Wound Care

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...Begins With A Thorough Patient Assessment

- Medical history, co-morbidities
- Watch for medications that may be counterproductive to healing
- Family history
- Social history including lifestyle, smoking, alcohol use
- Nutrition
- Patient and family level of understanding of current problem
- Footwear
- Speak with PCP regarding current medical condition
- Lower extremity assessment ( integument, vascular, neurologic, musculoskeletal )
Wound Care Must Be A Multidisciplinary Approach

- PCP
- General surgery
- Plastic surgery
- Orthopedics
- Infectious disease
- Hyperbaric medicine

- Endocrinology
- Podiatry
- Dietician
- Pedorthist/Orthotist
- Physical therapy
Mechanisms of Wound Repair

- Primary repair – sutured incision, good wound edge approximation with minimal tension
- Delayed primary repair – secondary closure as with a contaminated or infected wound where surgical debridement or incision or drainage are performed, with closure in 3 – 5 days
- Secondary repair – chronic wounds, ulcerations, wounds where there is no edge approximation, contributes to stalled healing, excessive inflammation
- Secondary repair can be brought to primary repair with closure or skin graft if wound bed, vascular status are appropriate
Local Tissue Concerns Affecting Wound Healing

<table>
<thead>
<tr>
<th>Macrosscopic</th>
<th>Microscopic</th>
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<tbody>
<tr>
<td>• Bioburden</td>
<td>• Microcirculation deficit</td>
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<tr>
<td>• Undermining</td>
<td>• Growth factor deficit</td>
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<tr>
<td>• Fibrosis</td>
<td>• Cell proliferation deficiency</td>
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<tr>
<td>• Exudate</td>
<td>• Level of matrix metalloproteases (MMP’s)</td>
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<tr>
<td>• Rim scar</td>
<td>• Medications, either topical or oral, that inhibit normal inflammation (NSAID), or are cytotoxic (certain topical anti-infective liquids)</td>
</tr>
</tbody>
</table>
Three Phases of Healing – Formation of an Ulceration Initiates ....

1. Inflammation phase
   - provides initial vascular influx with macrophage PMN’s, platelets, RBC’s
     - Macrophages remove bacteria and necrotic tissue
     - Growth factors begin to be produced by the PMN’s
     - Collagen production is stimulated by the PMN’s

2. Migratory phase
   - extracellular matrix firms up with fibroblast proliferation
     - fibroblasts begin to migrate from the edges of the wound contributing to wound contraction
     - further angiogenesis generates new granulation and formation of epithelium, leads to.....

3. Proliferative phase
   - continued cell migration leads to further vascular and epithelium formation and healing
Steps to Success on the Cellular Level...

- Extracellular matrix fibroblasts begin to form collagen
- The extracellular matrix proliferates through collagen, fibrin and fibronectin interaction allowing for wound bed formation
- Granulation begins with vascular/collagen production
- Epithelization occurs from the wound margins as the granulating bed fills in
Turning the Tide...

The goal is to bring the wound from the inflammatory to the proliferative phase

- Provide an adequate vascular status
- Bio-burden, scar tissue, fibrosis, excessive granulation, undermining need to be reduced
- Reduce proteolysis of healthy tissue by MMP,s
- Stimulate tissue repair by the addition of growth factors
- Sterile wound environment free of infection
- Appropriate unloading improves micro-vascularization, reduces excess MMP activity, promotes healthy granulation
MMP’s - The Good and the Bad

Normal matrix metalloprotease levels

- Promote fibrin clot formation
- Bacteriostatic
- Stimulate migration of cells
- Promote action of growth factors

Excess amounts of matrix metalloproteases

- Causes wound stagnation by breaking down extracellular matrix protein, reduced growth factor function
- Tissue necrosis increases with elevated protease activity
- Stimulates elastin and collagen breakdown
- Fibroblasts become abnormally effective
- Contribute to an inadequate micro-vascular environment
The Role of Growth Factors

- Monocytes during the inflammatory phase of healing produce proteins called growth factors.
- Centrally located growth factor proteins direct fibroblasts to stimulate target cells to proliferate a particular cell line (vascular, fibrous).
- Peripheral growth factor proteins stimulate formation of epithelium.
- Stagnant wounds are lacking in adequate growth factor function.
Bio-burden and MMP’s Must Be Reduced To Return To The Inflammatory Phase Of Healing

Methods to accomplish this are...

- Cleansing of the wound
- Debridement of the wound
Wound Cleansing

- Removes loosely adherent material
- Manually lavage with sterile saline in an 18g syringe generates 10psi of pressure
- Pressure irrigation system generates between 4 – 15psi of pressure
- Care must be taken to avoid aggressive scrubs and toxic antiseptics
Wound Debridement

A critical component to wound healing

- Mechanical
- Sharp
- Autolytic
- Enzymatic

Vague levels of debridement applied when referring to depth of debridement for Medicare coding

- Skin partial thickness
- Skin full thickness, epidermis and dermis
- Skin full thickness, epidermis, dermis and subcutaneous
- Skin full thickness, epidermis, dermis, subcutaneous, muscle
- Skin full thickness, epidermis, dermis, subcutaneous, muscle and/or bone
Mechanical Wound Debridement Methods

- **Whirlpool**
  - too aggressive, damages healing tissues, macerates, increased infection rate

- **Ultrasonic**
  - effective ultrasonic vibration combined with sterile saline, less pain

- **Wet to dry dressing**
  - more common in the past before better options were available, traumatic to healing tissues, painful

- **Pulsed lavage**
  - less pain, saline irrigation under controlled pulsed pressure removes necrotic tissue, less traumatic

- **Versajet**
  - combines saline high pressure jet for debridement with vacuum for debris removal, cleans and debrides
Sharp Wound Debridement

**Indications**
- Undermining, tunneling, sinus tract
- Extreme necrosis, fibrosis
- Sepsis

**Contraindications**
- Gangrene
- Tcom less than 30mmHg
Autolytic Wound Debridement

Pros

• Moist retentive dressing, promotes the ability of the body to remove necrosis
• Painless, safe with no damage to healthy tissue
• May be combined with mechanical debridement for more rapid healing response

Cons

• Very slow process
• Can not be used if ischemic condition or gangrene
• May contribute to bacterial overgrowth
Enzymatic Wound Debridement

- Proteolytic enzymes break down collagen, removing necrotic tissue, matrix metalloproteases
- Minimal effect on normal tissue
- Painless
- Used in cases of arterial insufficiency where sharp debridement is contraindicated
- Accuzyme (papain/urea)
- Santyl collagenase (collagenase enzyme)
- Panafil (papain/urea/chlorophyllin copper)
Lab Work Considerations

Chronic ulceration may accompany multiple abnormalities

- Complete blood count, hemoglobin, platelet count
- Alk phos, SGPT, SGOT to check liver function
- Renal function with albumin, creatinine, BUN
- Fasting glucose, HA1C if suspect diabetes
- Elevated homocysteine levels often with chronic ulceration, counterproductive to healing
The Role Of Homocysteine In Chronic Wounds

- Elevated in 50% of all patients with chronic non-healing skin ulcer
- Elevated in 63% of diabetics with neuropathy
- Increased risk of atherosclerosis, contributing to blood pressure, vasodilatation and arterial inflow problems
- Restoration of normal homocysteine levels can also restore normal healing

Nitric Oxide/Homocysteine Interaction And Wound Healing

- Nitric oxide is a critical mediator of normal tissue repair.
- Its production and bioactivity regulate angiogenesis, granulation tissue formation, epidermal migration, collagen deposition, and micro-vascular homeostasis.
- Optimal nitric oxide activity is required for full expression of multiple growth factors important in normal wound healing.
- Usually deficient nitric oxide bioactivity causing impaired diabetic wound healing.
- Wound contraction and epidermal migration are the mechanisms responsible for wound reduction – endogenous nitric oxide production is a prerequisite for epidermal cell migration and may function to switch on the migrating epidermal cell.
The Nitric Oxide/Homocysteine Connection

- Elevated homocysteine levels correlate with a significant reduction in nitric oxide bioactivity.
- This leads to impaired wound healing with reduced granulation tissue formation, growth factor function, angiogenesis, collagen deposition, epidermal migration, re-epithelialization, ALL CAUSED BY inhibition of nitric oxide production.
- Elevated homocysteine levels alter normal thrombosis and wound matrix structural integrity.
What Can Be Done?

- Chronic wound management requires combination therapy of L-methyl folate, pyridoxal 5’ phosphate and methylcobalamin to reduce elevated homocysteine levels.
- Allows normal nitric oxide bioactivity.
- Metanex, folate, B12.
- There is ongoing research regarding wound nitric oxide bioactivity as a promising diagnostic indicator for diabetic foot ulcer management.

Grading Ulcerations

Wagner Classification

- Diabetic ulcers only
- Grade 0, erythema, no break in skin
- Grade 1, superficial, partial skin thickness penetration
- Grade 2, full skin thickness penetration, ligament, tendon, capsule, fascia, no infection, abscess or osteomyelitis
- Grade 3, Deep ulcer with infection, abscess, osteomyelitis
- Grade 4, forefoot gangrene,
- Grade 5, Extensive gangrene, entire foot
Grading Ulcerations

National Pressure Ulcer Advisory Panel Classification

- All types of skin ulcers except diabetic ulcer
- Stage 1, non–blanchable erythema of skin
- Stage 2, superficial partial thickness skin loss, ulcer involves epidermis alone or epidermis and dermis (abrasion, blister)
- Stage 3, full thickness skin penetration, subcutaneous, extends to deep fascia
- Stage 4, full thickness skin penetration, subcutaneous, with necrosis and muscle, tendon, capsule and/or bone involvement
Four Basic Types Of Skin Ulceration

- Venous
- Pressure
- Arterial insufficiency
- Diabetic
Venous Ulceration

- Usually seen on the lower leg, commonly at the medial malleolus or anterior lateral aspect of lower shin area
- Non-painful
- Often associated with edematous extremity, varicosities
- Stasis dermatitis may be concurrent
- Suspect a different diagnosis if the lesion persists longer than 3 months with treatment, consider biopsy and culture (pyoderma, collagen vascular disease, fungal lesion, malignancy)
Venous Ulceration Requires Arterial Considerations

- Venous insufficiency can be combined with reduced arterial inflow.
- In treating venous insufficiency with edema, amount of compression must be considered when arterial insufficiency is also present.
- ABI >0.8, palpable pedal pulses indicates venous ulceration.
- ABI < 0.7, arterial insufficiency, avoid compression.
- Diabetic may have falsely elevated ABI due to vessel calcification (TBI, oximetry more reliable).
- Venous duplex ultrasound confirms diagnosis of venous lesion and level of involvement (deep, superficial, perforators).
In All Cases Of Ulceration, Infection Must Be Avoided

- Initial culture may show a mixed bag of organisms with probable contaminants depending upon wound appearance,
- If the wound appears clean, may treat topically two weeks, with culture if no improvement after that
- Culture may show skin structure organisms staph. aureus, staph. epidermidis, also possible beta streptococcus, GI organisms, psuedomonas auerginosa
- Combined with clinical picture, moderate to heavy growth usually indicates infection
- Also perform fungal culture
- With cellulitis culture right away
- Extended antibiotic coverage may contribute to overgrowth of resistant organism
- MRSA is on the rise
Additional Factors To Consider That Affect Healing

- Control of concurrent systemic disease
- Smoker?
- Medications (immuno-suppressives, anti-inflammatories)
- Arterial vascular perfusion, tissue oxygenation
- Nutritional status
Venous Ulcer Treatment

- Judicious hosiery compression (beware of patient noncompliance)
- Intermittent pneumatic leg compression
- Elevation
- Debridement (sharp, autolytic, and/or enzymatic)
- Local wound care with sterile saline lavage, non-adherent lowest layer, maintain moist wound bed, control exudate
- Skin graft
Pressure Ulceration

- Caused by sustained pressure against an area of prominence
- Often associated with debilitated state or wheelchair bound
- Posterior heel breakdown common in Podiatry
- Pain may be present but not in all cases
- Initially noticed by patient as drainage on the sock or in the shoe, or by odor
Pressure Ulcer Care

- Unloading is critical to alter predisposing factors of excessive pressure to skin
  - foam boot, ankle collar, felt/foam padding, wheelchair with leg support, frequent turning of patient if bed ridden
- Nutritional status
  - adequate protein intake, serum albumin levels
- Culture for infection if clinically indicated
  - prefer topical antibiosis unless cellulitis
- Debridement is necessary to reduce bioburden
- Appropriate local wound care, maintain a moist wound bed, control exudate, non-adherent dressing, reduce peri-ulcer irritation
- Surgical debridement if sinus tract, deep recess, extensive necrosis, osteomyelitis
- Preventative surgery to remove soft tissue or osseous contributors to pressure
- Podiatric/Pedorthic intervention to reduce recurrence
Arterial Vascular Insufficiency
Ulceration

- Often more painful than venous or pressure ulcer
- Lateral malleolar area common, but can occur at any area of friction
- Combination of diabetic or venous ulcer with arterial insufficiency not unusual
- Skin and toenail trophic changes are a clue to arterial insufficiency
- Vascular findings are the real diagnostic tip - off
Arterial Vascular Findings

- Reduced or absent pedal pulses to palpation and handheld doppler
- Skin rubor or pallor
- Absent digital hair growth
- Temperature of skin may be cool to the touch
- Texture thin, dry and atrophic
- Claudication, nocturnal leg pain
- Doppler signal mono- or biphasic,
- ABI < 0.8, > 1.2; TBI < 0.75
- Ulcer level tissue oximetry < 40 mmHg
Treatment Of Arterial Insufficiency Ulceration

- Infection management
- Unloading
  - local padding, soft multiple density orthoses, internal and/or external shoe modifications, Cam walker, Bledsoe boot, healing shoe
- Podiatric/Pedorthic intervention
  - regular podiatric visits, appropriate hosiery, shoe gear, orthoses, shoe modifications
- Vascular intervention
  - tissue oximetry < 0.30, ulcer will not heal without vascular intervention
- Judicious debridement, done after satisfactory vascular status attained
  - pressure, enzymatic, or autolytic preferred over sharp especially if the patient cannot be re-vascularized
- Definitive surgical intervention (amp) if revascularization fails and gangrene
- Hyperbaric O2 if O2 challenge > 200 mmHg, especially if osteomyelitis
The Diabetic Ulceration

- Epidemiology and contributing factors such as neuropathy covered in the “Save That Foot Lecture”, this presentation is centered on treatment

- All steps below must be taken for success -
  - assess arterial vascular status (ABI, TBI, Tcom)
  - rule out infection with cultures (bacterial, fungal)
  - X-ray, MRI, triphasic bone scan to determine depth of ulceration, osteomyelitis
  - sharp debridement
  - off loading, appropriate wound care
  - nutrition
  - daily home care
  - patient and family education
Off Loading Techniques

- Our unique role in wound care as Podiatrists
- Most diabetic wounds are plantar lesions
- Off loading reduces friction, shear force, allows for new tissue granulation
- Many different methods depending on severity including casting, shoe modifications, healing shoes, padding, ambulatory modalities
- Orthoses and extra depth shoes are used for prevention of reoccurrence
Forefoot off loading negative heel shoe

Ankle collar offloads heel, diamond pattern sole for healing boot
Off Loading By Casting

- Below knee removable cast with custom modification of outer sole and foot bed (plastizote, PPT multiple densities, rocker sole)
- Removable Bledsoe boot
- Total contact cast (the ultimate in off loading when there is no infection)
  - Armstrong et. al., Diabetic Care, vol. 24, 2001, Total Contact Cast Superior to Half Shoe, Aircast in Off Loading
- All of the above provide off loading by redirecting plantar pressures across the entire bottom of the foot
- Total contact cast must be reapplied every 7 – 10 days
- Bledsoe and below knee cam walker daily removal for wound care
Total Contact Cast

Indications

- Non-infected ulcer
- Acute Charcot arthropathy
- Where patient compliance is in question

Contraindications

- Peripheral vascular disease
- Osteomyelitis
- Infected ulcer
- Gait instability
- Use with care in cases where there is neuropathy
The Wound V.A.C.

- Vacuum Assisted Closure Therapy – KCI, San Antonio, Texas
- Negative pressure wound therapy
- Consists of a pump that provides continuous or intermittent negative pressure in a closed environment to the wound site
- Occlusive sac attached to the pump that maintains pressure
- Special foam dressing applied to the wound sealed with the negative pressure sac
- Container attached to the sac collects drainage
Using The Wound V.A.C.

Indicated in the treatment of...

- Pressure ulceration,
- Diabetic ulceration
- Wound dehiscence
- Trauma
- Burns, skin grafts
Vacuum Closure

**Indications**

- Tissue deficit
- Extreme exudate
- Large, non–granulating wounds
- Decreased arterial vascular status
- Infection
- Local edema

**Contraindications**

- Anticoagulant therapy
- Active bleeding
- Necrosis, gangrene
- Osteomyelitis
- Over a body cavity
- Malignancy
Negative Pressure Closure Mechanism

- Pressure exerts mechanical stress to cells, increasing proliferation
- Promotion of autolytic debridement
- Increases growth factor production and activity
- Reduces edema, increases arterial inflow
- Eliminates exudate and infected tissue by promoting an increase in drainage
- Provides a moist wound environment, increasing epithelization, drawing the wound together
The Ulcer Is Unloaded, Infection Is Controlled, Debridements Are Ongoing And The V.A.C. Is Working... ...What Wound Dressing Products Are Indicated?
Wound Dressing Goals

- Create an optimal healing environment for the body to heal itself
- Do not compromise healing
- Prepare the wound for healing by reducing exudate, MMP’s and resolving infection
- Heal the wound by introducing factors that the wound is deficient in
Pairing The Wound With The Correct Dressing

- Dry wound > needs hydration > hydrogel dressing
- Minimal drainage > maintain > hydrogel or moisture hydrocolloid
- Heavy drainage > absorption > alginates, hydrofiber foam
- Ulcer margin > protect from > zinc oxide topical maceration cream
- Infected wound > decrease bacterial proliferation > cadaxamer iodine silver dressing honey
- Undermining, tunneling > fill in > alginate, hydrofiber rope drain
- Chronic wound (over 2 months) > growth factors, biologics
- Lowest layer must have a non-adherent dressing > adaptic, Owens silk, hydrogel
Preparing The Wound For Healing - the first 2-3 weeks...

Minimally Draining Wound

- Hydrogels
- Grade II wound, partial or full thickness
- Will not allow adherence to the wound

Hydrocolloids (duoderm) stage II partial or full thickness

Heavy Drainage

- Ca, Ag alginate, Grade II, partial or full thickness
- resorbs excess exudate
- good for packing in area of tunneling, undermining

- Hydrofiber, grade II partial or full thickness
- promotes autolytic debridement
-the first 2 – 3 weeks....

**Infected Wound**

- Cadaxamer iodine
  - grade III full thickness
  - Iodoflex, Iodosorb, putty – like, easily pack into wound
  - absorbs drainage
  - decreases bacterial bio-burden (MRSA, staph, strep, pseudomonas)
- Medihoney
  - centuries old technology
  - antibacterial spectrum similar as cadaxamer iodine
  - autolytic debridement
- Silvercel, Prisma, Acticoat 7, Aquacel Ag dressings
  - grade III full thickness
  - broad spectrum bactericidal
- do not use H2O2, bleach, betadine, alcohol, hibiclen as they are cytotoxic and create an excessively dry wound bed
Preparing The Wound

- Silvercel Antimicrobial Alginate Dressing
  - Effective against over 150 clinically relevant strains of staphylococcus including MRSA, pseudomonas, E. coli, strep, viruses and fungi
  - Alginate forms a gel to absorb moderate to large amounts of exudate
Preparing The Wound

- Promogran Prisma Matrix Dressing
  - grade II, or for contaminated or colonized grade III wounds
  - removes destructive components in wound fluid
  - contains low levels of silver for bactericidal effect
  - provides collagen for a biodegradable matrix for cell and capillary growth
  - creates an environment that promotes granulation tissue, epithelialization and wound healing
Healing The Wound....

- Eliminate the factors that contribute to wound chronicity (high levels of metalloprotease, elastase leading to extracellular matrix protein degradation and reduced new tissue growth)
- Provide the wound with the factors it is deficient in for healing (growth factors, fibroblast cell proliferation and collagen production)
...Healing The Wound

- Promogran Matrix Wound Dressing
  - grade II partial or full thickness
  - binds and inactivates harmful matrix metalloproteases
  - provides fibroblast proliferation and growth of collagen
  - controls chronic wound fluid
Healing The Wound

Integra
- Grade II partial or full thickness ulcer of any type
- used with larger diameter lesions
- a bi-layer dressing that has epidermal and dermal components
- can be used in conjunction with V.A.C.

Hydrofera Blue
- Grade II or III, all types of partial or full thickness wounds except 3rd degree burn
- highly absorbent polyvinyl alcohol sponge complexed with methylene blue and gentian violet for antibacterial effect (even MRSA)
- absorbs 25X it’s weight in exudate volume
- pack or line recesses, large voids
- moisten with saline to activate
Oasis Wound Matrix

- Non-infected full thickness venous, pressure or diabetic stalled ulcer (over 4 weeks failed standard care)
- Derived from porcine small intestine submucosa
- Has all necessary dermal components to produce wound matrix (collagen, elastin, glycosaminoglycans, glycoproteins, proteoglycans)
- Replaces missing extracellular matrix and growth factors, develops scaffold for cell adherence
- Combine with surgical debridement, will granulate over bone or tendon
- Weekly application, combine with alginate or hydrogel depending upon wound moisture

Fenestrations in Oasis allow for exudate transfer
# Healing The Wound.... Specifically Engineered Wound Dressings

<table>
<thead>
<tr>
<th>Autologous Growth Factors</th>
<th>Recombinant Growth Factor</th>
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<tbody>
<tr>
<td>• autologous platelet rich plasma</td>
<td>• Regranex gel (becaplermin)</td>
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<tr>
<td>• produced by drawing blood from the patient</td>
<td>• diabetic foot ulcers</td>
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<tr>
<td>• extracting plasma which contains platelets</td>
<td>• must clear infection first with silver based dressings, systemic antibiotics</td>
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<tr>
<td></td>
<td>• contains PDGF platelet derived growth factor</td>
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<td>• communicates with a wide variety of cell types, stimulating granulation tissue and angiogenesis</td>
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Healing The Wound.... Engineered Dermal Substitute

**Apligraf**
- Grade II, full thickness wound,
- tissue engineered skin with cultured keratinocytes and fibroblasts
- venous leg and diabetic foot ulcers
- non – infected
- may need more than one application pending wound progress

**Dermagraft**
- Grade II, full thickness
- tissue engineered skin with fibroblast/vicryl mesh
- weekly application
- non – infected
- diabetic foot ulceration
In Conclusion...

- There are four basic types of skin ulceration, each requiring a specific approach to healing.
- Contributing medical morbidities must be under control for healing to occur.
- Vascular status must be intact for healing.
- There are multiple different methods for wound care debridement.
- Wound appropriate debridement is critical in reducing bio-burden on the path to healing.
- Off loading is both necessary for healing and also for reduced recurrence.
- Vacuum assisted closure can dramatically accelerate healing.
- Various wound products are available, each with specific characteristics matched to the type of wound.
Acknowledgements

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