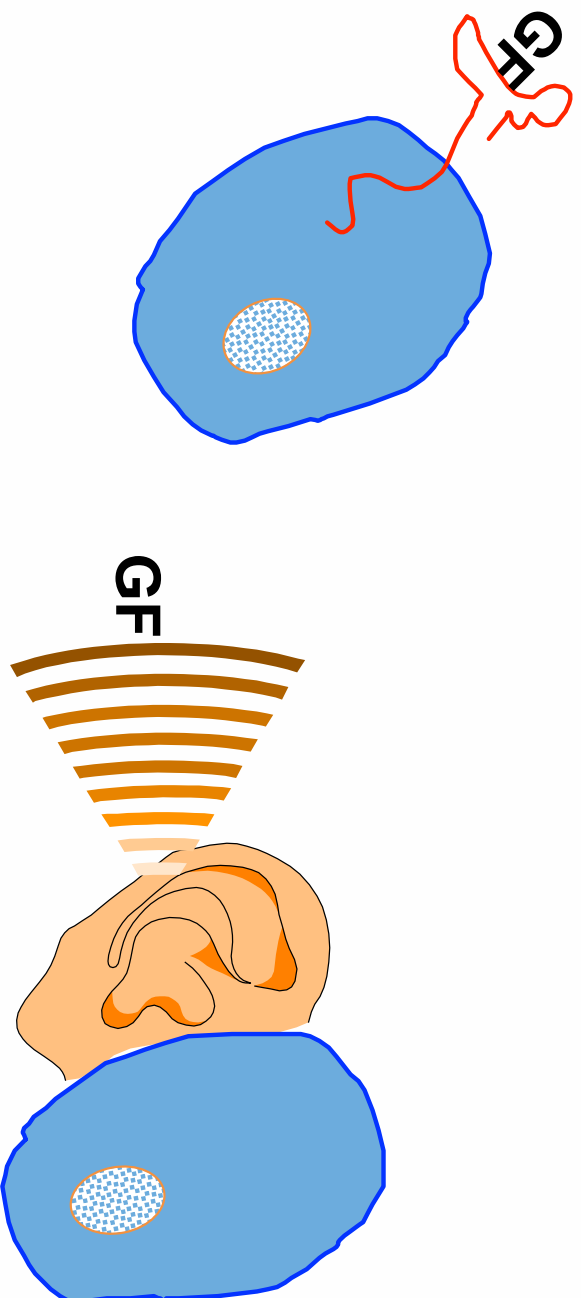
**TNF $\alpha$**



**KGF**



**Receptors are like ears.....**



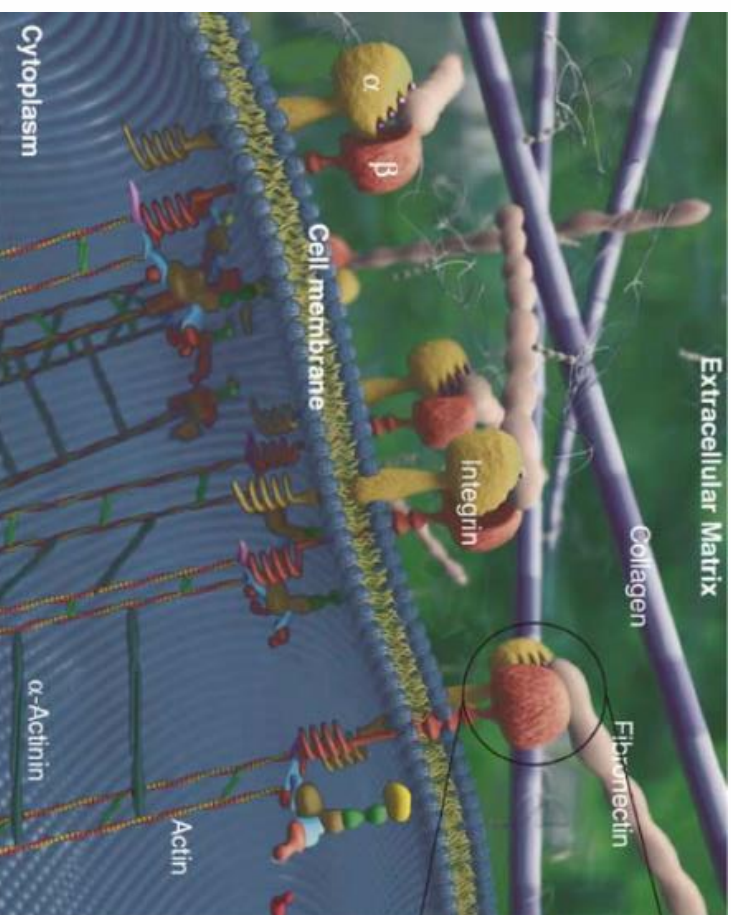
Factor	Cell or tissue of origin	Target cells or tissue	Effect
Platelet Derived GF (PDGF)	Platelets, macrophages, neutrophils, smooth muscle cells	Fibroblasts, smooth muscle cells, endothelial cells, macrophages, neutrophils	collagen synthesis, endothelial cells, keratinocyte migration, promotes ECM
Vascular endothelial GF (VEGF)	Endothelial cells, keratinocyte, fibroblasts, smooth muscle cells, platelets, neutrophils, macrophages	Endothelial cells	Early events in angiogenesis, endothelial cell migration
Transforming Growth factor (TGF- $\beta$ )	Platelets, bone, most cell types	Fibroblasts. Endothelial cells, keratinocytes, lymphocytes, monocytes	ECM, fibroblasts activity, chemotaxis, inhibition of keratinocytes (scarring)
Fibroblast Growth Factor (FGF)	Monocytes, macrophages, endothelial cells	Endothelium, fibroblasts, keratinocytes	Proliferation of endothelial cells, keratinocytes and fibroblasts, promotes ECM
Epithelial Growth Factor (EGF)	Monocytes, macrophages	Epithelium, endothelial cells	Proliferation of keratinocytes, fibroblasts and endothelial cells, keratinocyte migration

<http://www.bu.edu/woundbiotech/growthfactors/gfdevtest.html>. (accessed 11/8/15).  
 Barrantes S et al. Growth factors and cytokines in wound healing. *Wound Rep Reg* 2008;16:585-601.



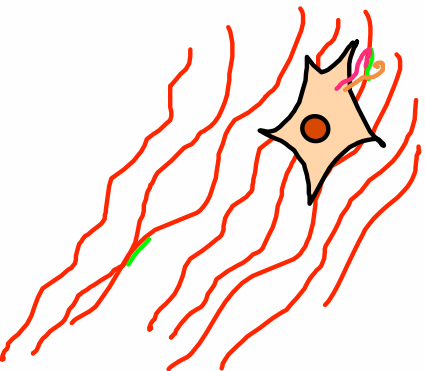
# Integrins

- Family of cell surface receptors
- Enable cells to detect and interact with components of the extracellular matrix
- Transmembrane receptors
  - Alpha subunit
  - Beta subunit
- Reversible binding to portions of ECM

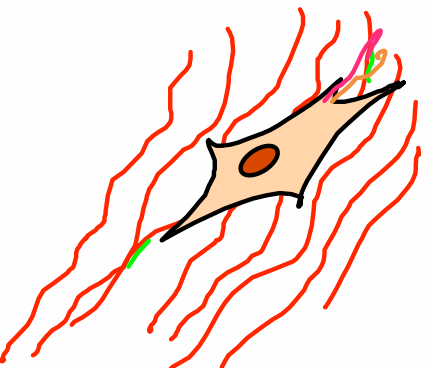


# Proliferation: Fibroblasts

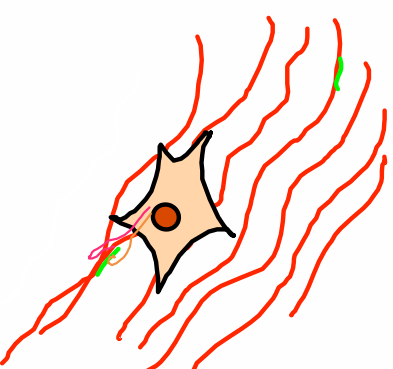
## *Fibroblast Migration into Wound Site*



Integrin on one  
end of cell attaches to  
binding site



Cell stretches  
toward next binding  
site on matrix



Cell binds new  
site and releases first  
site, moving forward

## **Integrins: When are they used?**

- Platelet interactions with collagen during hemostasis
- Leukocyte extravasation during early inflammation
- Endothelial cell budding and ingrowth during angiogenesis
- Epithelial cell migration
- Fibroblast movement through granulation tissue

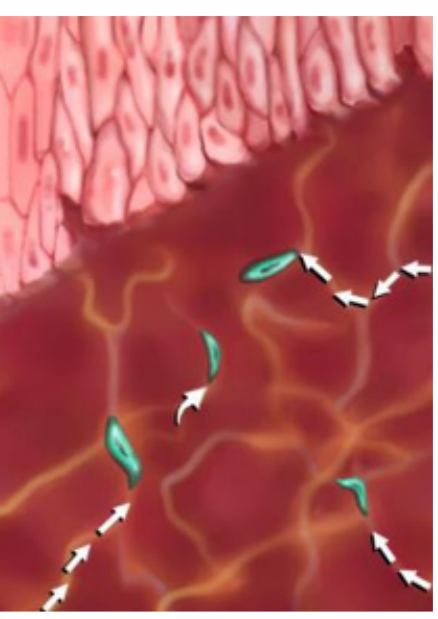


# Key Cells in Wound Healing

- **Erythrocyte:** Major cellular element of circulating blood, contains hemoglobin, major function is to transport oxygen
- **Fibroblast:** Key cell; synthesizes collagen and other ECM substances, depending on need of surrounding tissue
- **Myofibroblast:** Modified fibroblast found at periphery of wound responsible for contraction
- **Endothelial cells:** Line vessels, particularly capillaries; migrate and form new vessels in wound healing
- **Keratinocytes:** Epidermal cell that synthesizes keratin

# Cellular Migration and Proliferation

- Initial matrix heavily populated with inflammatory cells
- Fibroblasts in normal dermal tissue are quiescent and sparsely distributed
  - Active and numerous in granulation tissue
- Fibroblasts and endothelial cells predominate as healing progresses
- Native cells are lost or damaged in any injury so additional fibroblasts required in the healing wound, migrate from adjacent tissues
- Cytokines may transform undifferentiated cells in the wound into fibroblasts

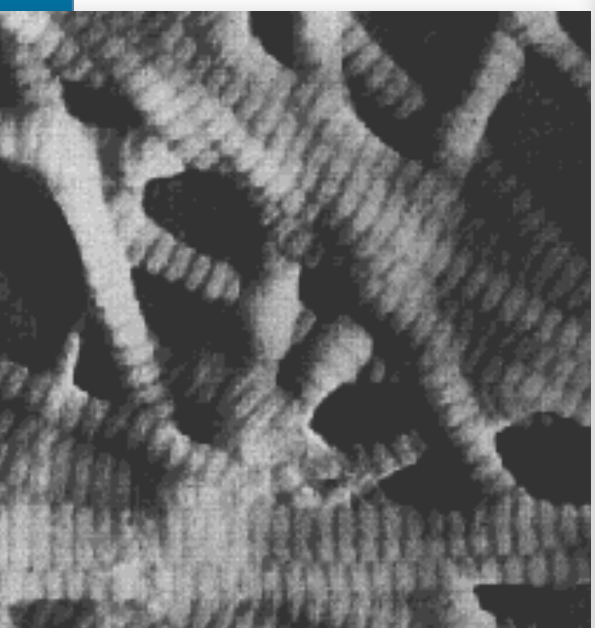
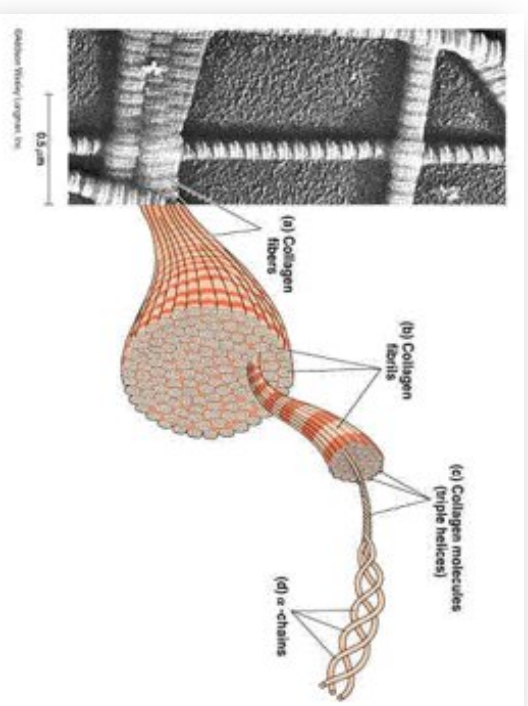


# Protein Synthesis

- Fibroblasts most responsible
- Stimulated by growth factors
- Initial matrix changes to collagen and other proteins
- Elastin synthesized in seemingly decreased quantities

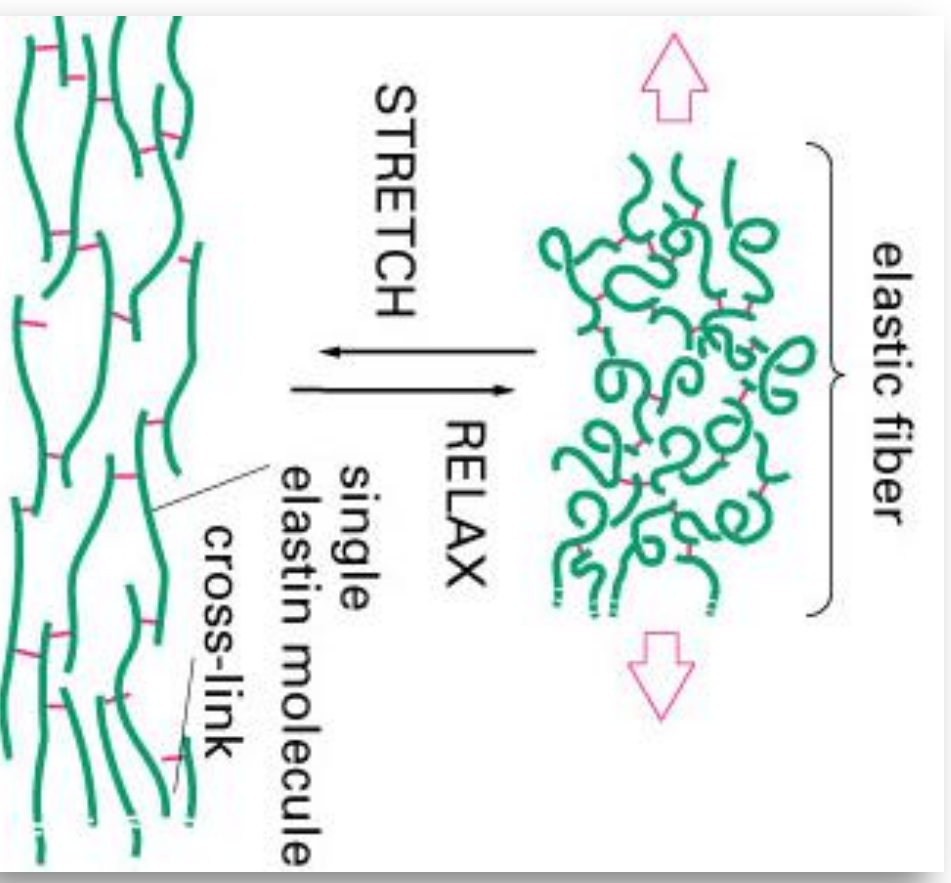
# Dermal Proteins

- Collagen
  - Major structural protein
  - Secreted by dermal fibroblasts
  - Primarily Type I (77% to 85%)
  - Lesser type III (15%)



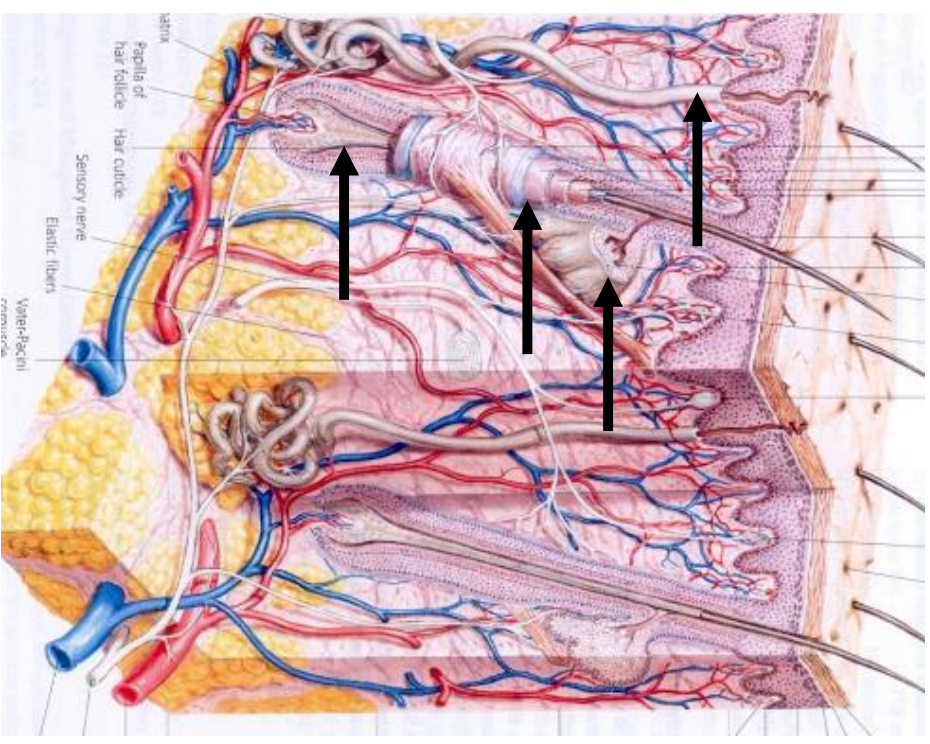
# Dermal Proteins

- Elastin
  - Provides skin with its elastic recoil
  - Secreted also by fibroblasts



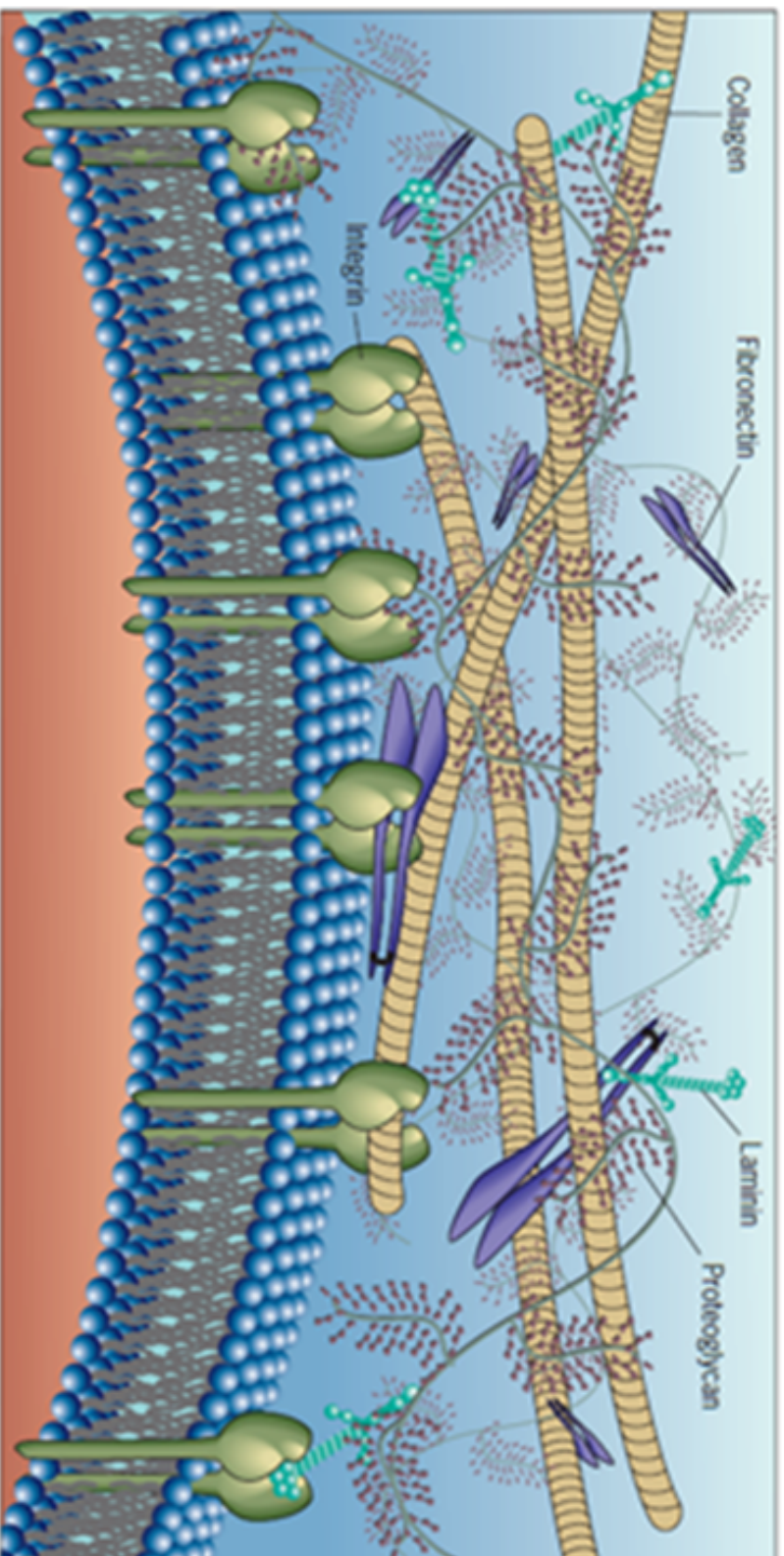
# Dermal Appendages

- Hair follicles
- Sebaceous glands
- Sweat glands
- Lined with epithelial cells
  - Epithelial islands





# Extracellular Matrix



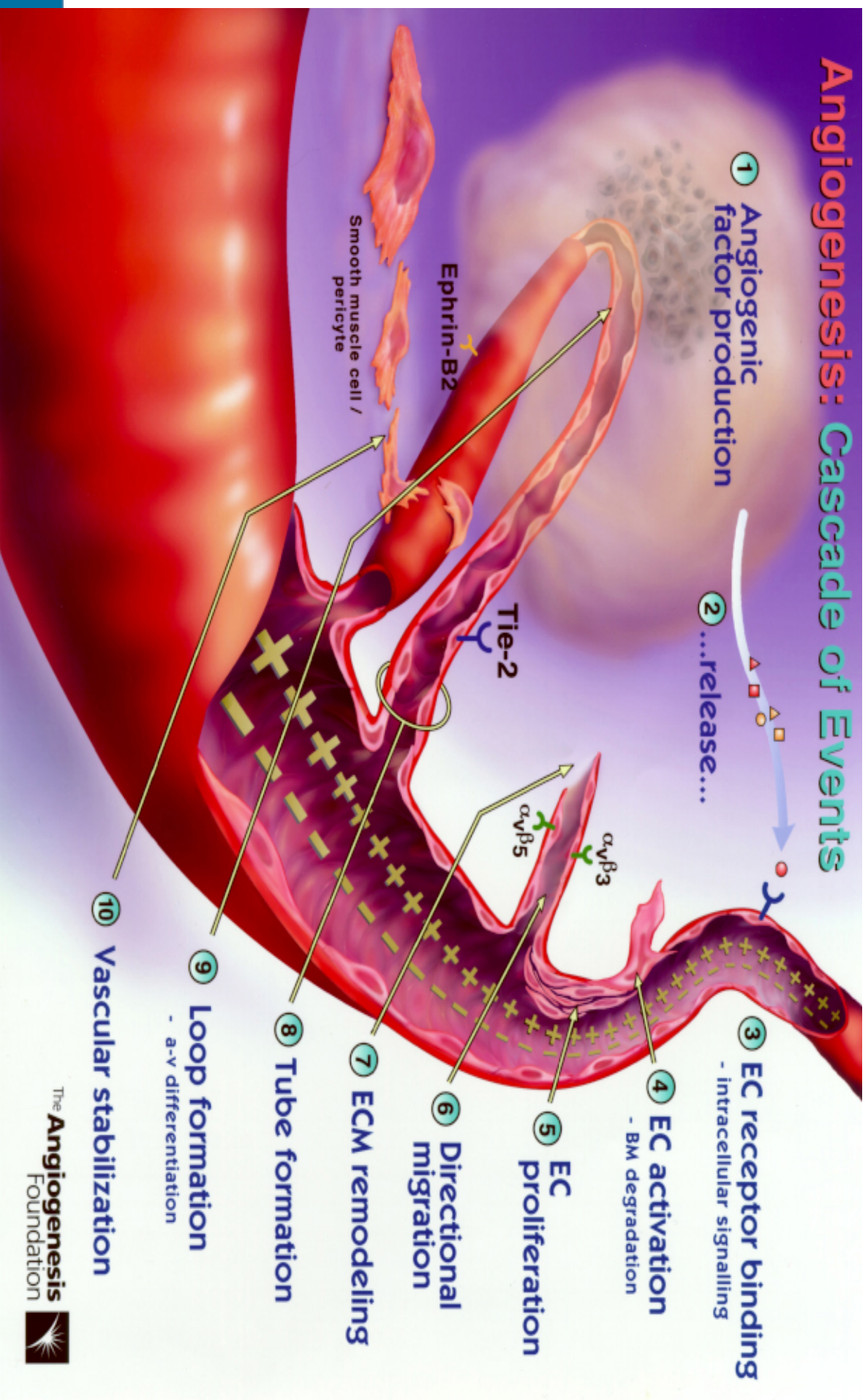


# Angiogenesis

- Process that becomes active from day 2 after wounding
- Triggered by decreased oxygen tension
  - Caused by disruption of native vasculature and increased oxygen consumption by other cells in the wound environment
  - Proliferating cells consume oxygen 3-5 times faster than resting cells
- Endothelial sprouts derive from intact capillaries at the wound periphery



# Angiogenesis: Cascade of Events



## Wound Contraction

- Characterized by a predominance of myofibroblasts at the wound periphery
  - Modified fibroblasts
  - Appear 4 to 6 days after initial injury and commonly seen in the wound during the following 2 to 3 weeks
  - Generate strong contractile forces on the wound edges
- Wound edges draw closer to each other
- Rate of contraction varies between anatomic locations, averages 0.6 to 0.7 mm per day
  - Rate can be predicted by the degree of skin laxity at the site
  - Scalp or pretibial area contracts more slowly than a buttock wound



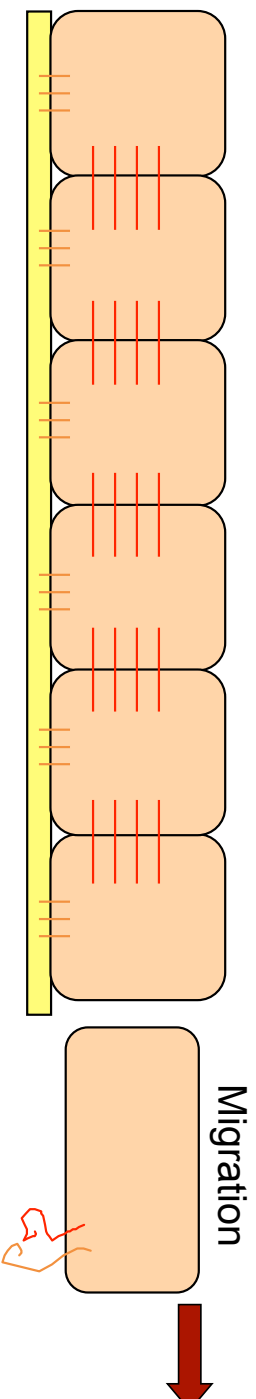


# Epithelialization

- Reconstruction of injured epithelium begins almost immediately after wounding
  - Incisional injuries are typically re-epithelialized within 24-48 hours after injury
- Basal cells present at wound edge elongate and begin to migrate across denuded wound surface
- Hair follicles and sweat glands contribute migratory epithelial cells if not destroyed during initial injury
- Cells migrate as a monolayer, begin to proliferate approximately 24 hours after initiation

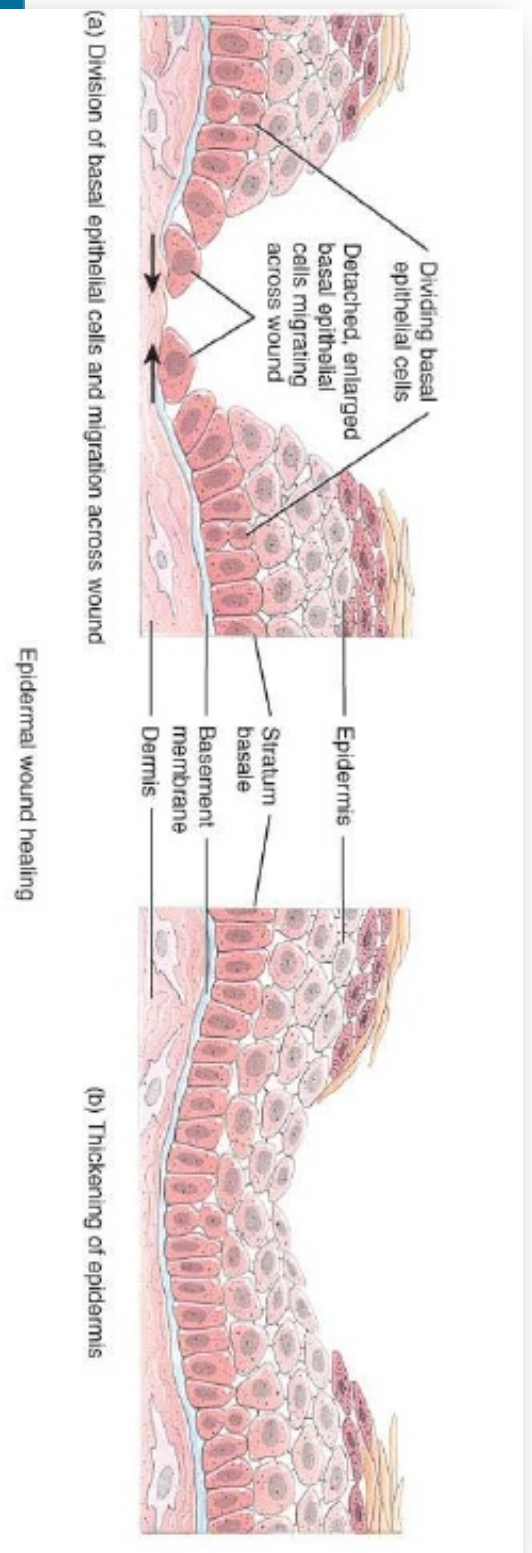
# Epithelialization

- Cells at leading edge flatten, express integrins, secrete proteolytic enzymes, move
- Desmosomes attach cells to each other, hemidesmosomes attach to basement membrane



# Epithelialization

- Basal epithelial cell detachment from basement membrane; migrate until they meet and form confluent layer
- Cellular migration
- Monolayer proliferates, stratified layers re-establish and barrier function is restored





# Remodeling

## Reorganizing the interior.....

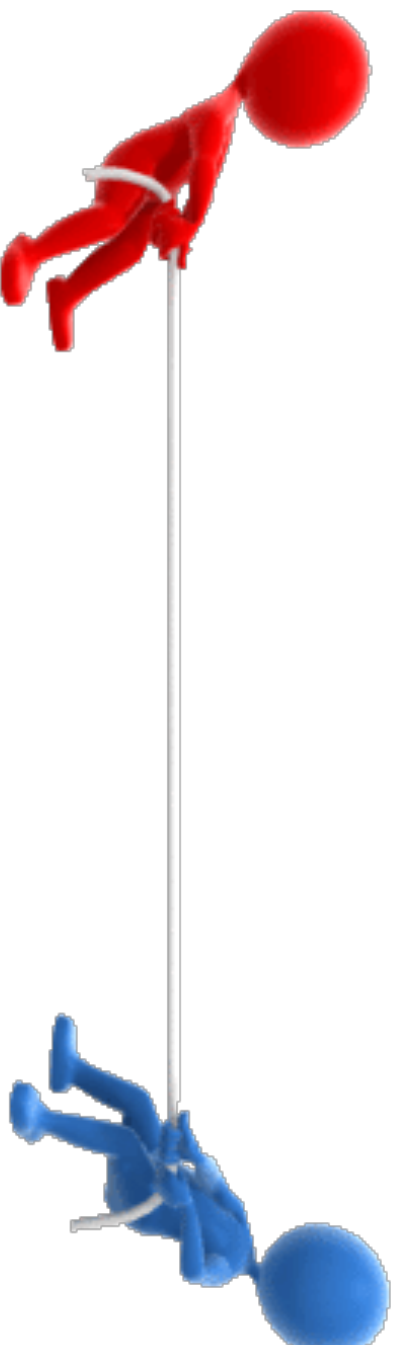


# Remodeling

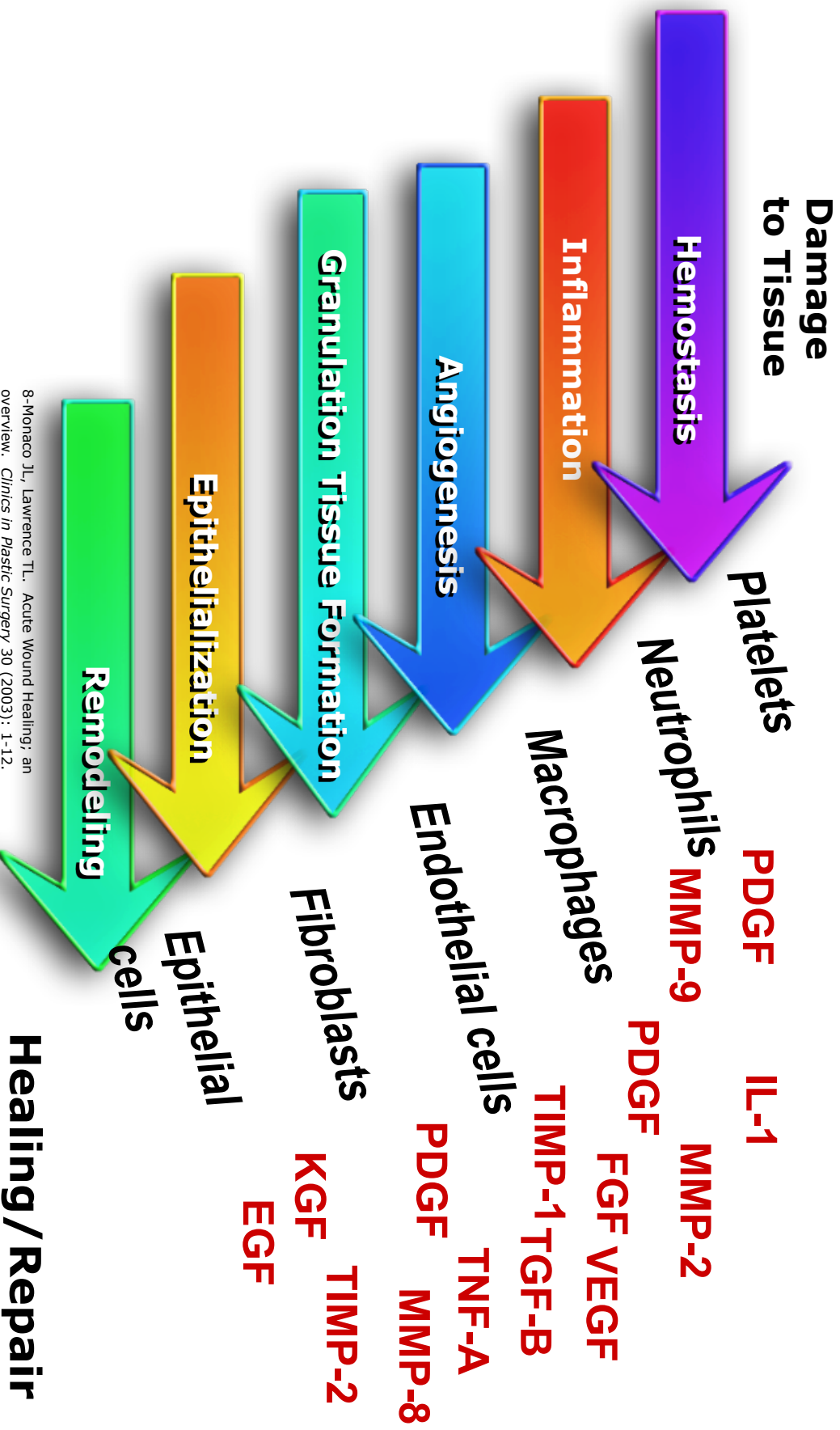
- Predominates as primary wound healing activity approximately 21 days after injury
- MMPs involved with breakdown of collagen
- Nature of wound matrix changes with scar remodeling
  - Immature scar contains disorganized fine collagen fibers
  - Replaced by thicker fibers (type 1 collagen) arranged in an orientation paralleling skin stresses
- Remodeling essential to the formation of a strong wound

# Remodeled Wound Strength

- Wound strength
  - 1 week after closure = 3% of normal tissue
  - 3 weeks after closure = approx. 20% of normal
  - 3 months after closure = 80% of normal tissue
- Remodeling continues for up to 24 months but will never regain strength of normal tissue



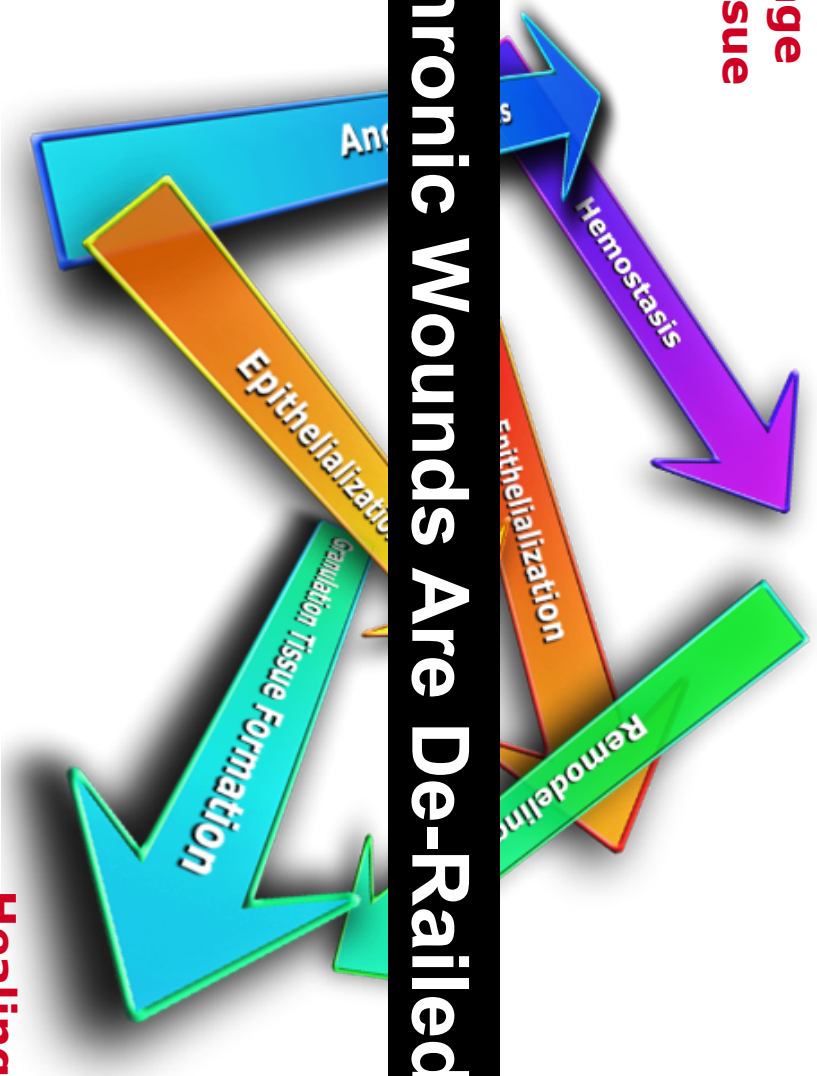
# Events of the Normal Healing Process



8-Monaco JL, Lawrence TL. Acute Wound Healing: an overview. *Clinics in Plastic Surgery* 30 (2003): 1-12.

# Healing Cascade in Chronic Wounds

**Damage  
to Tissue**



# Acute Wounds

“normally proceed through an orderly and timely reparative process that results in sustained restoration of anatomic and functional integrity”



## Wound Chronicity

- Accepted term, but no real definition agreed upon
- Most consider wound not healed or healing well by 4 weeks to be chronic
- Much of the literature defines it as a wound that has not healed in 12 weeks
- Centers for Medicare and Medicaid Services often consider 4 weeks before use of advanced products
- Term likely should be defined by the individual wound and the patient comorbid conditions



# Focus on Time to Healing: Use of Prognostic Indicators

## Venous Leg Ulcers

- <40% reduction in wound size by week 4
- Unlikely to achieve complete wound closure at 24 weeks<sup>1,2</sup>

## Diabetic Foot Ulcers

- <50% reduction in wound size by week 4
- Unlikely to achieve complete closure at 12 weeks<sup>3</sup>

1. Gelfand JM. *J Invest Dermatol*. 2002;119:1420-1425. 2. Phillips TJ, et al. *J Am Acad Dermatol*. 2000;43:627-630.  
3. Sheehan P. *Diabetes Care*. 2003;26:1879-1882.



# Chronic Wounds

“have failed to proceed through an orderly and timely process to produce anatomic and functional integrity, or proceeded through the repair process without establishing a sustained anatomic and functional result”

Mustoe TA, O'Shaughnessy K, Kloeters O. Chronic wound pathogenesis and current treatment strategies: a unifying hypothesis. *J Plast Reconstr Surg.* 2006;117:35-41.

# What Makes a Wound Chronic? Is It Really 30 Days?

## Time Driven?

- Excessive proteases
  - Degraded
  - Growth factors
  - Matrix proteins
  - Cell surface receptors
- Prolonged inflammation
- Cellular senescence
- Increased bacterial load
- Inadequate / inappropriate treatment

## Patient Driven?

- Diseases or conditions
  - Competing for oxygen or metabolic resources
- Medications
  - Steroids
  - Immunosuppressive agents
  - Chemotherapy
- Patient adherence
  - Diet/blood glucose
  - Smoking
  - Off-loading

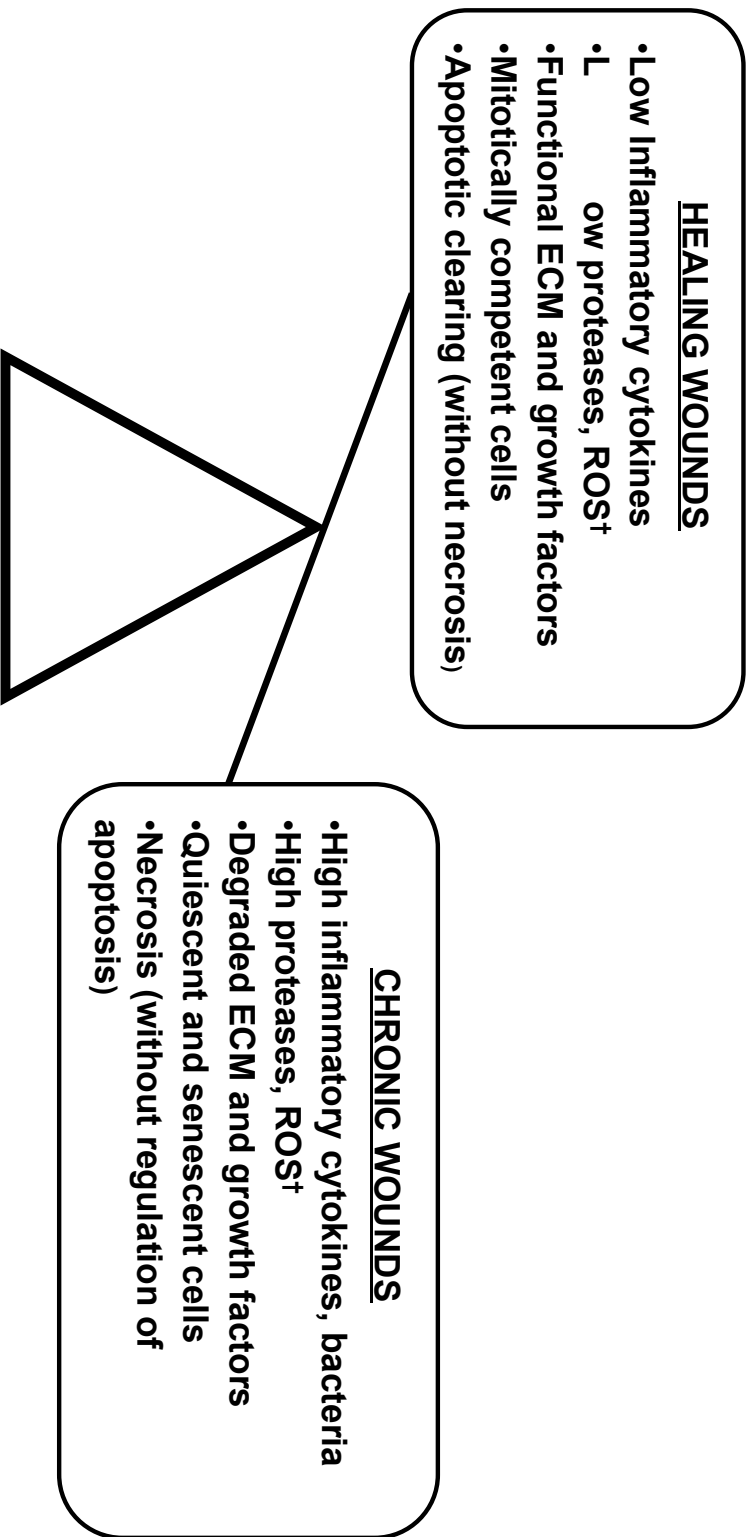
# Treat the Whole Patient....

- Complete history and physical exam
  - Review of systems
  - Family history
  - Surgical history
- Medications
  - Rx and OTC
  - Allergies
- Social history
- Smoking / alcohol history

*Not just the “hole” in the patient...*

Mast BA, Schultz GS. Interactions of cytokines, growth factors, and proteases in acute and chronic wounds. *Wound Repair Regen.* 1996;4(4):411-442.

# The Microenvironment of the Chronic Wound is imbalanced



Lobmann R, Shultz G, Lehnert H. et al. Proteases and the diabetic foot syndrome: mechanisms and therapeutic implications. Diabetes Care. (2005) 28:461-471.

# So What Can “Derail” Wound Healing

- Patient Factor
  - Etiology and Co-morbid conditions
  - Healability
  - Patient centered factors
- Wound Factors
  - Bacteria and biofilm
  - Necrotic tissue or other debris
- Treatment Factors
  - Need for advanced modalities

# Patient Factors





# “Healability”

- **Healable**

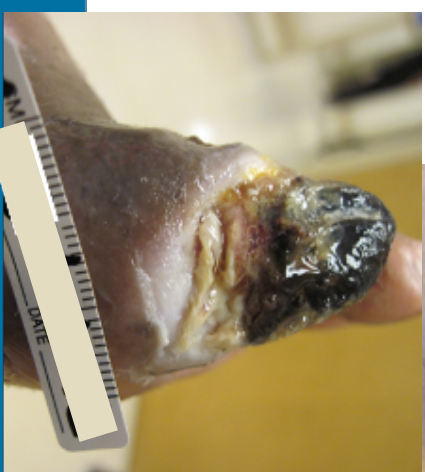
- With adequate blood supply that can be healed as long as the underlying problem can be addressed

- **Maintenance**

- Have healing potential, but also have patient or health system barriers compromising healing, including patient non-adherence to treatment plan or healthcare resource limitations

- **Non-Healable**

- Including palliative wounds, cannot heal because of irreversible causes or associated illnesses including critical ischemia or non-treatable malignancy



# Stress and Wound Healing



## **•42 married couples attended 2 hospital visits where each was given a blister wound**

- On first visit, interview was about supportive aspects of relationship and second was about areas of conflict in relationship
- Blister wounds healed more slowly following conflict interviews than after support interviews
- Couples categorized as high-hostile were slower healers

Kiecolt-Glaser, et al. Hostile marital interactions, pro-inflammatory cytokine production and wound healing. Arch Gen Psychiatry 2005

# Stress and Wound Healing

## • Mucosal Wound Healing Impaired by Exam Stress

- 2 punch biopsy wounds placed on hard palate of 11 dental students.
- First biopsy during summer vacation
- Second on contralateral side 3 days before first major exams of term
- 3 days (40%) longer to heal pre-exam wounds
- Decreased levels of IL-1 and slower healing during exams

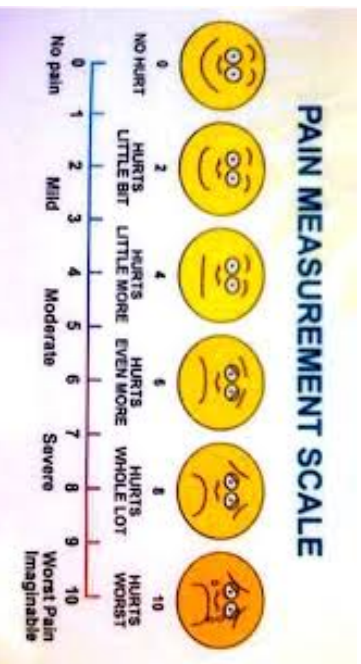
Marucha, et al. Psychosomatic Medicine 1998



# Address and Manage Pain



# Plan for the Pain

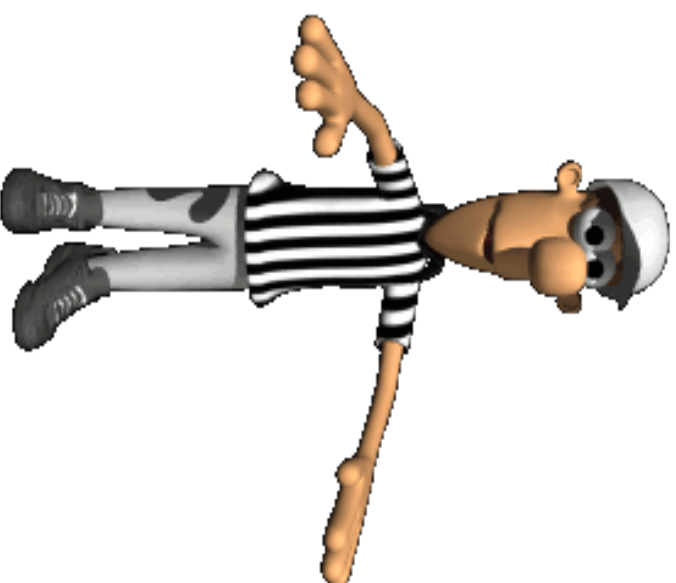


- Nursing - pre-medicate patient
  - Others: Call ahead!
  - Medicate prior to leaving facility for procedure
  - Take once at appointment if they have a driver
- Use topical anesthetics



# Avoid Verbal Anesthesia

- “It’s almost over”
- “It’s OK”
- “Try to calm down”
- “Don’t move!”
- “I’m sorry!”



# Other Patient Centered Factors

- Co-morbid conditions
- Living conditions
- Non-adherence to treatment plan
- Consider alternative plan??
- Smoking
  - Poor dietary intake
    - Bad food is cheaper than good food





# Nutritional Status

- Protein assessment
  - Serum albumin
  - Pre-albumin
  - Total lymphocyte count
  - CRP / Sed Rate
  - Deviation from UBW
- Carbohydrates
- Fats
- Vitamins
- Minerals
- Water



# Smoking



- Impedes healing by:
  - Local vasoconstriction and hypoxia
  - ↑ CO delivery to wound
- Endothelial and vasomotor dysfunction
- Accelerated atherosclerosis
- ↑ platelet activation
- ↓ collagen synthesis
- ↑ risk of post-op wound infection and rupture

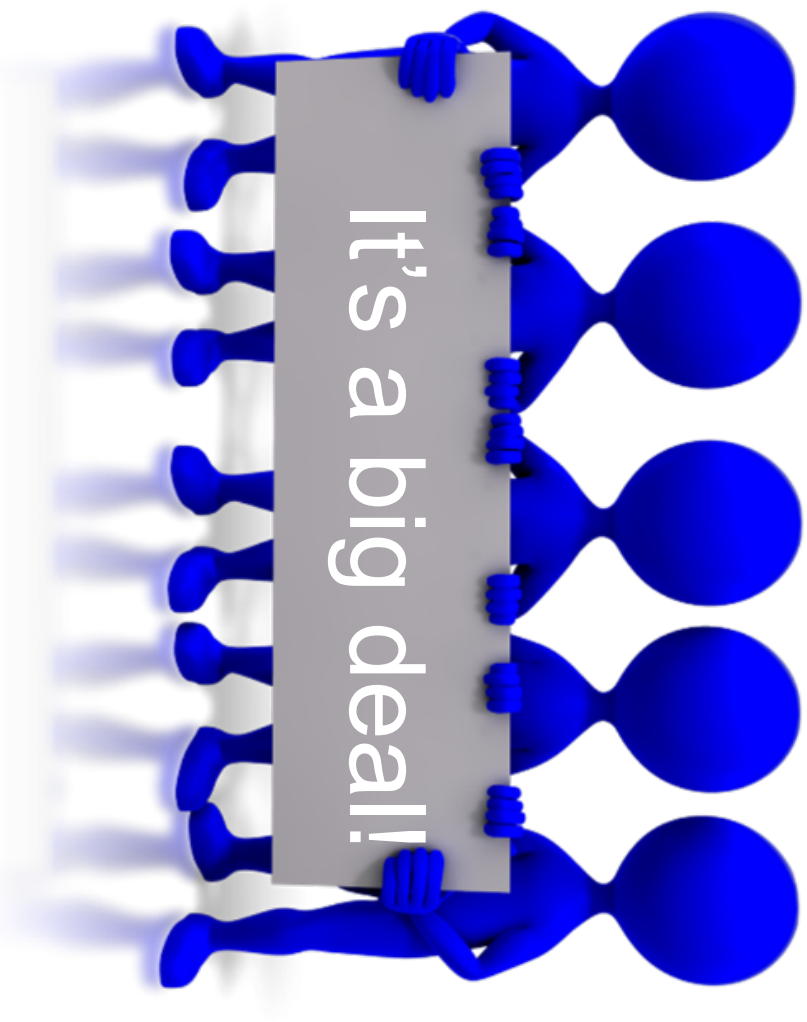


# Medications

- Systemic Steroids
- NSAIDs
- Methotrexate
- Heparin
- Hydroxyurea
- Amlodipine/Nifedipine
- Warfarin
- Select chemotherapeutics
- Povidone

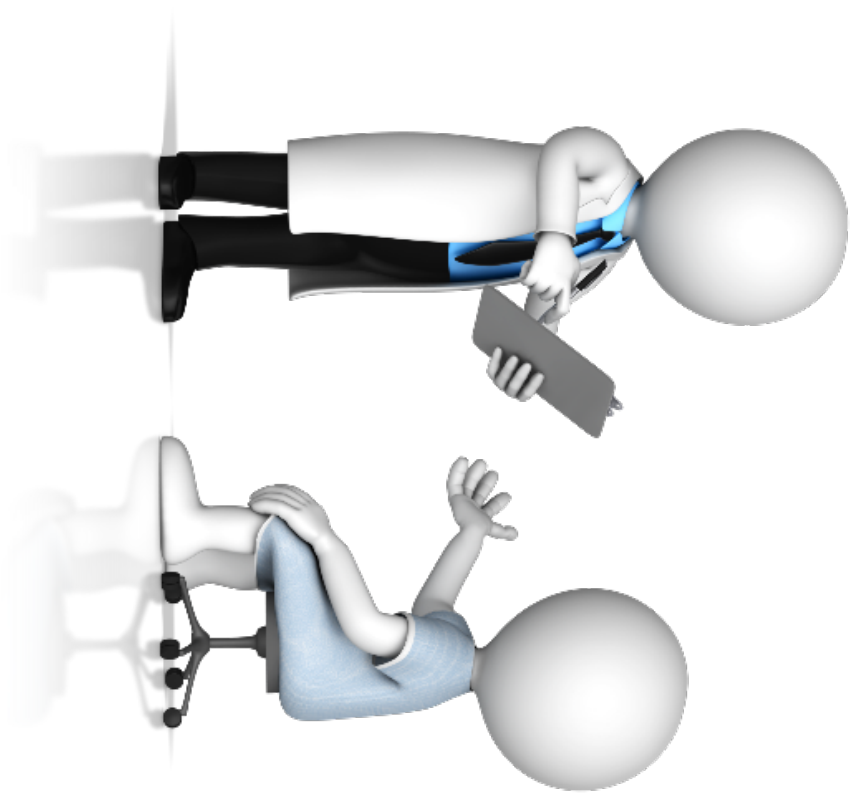


# Etiology



# Treat the Cause

- Good patient and family history critical
- Determine the etiology
- Assess ability to support
  - Offloading / pressure redistribution
  - Compression
  - Glucose control
  - Vascular interventions
  - Nutritional support
- Assess potential for healing
- Assess comorbid conditions

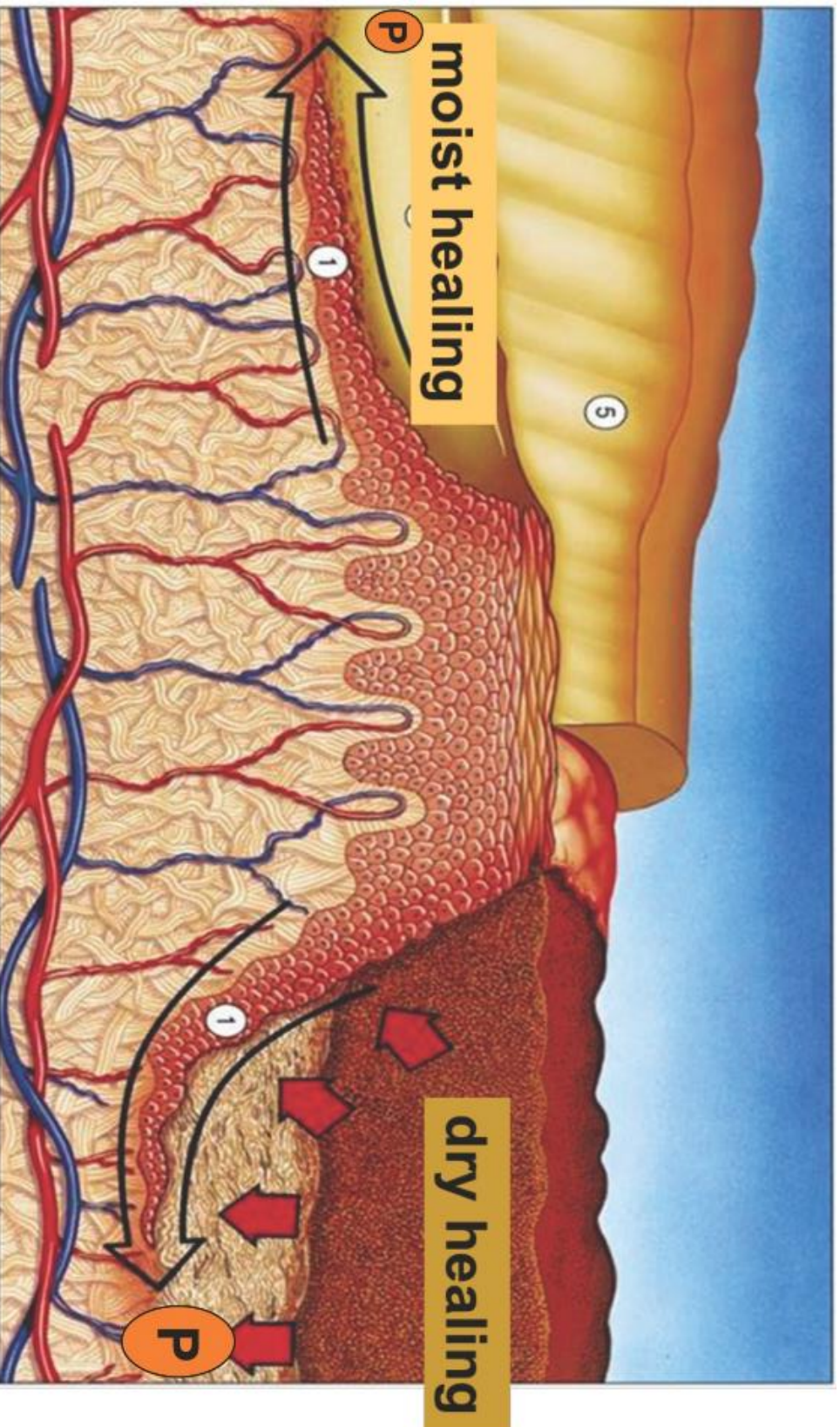


# Wound Factors

- Wound environment
- Necrotic / devitalized tissue
- Bacteria and biofilm

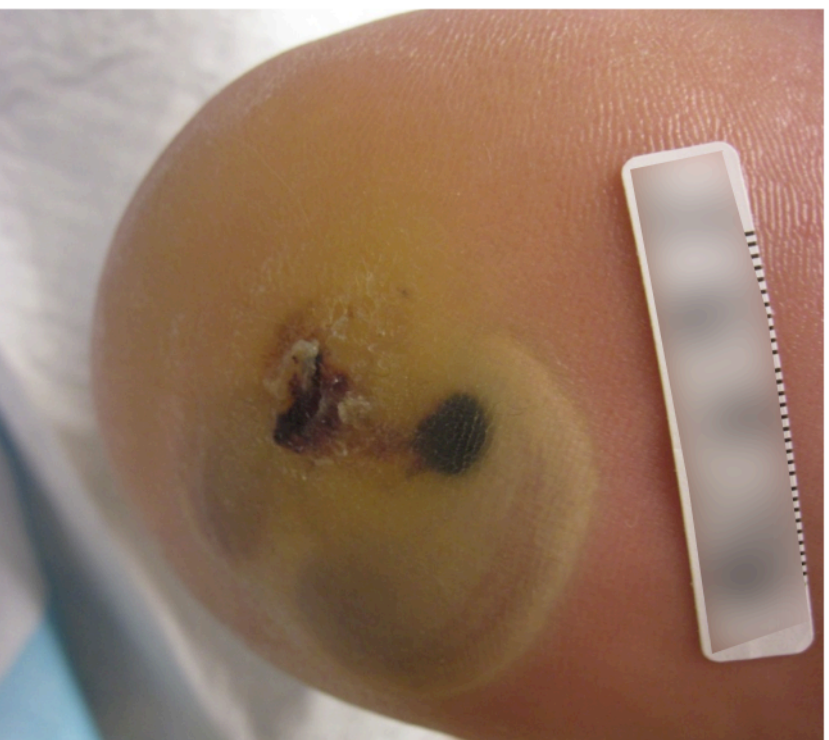


# Epithelial Healing of Deep Skin Wounds



Winter GD. Formation of the scab and the rate of epithelialization of superficial wounds in the skin of young domestic pigs. *Nature*. 1962;193:293-294.

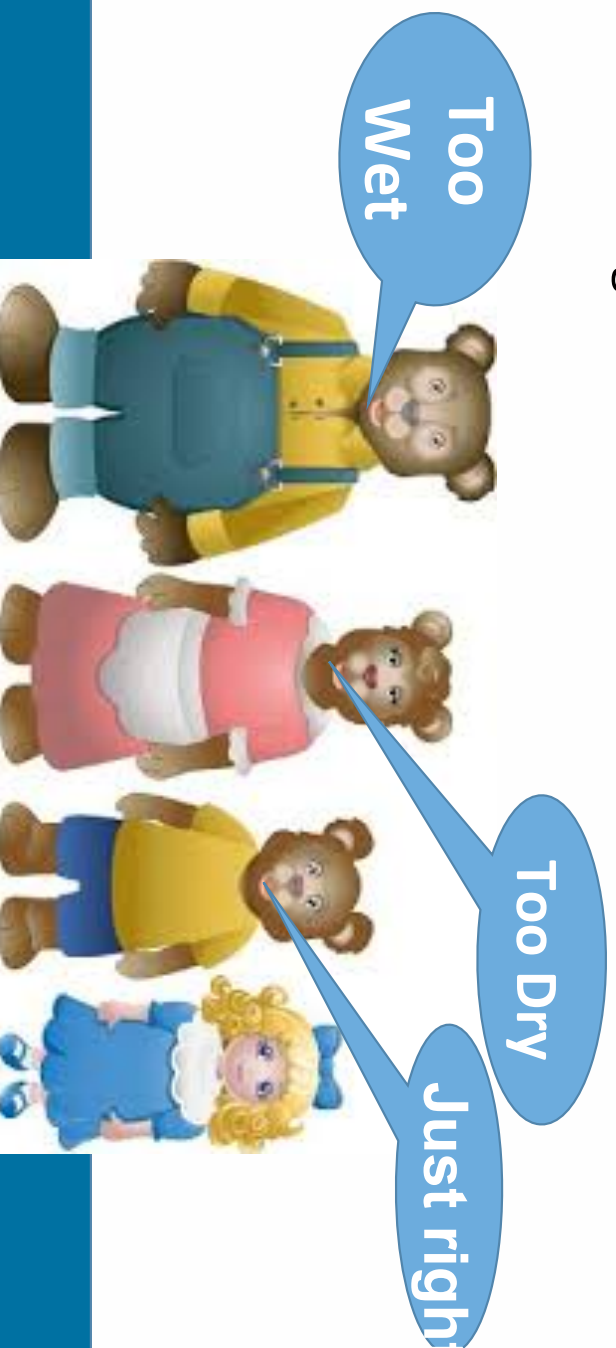
# Epithelial Migration Beneath a Blister





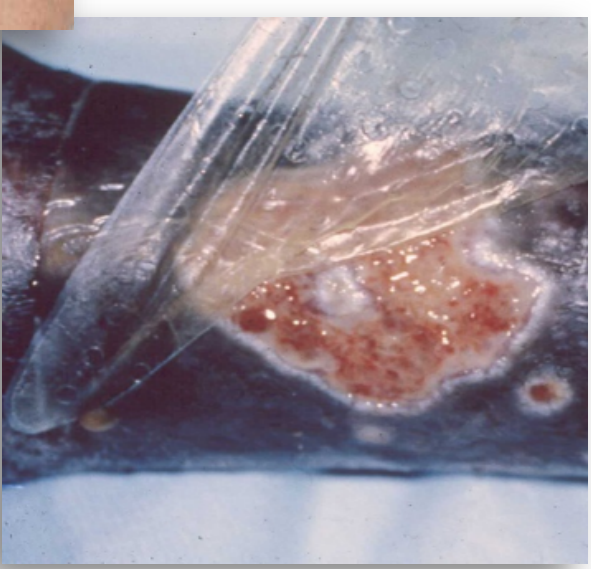
# Moist Wound Environment

- Insufficient moisture in exposed wound tissues causes desiccation and cell death
- Excessive moisture due to exudate inhibits cell proliferation and breaks down matrix components
- Moisture balance in the wound bed is maintained by appropriate choice of dressings



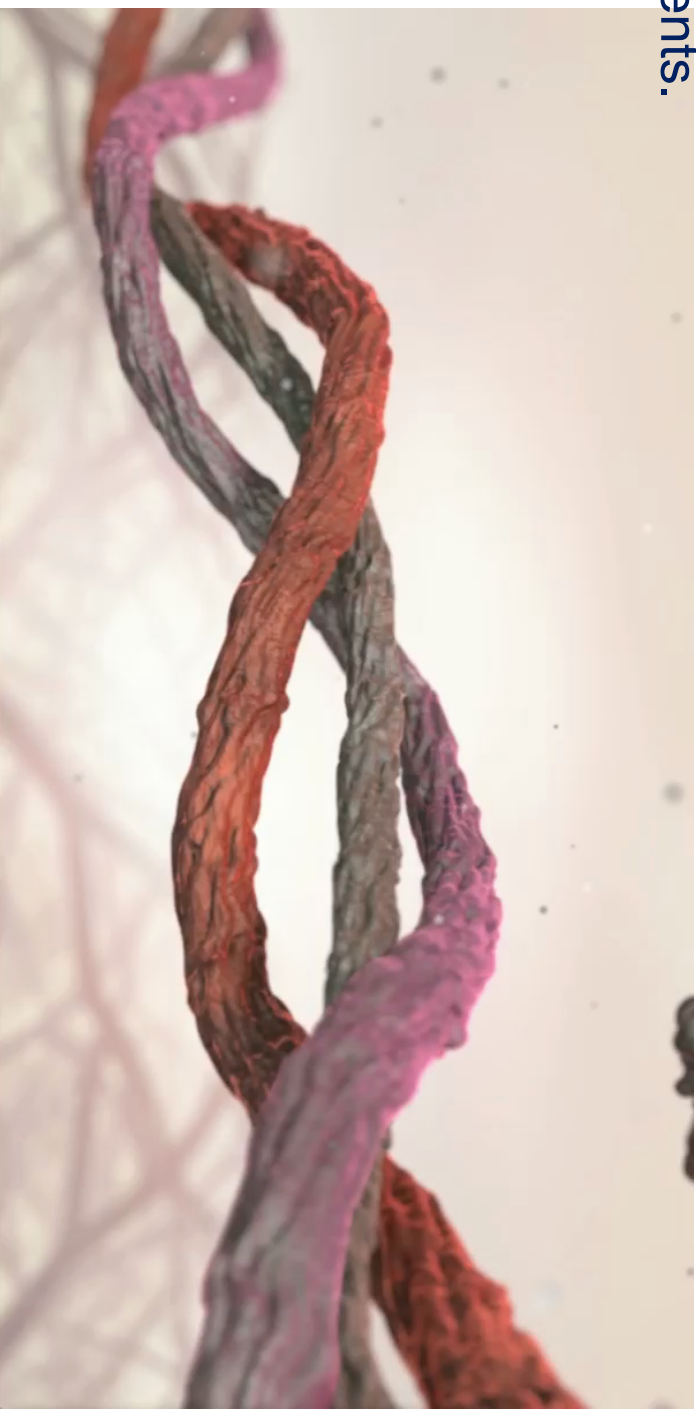
# All Wound Fluid Is Not Alike

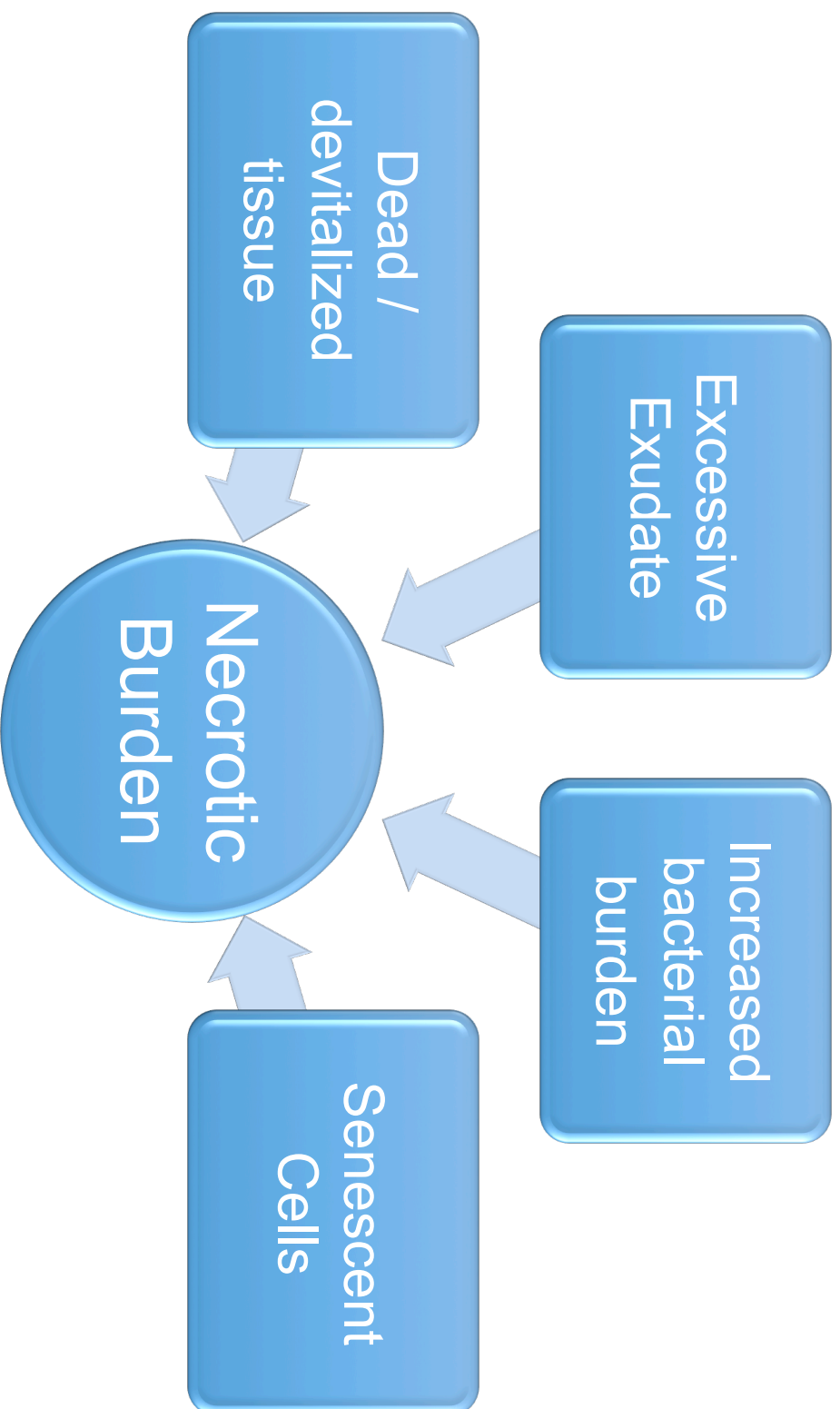
- Acute vs Chronic Wound Fluid
  - Metabolic activity of cells
  - Growth factor availability
  - Levels of pro-inflammatory cytokines (TNF- $\alpha$ , IL-1b, etc.)
- Balance between MMPs and TIMPs



# Sequential Degradation of ECM

- Chronic wounds typically have high levels of MMPs. These MMPs sequentially degrade the ECM delaying healing.
- First collagenases (MMPs 1 and 8) cause initial breakdown of vital structure
- Next gelatinases (MMPs 2 and 9) further degrade already damaged ECM into even smaller components.





# Necrotic Tissue

- Moist, devitalized tissue supports the growth of pathological organisms
- Bacteria thrive in the presence of necrotic tissue
- Provides a medium for infection
- Initiates an inflammatory response
- Retards wound healing
- Acts as a barrier to new granulation

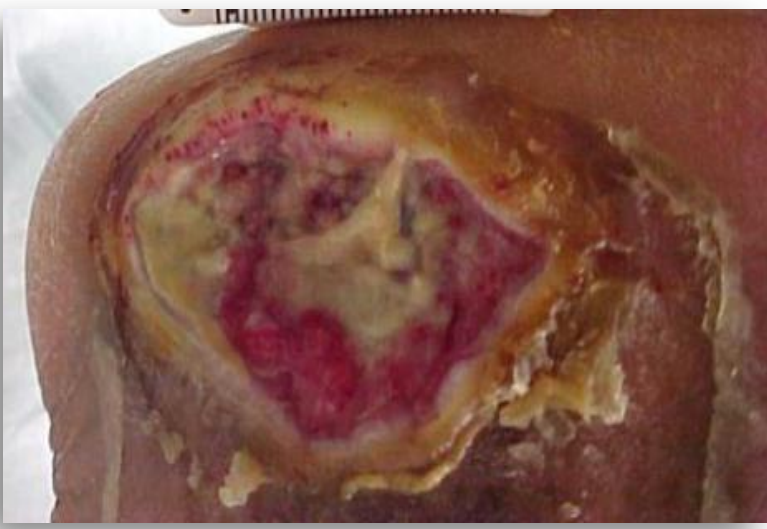




# Eschar



# slough



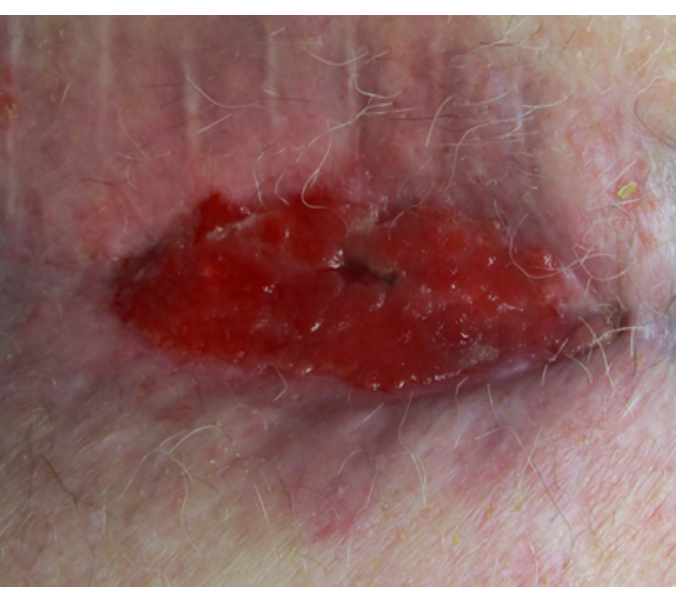
# Yellow Stuff??





# Senescent Cells in Chronic Wounds

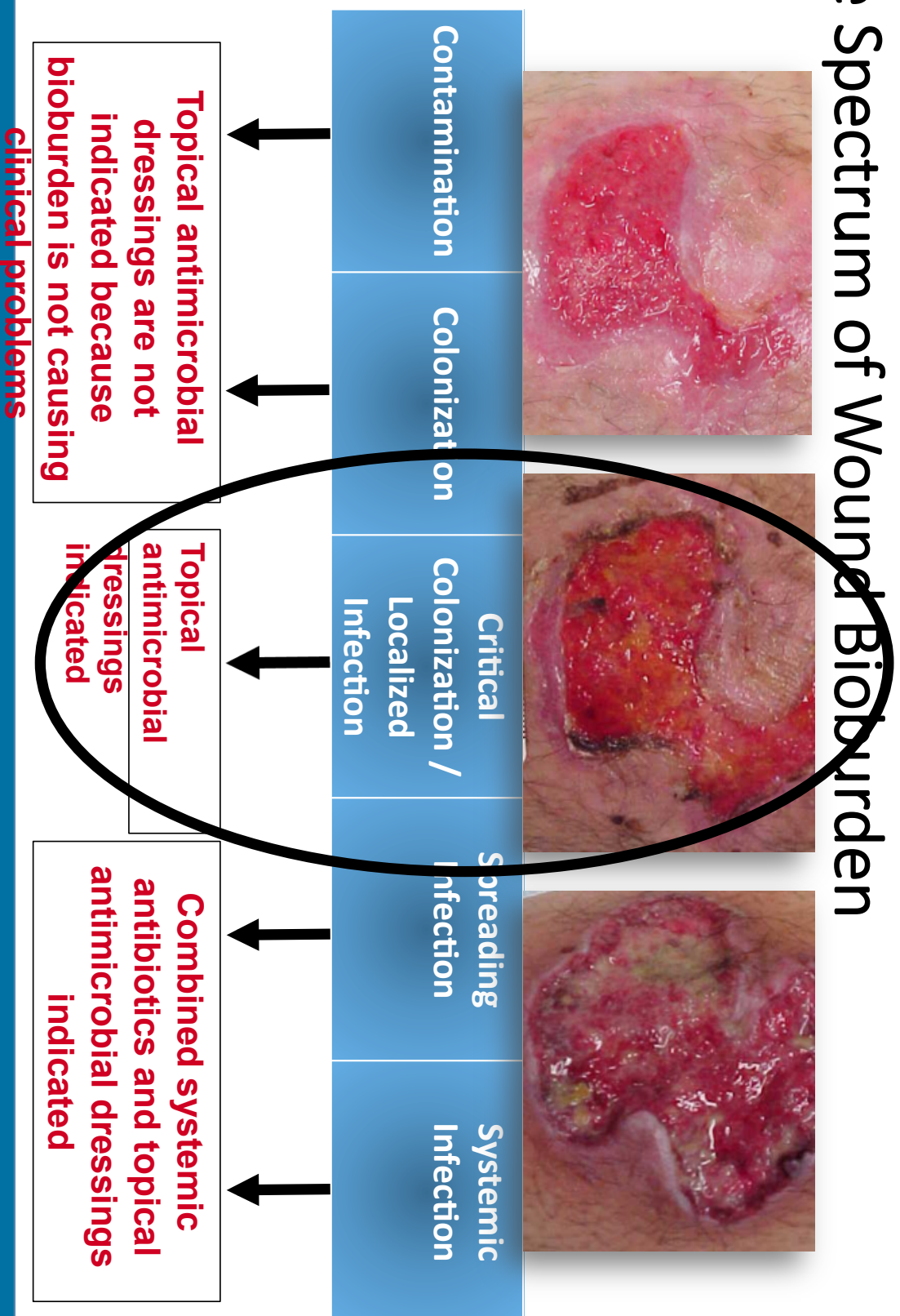
- Metabolically active, but nonproliferative or proliferating at greatly reduced rates
- Unresponsive or sluggishly responsive to chemical and physical stimuli
- Decreased migration rates
- Decreased production of matrix proteins and growth factors



# Defining Bioburden

- Degree of microbial contamination or microbial load; the number of microorganisms contaminating an object<sup>1</sup>
  - Assumed to be quantified relative to the object
- In wound management and wound healing, often a statement as to the presence of bacteria on a wound, as well as a qualitative descriptor of the bacterial status of a wound
- As a result, it has also become a term used to document and consequently rationalize and support the use of various treatment alternatives
  - Specifically dressings and devices known to impact surface wound bacteria, such as antiseptics, antimicrobial dressings, and modalities such as ultrasound

# The Spectrum of Wound Bioburden

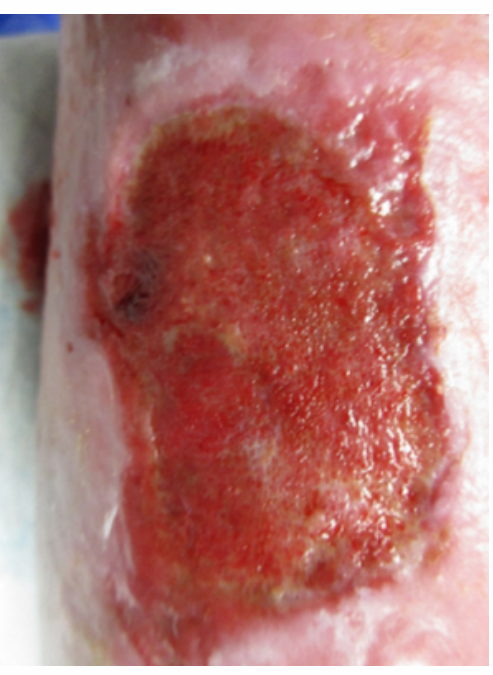


## Critical Colonization

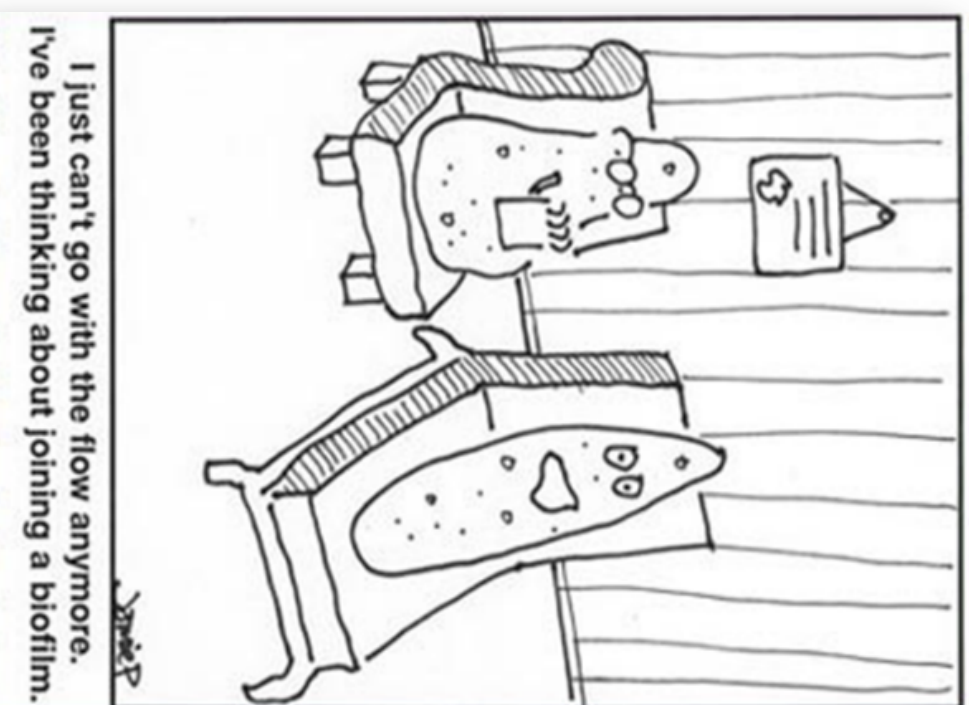
- Presence of bacteria on the wound
  - Healing process compromised
  - No standard signs of infection
- May delay healing via bacterial competition with tissue cells for oxygen and nutrients, production of bacterial toxins, and inflammatory mediators
- Also referred to as “locally infected”  
(vs spreading infection)

# Signs of Critical Colonization

- Granulation tissue
  - Color
  - Friability
  - Absent or abnormal
- Odor – subtle or dramatic change
- Increased/high exudate levels
  - Wounds attempt to “flush out” foreign particles or chemicals

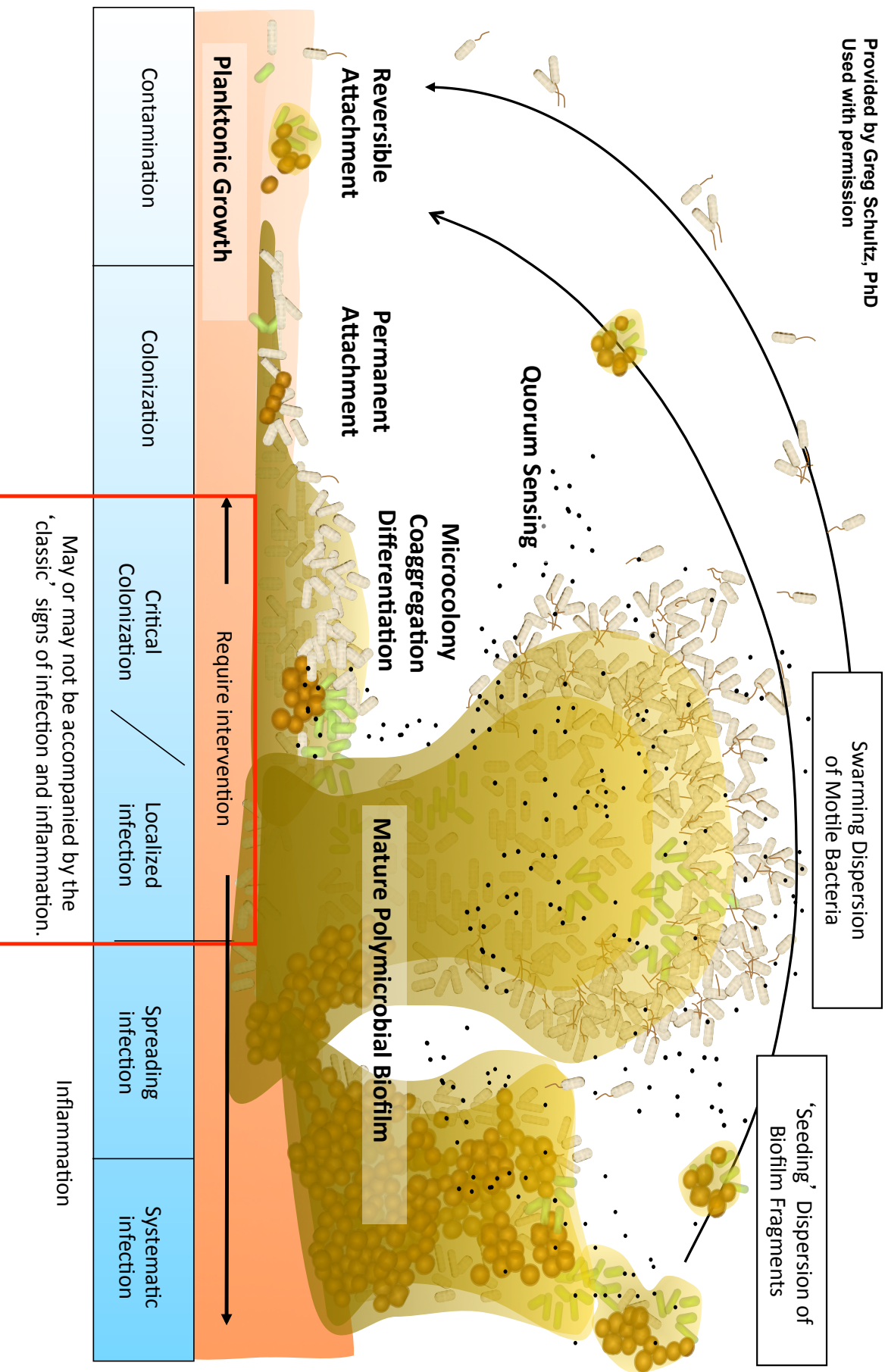


Bacteria are getting  
smarter...we must also.





Provided by Greg Schultz, PhD  
Used with permission





# Bacteria and Biofilms

- Bacteria grow in various forms:
  - Planktonic
    - Free floating
    - Antibiotics can destroy easier
    - Most antibiotic testing is on planktonic
  - Biofilms
    - Complex communities of bacteria (and other organisms) that adhere to solid surfaces
    - Embedded in an extracellular polysaccharide matrix

# 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

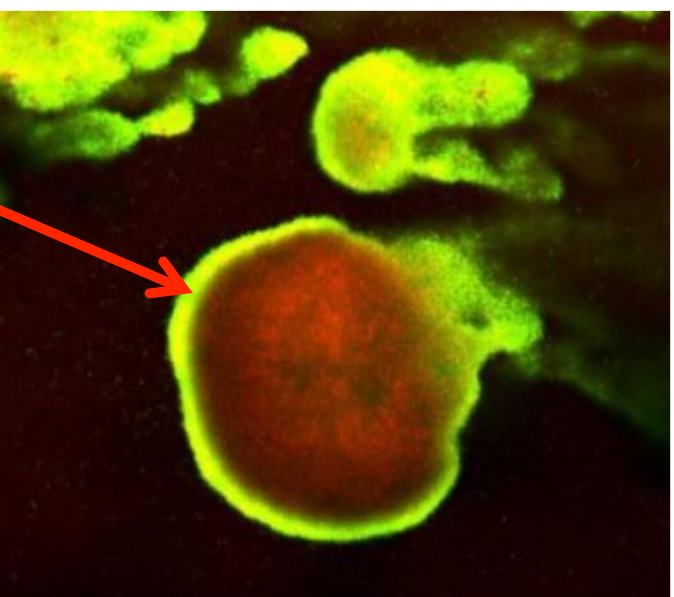
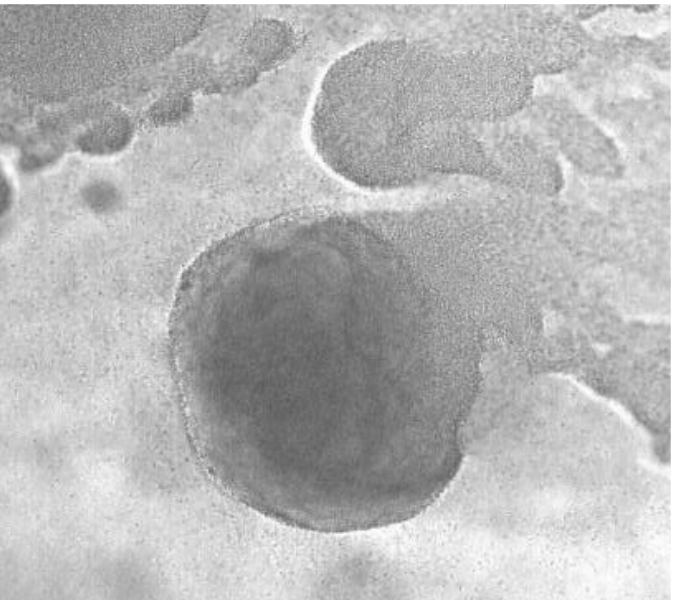
# Why are Bacteria in Biofilms Hard to Kill?

- Exopolymeric material of the biofilm
  - Dense matrix impairs diffusion of large antibodies
  - EPM materials chemically react (neutralize) microbicides
- 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

- Phil Stewart, Montana State University Center for Biofilm Engineering.



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

# 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
  - Better wound cleansing!
  - Followed by antimicrobial dressings
- Cadexomer Iodine / Iodine PVA foam dressings
- Surfactant gels
- Anti-biofilm gels and agents



# Bacterial Burden

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





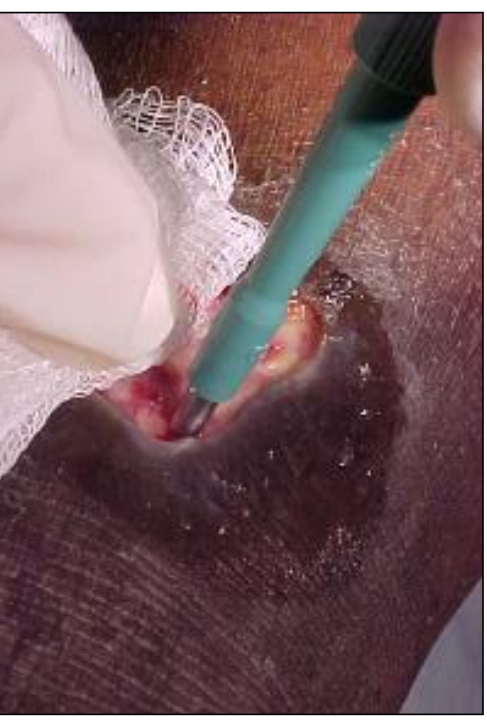
# Methods of Wound Culture

- Aspiration
- Biopsy
- Swab



# Quantitative Tissue Biopsy

- Historically the “gold standard” or at least best practice
- Painful (may need anesthetic)
- Skill Intensive
  - Unavailable in many settings
- Used more in research than clinical practice
- $>10^5$  (100,000) colony-forming units (CFU) per gram of tissue considered to be infected



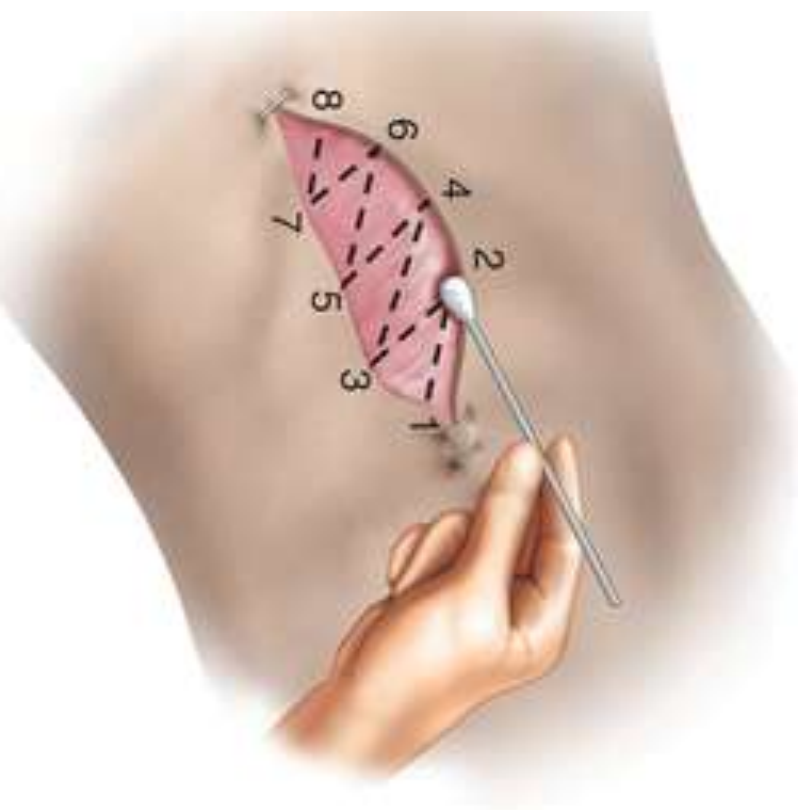
# Improve Swab Technique

- Thoroughly rinse wound surface with non-preserved saline/ cleanser
  - Don't swab through dressing residue, old exudate, necrotic tissue, blood
- Choose area that is free of non-viable tissue if possible
- Don't bother with dry surfaces
- Place in carrier, transport ASAP



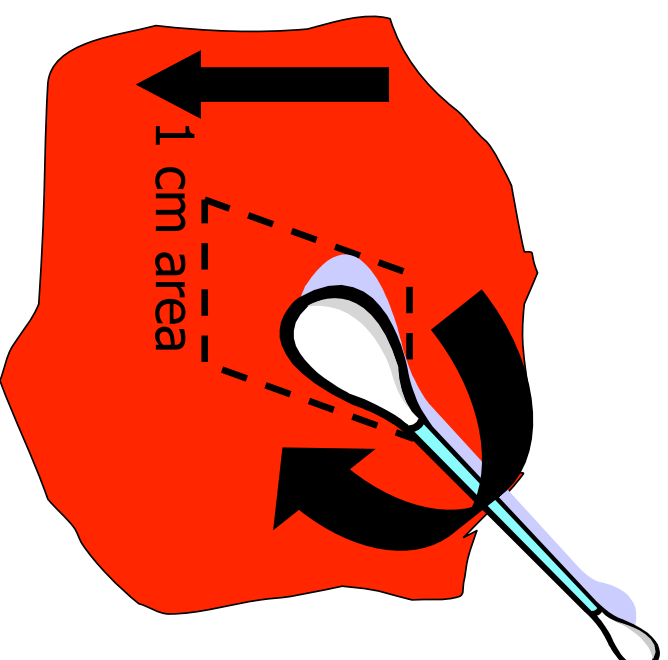
# Methods of Swab Culture

- **Z Stroke**
- Levine's Technique
- Deep Tissue Swab Culture



# Levine's Technique

- Clean the wound!
- Surface swab of a 1 cm<sup>2</sup> area of healthy tissue in the wound
- Press into wound to obtain fluid



# Levine's Technique





# Levine's Technique





# Levine's Technique



# Levine's Technique



**Location, Location, Location...**



# Tissue Swab Culture



So What to Do?

**Try to change  
the healing  
trajectory....**



# It Starts with Assessment: The Key To Decision Making!

