Onychocryptosis

- **Treatment**
  - Education (hygiene, debridement, Ect.)
  - Infection?
  - Granulation Tissue
  - Remove offending nail (total vs partial avulsion)
  - Matrixectomy (surgical vs chemical)
  - After care

Paronychia

- **Infection or inflammation around the nail complex**

- **Causes**
  - Trauma
  - Onychocryptosis
  - Onychomycosis
  - Hyperthyroidism
  - Contact dermatitis
  - Infection (Staph or Strep)

- **Treatment**
  - Diagnose and treat cause
  - Infection (Empiric vs Cultures)

Onychomycosis

- **Clinical Patterns**
  - Distal subungual

Onychomycosis

- **Clinical Patterns**
  - Superficial white
  - Proximal subungual

Onychomycosis

- **Treatment**
  - No treatment!!
  - Topical (Ciclopirox 8%) 
  - Oral antifungals

- **Oral antifungals**
  - Terbinafine: Best therapeutic profile
  - Itraconazole
  - Fluconazole

Oral Antifungals—Confirm Diagnosis First

- **Terbinafine**: 250 mg OD for 12 weeks/90 days
  - Drug interactions: warfarin
  - Risks: abnormal LFT’s, loss of taste 2.7% (reversible)

- **Itraconazole**: 200 mg BID with food, 1 week per month, x3
  - Drug interactions: warfarin, cyclosporine,Lovastatin, phenytoin, viagra, xanax, plendil, versed
  - Risks: abnormal LFT’s, drug interactions

- **Oral Antifungals**
  - Reserve in nail approximately 6 months after therapy ends
  - Hallux takes 12-16 months for growth out
  - 35% cure rate: 60% clinical cure
  - Up to 90% clinical cure with addition of a topical
Nail Tumors

- **Digital Mucinous Cyst**
  - Recurrence common
  - Benign
  - Treatment: drainage, cortisone, excision
- **Malignant Melanoma**
  - Usually subungual
  - Hutchinson’s sign
  - All races: Japanese predisposed
  - Nail dystrophy
- **Squamous Cell Carcinoma**
  - Rough, keratotic
  - Periungual
  - Hands/feet
  - Surgical removal

Nail Tumors

- **Subungual Fibroma**

Melanonychia

- **Longitudinal hyperpigmented band/streak**
- **Hyperproductive melanocytes of the nail matrix**
- **Can be accentuated by chemotherapy, medications**
- **Usually multiple nails involved (hands/feet)**
- **Differential diagnosis**
  - Onychomycosis
  - Trauma/subungual hemorrhage
  - Malignant melanoma
  - When in doubt biopsy
Nail Changes in Psoriasis
- Nail Bed
  - Onycholysis and subungual debris
- Nail Plate
  - Dystrophic crumbling nail
- Nail Matrix
  - Pitting
- DIPJ psoriatic arthritis

Nail Changes in Systemic Disease
- Terry’s Nails
  - White nail with red rim
  - Liver disease
- 1/2 and 1/2 Nails
  - Proximal nail white and distal nail normal
  - Renal disease
- Beau’s Line
  - Transverse furrow
  - Grooves out
  - Local or systemic trauma

Beau’s Line

Terry’s Nails

1/2 and 1/2 Nails
The initial evaluation of the diabetic foot ulcer must be comprehensive and systematic to ascertain the parameters that might have led to its onset as well as determine the presence of factors that can impair wound healing. Critical in this regard are assessments for vascular perfusion (ischemia), infection/osteomyelitis, and neuropathy.

As previously discussed, a thorough vascular evaluation must be performed; this includes palpation of pulses, clinical evaluation of capillary filling time, venous filling time, pallor on elevation, and dependent rubor (283). If pulses are not palpable or if clinical findings suggest ischemia, noninvasive arterial evaluation (eg, segmental Doppler pressures with waveforms, ankle brachial indices, toe pressures, TcPO2 measurements and vascular surgical consultation are warranted. When required, these physiologic and anatomic data can be supplemented with the use of magnetic resonance angiography or CT angiography (CTA) and subsequent use of arteriography with digital subtraction angiography (DSA) as necessary.

Description of the ulcer characteristics on presentation is essential for the mapping of the ulcer’s progress during treatment. While some characteristics are more important than others, they all have prognostic value during management. The presumed etiology of the ulcer (i.e, chemical vs mechanical) and character of the lesion (neuropathic, ischemic, or neuroischemic) should be determined. The evaluation should also describe the size and depth of the ulcer as well as the margins, base, and geographic location on the extremity or foot. All but the most superficial ulcers should be examined with a blunt, sterile probe. The description should note whether the sterile probe detects sinus tract formation, undermining of the ulcer margins, or dissection of the ulcer into tendon sheaths, bone, or joints. A positive probe to bone (PTB) finding is highly predictive of osteomyelitis, although the frequency of false-negative tests reduces its sensitivity.

Perhaps most importantly, the positive predictive value for PTB falls off significantly when the prevalence of osteomyelitis decreases. The existence and character of odor or exudate should be noted. Cultures may be necessary when signs of inflammation are present. Generally, clinically uninfected ulcers without inflammation should not be cultured. Current recommendations for culture and sensitivity include thorough surgical preparation of the wound site with curettage of the wound base for specimen or with aspiration of abscess material.

Wound Healing

Phase 1- Inflammatory Phase Substrate or Lag Phase: This stage lasts 3-4 days and has 3 parts, vascular, hemostatic and cellular.

a. Hemostasis is obtained via active vasoconstriction of blood vessels damaged in the wound. Aggregation of platelets also leads to the formation of a hemostatic plug.

b. Platelet adhesion is in part stimulated by exposure of the platelets to the proline and hydroxyproline matrix of mature collagen and other connective tissue components exposed by the injury.

Muscle wasting occurs. The plantar fat pad becomes displaced and the metatarsal heads become more prominent. Limited joint mobility occurs and contributes to the potential for toe and foot injury. If Charcot foot is present, there are severe bone and joint changes and the foot is swollen and warm to the touch.

c. Once platelets are exposed to and adhere to the connective tissue matrix, the platelets are activated. This can only occur in the presence of von Willebrand components of factor VIII which is released from adjacent endothelial cells.
Activation involves the release of ADP from the platelet. The ADP stimulates other platelets to stick to one another platelet aggregation.
d. Platelets store calcium and 5-hydroxytryptamine in intracytoplasmic granules as well as many other growth factors. These are released upon adhesion and promote further platelet aggregation and vasoconstriction. This process is termed “degranulation”
e. Platelet stimulation results in activation of phospholipase and hydrolyzed membrane lipids and the liberation of arachidonic acid. Arachidonic acid is converted by platelet cyclo-oxygenase into thromboxane A2. Thromboxane A2 further stimulates platelet aggregation and is also a potent vasoconstrictor.
f. Contractile protein in the platelet, thrombosthenin, promotes clot retraction. Clot retraction will not occur unless platelets are present.
g. Coagulation occurs due to the activation of clotting factors.
i. Intrinsic system
 ii. Extrinsic system
The end result is the activation of factor X which then converts prothrombin to thrombin. Thrombin then converts fibrinogen to fibrin monomers, which polymerize to form a fibrin clot. Fibrin besides promoting hemostasis, provides a scaffolding for the ingrowth of cells at a later stage.
h. Platelets release a number of other factors at this point which promote wound healing. These include:
i. Proteolytic enzymes activate the complement system. Also released is 12-HPETE which in turn stimulates the release of leukotriene B4 an important chemotaxic agent.
 ii. Various platelet derived growth factors which promote various components of wound healing. (See below)
h. Other substances in the plasma increase vascular permeability. Histamine is released by mast cells. Histamine increases vascular permeability by causing contraction of endothelial cells and uncovering gaps between the cells. Histamine is also a powerful vasodilator. Serotonin released from the platelet and kinins made from plasma alpha globulins at the site of injury, also increase vascular permeability.
i. Neutrophils, attracted by the chemotaxic factors arrive in the wound about 6 hours after the injury. They reach their highest numbers at 1-2 days post injury. If no infection is present, their numbers decline after this. Neutrophils are responsible for wound debridement through the release of collagenolytic and fibrinolytic enzymes. Additionally, neutrophils ingest bacteria.
j. Lymphocytes reach their maximum number in the wound at day 6. The most important role of the lymphocyte is the synthesis of lymphokines. Two of the best known lymphokines are the migration inhibition factor (MIF) and macrophage activation factor (MAF). MIF attracts macrophages to the wound and MAF activates them.
k. The macrophages attracted to and activated in the wound are actually derived from monocytes in the blood. They are the most important inflammatory cells involved in wound healing for the following reasons:
i. They are the only cells able to tolerate the low oxygen tensions at the wound edge.
 ii. They appear in the wound during the first 5 days and have a long life span (7-10 days).
 iii. Wound healing is severely inhibited in the absence of monocytes.
 iv. They process and present antigens to the lymphocytes to initiate immune response.
l. Migratory fibroblasts originate from mesenchymal cells near the wound edge. These cells become bound to the fibrin laid down in the wound and proliferate. They then produce glycoproteins. Collagen synthesis begins on the 5th day post injury and lasts 2-4 weeks.
m. Eosinophil concentration reaches a peak in the injured area between days 7-14. They may be associated with collagen remodeling and synthesis occurring at the same
time. There are very few and their role is unclear. 
n. Fibronectin is a glycoprotein produced by 
fibroblasts, endothelial cells and hepatocytes. Among the functions attributed to fibronectin are:

I. Fibronectin coats macrophages, aiding in opsonization and phagocytosis.

ii. Fibronectin is found on the surface of fibroblasts, where it may aid on the adhesion of these cells to the extracellular matrix.

iii. Fibronectin cross-links with collagen and glycosaminoglycans. This results in increased adhesion of epidermal cells and endothelial cells to the dermis.

iv. The matrix formed by fibroblasts and fibronectin creates a framework over which epidermal cells may migrate.

Phase 2- The Proliferative Phase (Migratory/Lag Phase): This stage lasts from 5-20 days and has three parts: epidermal regeneration, neoangiogenesis, and collagen synthesis.

a. Epidermal regeneration:

i. The cells at the wound edges flatten out and develop pseudopods (extensions of their cytoplasm), then migrate across the wound, only migrating over viable tissue, at a rate directly proportional to the oxygen tension of the tissues which is highest under hyperbaric conditions. The aforementioned fibrin-fibronectin network serves as a framework over which this migration occurs.

ii. Intracellular contractile filaments (actin) develop at the periphery of the migrating cells. These filaments align themselves with the fibrinectin strands in the extracellular matrix. The interaction of these strands actually pulls the epithelial cells along.

iii. Other changes occur: the basement membrane under the epidermal cells changes; the epidermal cells themselves elongate in the direction of the wound defect; mitotic activity of the epidermal cells dramatically increases, and the division and movement of epidermal cells may be under the direction of epidermal growth factor (EGF).

b. Neoangiogenesis: The formation of new capillary buds from blood vessels near the wound occurs at the same time as the migration of the epidermis. The development of capillaries towards the center of the wound may be under the influence of growth factors released by macrophages. As oxygen tension increases with the opening of new vascular channels, these growth factors are inhibited and capillary growth slows and then stops.

c. Collagen Synthesis:

i. Within the injured dermis, fibroblasts (surgeon's cell) begin to appear at the end of the inflammatory process, and adhere to the dermal collagen and fibrin. As the capillary structure returns to the wound and oxygen tension increases, fibroblast replication slows. As oxygen tension further increases, fibroblasts begin synthesizing collagen.

ii. There are at least 5 types of collagen.

iii. Collagen production by the fibroblasts are under control of at least 5 growth factors.

iv. Collagen at this point represents 50% of the scar.

v. The amount of collagen in the healing wound reaches a maximum at two to three weeks post injury. Remodeling now begins. Phase 3-The Remodeling Phase (Maturation)- Can last up to a year. At two weeks post injury, a wound has regained only 35% of its tensile strength. By one month this has increased to 40-50%. A number of processes occur during the remodeling phase:

a. The entire remodeling process is really an equilibrium between enzymatic processes lysing and resorbing old collagen and forming new collagen.

b. Wound contraction is part of this remodeling stage of healing. Contraction progresses at 0.6 to 0.7 mm/day independent of the wound size, but certain shapes heal faster. Round wounds do not contract as quickly as rectangular wounds.

Factors That Interfere With Wound Healing

1. Age: Growth rate and multiplication of fibroblasts decrease with age.
2. Inadequate Perfusion: Inadequate perfusion of
results in a decrease in oxygen delivery to the wound, thereby impairing healing.

3. Infection: Infection leads to tissue destruction and edema, both of which interfere with the healing process.

4. Edema: Interferes with tissue perfusion and leads to tissue destruction.

5. Poor Nutrition: Protein depletion results in alterations in collagen synthesis and cross linking.

6. Vitamin and Trace Element Deficiencies:
   a. Vitamin A deficiency can interfere with wound healing, and may reverse wound healing problems associated with steroids.
   b. Vitamin C deficiencies lead to scurvy, a disease associated with the failure of collagen synthesis.

7. Steroid and Cytotoxic Medications:
   a. Steroids slow protein synthesis when given exogenously. Steroids interfere with capillary budding, slow fibroblast proliferation as well as the rate of epithelialization.
   b. Cytotoxic drugs commonly used in chemotherapy, inhibit cellular proliferation. In general, wound healing is slowed but not prevented.

8. Radiation: Microvascular changes occurring after tissue is exposed to radiation at therapeutic doses will lead to perfusion problems if that tissue is later injured. All cell types involved in healing may be adversely affected by radiation. Malignant change may also occur.

9. Diseases Which are Associated With or Predispose to Chronic Wounds:
   a. Diabetes Mellitus:
      i. Deposits in the arteries interfere with tissue perfusion.
      ii. Diabetic neuropathy leads to reduced sensation and gait abnormality. Metabolic problems lead to a reduction in nutrients available for wound healing.
      iv. Impaired phagocytosis seen as part of the disease spectrum on diabetics leads to an increase in bacterial infections and subsequent tissue destruction.
   b. Venous Stasis: Poor venous return leads to an increase in tissue pressure. The increase in tissue pressure results in underperfusion of the skin and wounds, as well as accumulation of inhibitory metabolites.

   c. Collagen Vascular Disease: These diseases have an autoimmune basis and result in capillary damage leading to poor tissue perfusion and hypoxia, and immune response to other cells or cell constituents.

   Treatment of Non-healing Wounds:
   a. Debridement of necrotic tissue
   b. Control of infection
   c. Control of diabetes mellitus
   d. Nutritional support
   e. Avoidance of trauma
   f. Aggressive intermittent compression and elevation to eliminate limb edema
   g. Tapering of steroids (paradoxically, topical steroids applied to wound in patients with collagen vascular disease may control the vasculitis and actually stimulate wound healing)
   h. Revascularization of an ischemic wound through angioplasty or reconstructive vascular surgery
   i. Use of hyperbaric oxygen
   j. Plastic reconstructive surgery
   k. Application of growth factors (still experimental)

Growth Factors in Wound Repair
With the production of a platelet derived wound healing formula (PDWHF) known as PROCUREN, a mixture of 5 platelet-produced growth factors, angiogenesis and other aspects of wound healing are stimulated. The 5 growth factors are
1. Platelet derived growth factor (PDGF)
2. Platelet derived angiogenesis factor (PDAF)
3. Platelet derived epidermal growth factor (PDEGF)
4. Transforming growth factor (TGFB)
5. Platelet factor 4 (PF-4)

Classification of Ulcers
Appropriate classification of the foot wound is based on a thorough assessment.

Classification should facilitate treatment and be generally predictive of expected outcomes. Several systems of ulcer classification are currently in use in the US and abroad to describe these lesions and communicate severity. Perhaps the easiest system is to classify lesions as neuropathic, ischemic, or...
neuroischemic, with descriptors of wound size, depth, and infection. Regardless of which system is used, the clinician must be able to easily categorize the wound and, once classified, the ensuing treatment should be directed by the underlying severity of pathology.
Debridement. Debridement of necrotic tissue is an integral component in the treatment of chronic wounds since they will not heal in the presence of unviable tissue, debris, or critical colonization (314, 315). Undermined tissue or closed wound spaces will otherwise harbor bacterial growth. Debridement serves various functions: removal of necrotic tissue and callus; reduction of pressure; evaluation of the wound bed; evaluation of tracking and tunneling; and reduction of bacterial burden (318, 319). Debridement facilitates drainage and stimulates healing. However, debridement may be contraindicated in arterial ulcers. Additionally, except in avascular cases, adequate debridement must always precede the application of topical wound healing agents, dressings, or wound closure procedures. Of the five types of debridement (surgical, enzymatic, autolytic, mechanical, biological), only surgical debridement has been proven to be efficacious in clinical trials.

Surgical debridement. Surgical debridement is the cornerstone of management of diabetic foot ulcers. Thorough sharp debridement of all nonviable soft tissue and bone from the open wound is accomplished primarily with a scalpel, tissue nippers, curettes, and curved scissors. Excision of necrotic tissue extends as deeply and proximally as necessary until healthy, bleeding soft tissue and bone are encountered. Any callus tissue surrounding the ulcer must also be removed. The main purpose of surgical debridement is to turn a chronic ulcer into an acute, healing wound. A diabetic ulcer associated with a deep abscess requires hospital admission and immediate incision and drainage. Joint resection or partial amputation of the foot is necessary if osteomyelitis, joint infection, or gangrene are present.

Necrotic tissue removed on a regular basis can expedite the rate at which a wound heals and has been shown to increase the probability of attaining full secondary closure. Less frequent surgical debridement can reduce the rate of wound healing and secondarily increase the risk of infection. Surgical debridement is repeated as often as needed if new necrotic tissue continues to form. Frequent debridement, referred to as “maintenance debridement,” is commonly required. While the terms surgical debridement and sharp debridement are often used synonymously, some clinicians refer to surgical debridement as that done in an operating room whereas sharp debridement is performed in a clinic setting.

Enzymatic debridement. A highly selective method, enzymatic debridement consists of the application of exogenous proteolytic enzymes manufactured specifically for wound debridement. Various enzymes have been developed, including bacterial collagenase, plant derived papain/urea, fibrinolysin/DNAse, trypsin, streptokinase-streptodornase combination; only the first three products are widely available commercially. Collagenases are enzymes that are isolated from Clostridium histolyticum. These display high specificity for the major collagen types (I and II), but they not active against keratin, fat, or fibrin. Papain, obtained from the papaya plant, is effective in the breakdown of fibrinous material and necrotic tissue. When combined with urea, it denatures nonviable protein matter. The enzymatic compounds are inactivated by hydrogen peroxide, alcohol, and heavy metals, including silver, lead, and mercury. One study found that wounds treated with papain-urea developed granulation tissue faster than those treated with collagenase, but no contrasts between rates of complete wound healing were made. Autolytic debridement. Autolytic debridement occurs naturally in a healthy, moist wound environment when arterial perfusion and venous drainage are maintained.

Mechanical debridement. A nonselective, physical method of removing necrotic tissue,
mechanical debridement may include wet-to-dry dressings and high-pressure irrigation or pulsed lavage and hydrotherapy. Wet-to-dry is one of the most commonly prescribed and overused methods of debridement in acute care settings. Hydrotherapy in the form of whirlpool may remove surface skin, bacteria, wound exudates, and debris. There may be justification in the early stages of a wound for the use of this technique, but it is detrimental to friable granulation tissue.

Biological (larval) therapy. Larval therapy utilizes the sterile form of the Lucilia sericata blowfly for the debridement of necrotic and infected wounds. Maggots secrete a powerful proteolytic enzyme that liquefies necrotic tissue. It has been noted that wound odor and bacterial count, including methicillin-resistant Staphylococcus aureus, diminish significantly (343) with larval therapy. Larval therapy seems to be beneficial, but there is paucity of controlled studies to support its routine use in the diabetic foot wound.

Moisture Balance. One of the major breakthroughs in wound management over the past 50 years was the demonstration that moisture accelerates re-epithelialization in a wound. Tissue moisture balance is a term used to convey the importance of keeping wounds moist and free of excess fluids. A moist wound environment promotes granulation and autolytic processes. Effective management of chronic wound fluids is an essential part of wound bed preparation; it also helps in addressing the issues of cellular dysfunction and biochemical imbalance.
Wound Dressings. Wound dressings can be categorized as passive, active, or interactive. Passive dressings primarily provide a protective function. Active and interactive dressings and therapies are capable of modifying a wound’s physiology by stimulating cellular activity and growth factor release. A wide variety of wound care products is available; a brief listing of dressings and topical agents is presented in Table 8.

Inflammation and Infection. In chronic wounds, inflammation persists due to recurrent tissue trauma and the presence of contaminants. Nonhealing wounds can become “stuck” in the inflammatory phase of healing, increasing cytokine response with subsequent elevated protease levels and impaired growth factor activity. The presence of infection must be ascertained and identified as local (soft tissue or osseous), ascending, and/or systemic. In diabetes, where the host response is reduced and normal signs of infection (i.e., fever, pain, leukocytosis) may be absent, other factors such as elevated glucose levels can be helpful as an indicator of infection. It is important to obtain specimens for culture prior to antimicrobial therapy.

Tissue specimens collected by curettage or biopsy are preferred, because they provide more accurate results than superficial swabs.
<table>
<thead>
<tr>
<th>Table 8</th>
<th>Wound Care Products</th>
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<tbody>
<tr>
<td><strong>Category</strong></td>
<td><strong>Indications</strong></td>
</tr>
<tr>
<td><strong>Dressings</strong></td>
<td></td>
</tr>
<tr>
<td>Gauze pads (312, 356, 362)</td>
<td>- Low to heavily draining wounds or surgical wounds</td>
</tr>
<tr>
<td>- sterile gauze</td>
<td>- Wet to dry debridement</td>
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<tr>
<td>- sterile cotton</td>
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<tr>
<td>Transparent films (312, 352)</td>
<td>- Dry to minimally draining wounds</td>
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<tr>
<td>- polyurethane film with drainage cohesive layer, semipermeable</td>
<td>- Promote tissue hydration</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Hydrogels (312, 352)</td>
<td>- Gel, sheet, gauze (35% water or glycerin)</td>
</tr>
<tr>
<td></td>
<td>- Dry to minimally draining wounds</td>
</tr>
<tr>
<td>Foam (312, 352)</td>
<td>- Moderate, large exudate</td>
</tr>
<tr>
<td>- polyurethane foam (open cell, absorbent)</td>
<td>- Clean wound surface</td>
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<tr>
<td></td>
<td>- Super absorbent and conformable to topography</td>
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<tr>
<td>Hydrocolloids (312, 352)</td>
<td>- Wafer with adhesion, (carboxymethylcellulose, pectin, gelatin) impermeable to oxygen</td>
</tr>
<tr>
<td></td>
<td>- Low to moderate drainage</td>
</tr>
<tr>
<td>Calcium alginites (312, 352)</td>
<td>- Filter pad derived from seaweed (may be combined with silver or collagen)</td>
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<tr>
<td></td>
<td></td>
</tr>
<tr>
<td>Collagen dressings (302, 312, 325, 352)</td>
<td>- Particles or composite pads with collagen component (derived from bovine collagen)</td>
</tr>
<tr>
<td></td>
<td>- Low to heavily draining wounds</td>
</tr>
<tr>
<td>Antimicrobial dressings (312, 334, 352)</td>
<td>- Infected or clean wounds to prevent infection</td>
</tr>
<tr>
<td>- contain silver, iodine in various forms preparations (eg, cadexomer iodine)</td>
<td></td>
</tr>
</tbody>
</table>

| **Topical Therapies / Agents** | | |
| Saline (302, 363) | - Clean or infected wounds | - Undefined |
| Amorphous hydrogels Skin cleansers | - Isotonic solutions for irrigation, hydrating dressings | | |
| Detergents/Antiseptics (302, 352) | - Contaminated or infected wounds | - Healthy granulating wounds |
| - povidone-iodine, | | |
| - chlorhexidine | | |
| - chloroxylenol | | |
| - hypochlorite | | |
| - benzalkonium chloride | | |
| Topical Antibiotics (302, 320, 352) | - Contaminated or infected wounds | - Healthy granulating wounds |
| - bacitracin, neomycin | | |
| - mupirocin, polymyxin B | | |
| - silver sulfadiazine | | |
| - mafenide (creams, ointments) | | |
| Enzymes (302, 312, 319, 328, 332-335) | - Necrotic tissue | - Healthy or infected wounds |
| - collagenase | | |
| - papain-urea | | |
| - Enzymes | - Eosinophilic wounds | | |
Pressure Relief/Off-loading. The reduction of pressure to the diabetic foot ulcer is essential to treatment. Proper off-loading and pressure reduction prevents further trauma and promotes healing. This is particularly important in the diabetic patient with decreased or absent sensation in the lower extremities (50, 418). Furthermore, recent studies provide evidence that minor trauma (eg, repetitive stress, shoe pressure) plays a major role in the causal pathway to ulceration.

The choice of off-loading modality should be determined by the patient’s physical characteristics and ability to comply with treatment as well as by the location and severity of the ulcer. Various health care centers prefer specific initial modalities, but frequently clinicians must alternate treatments based on the clinical progress of the wound. Even as simple a method as a felted foam aperture pad has been found to be effective in removing pressure and promoting healing of foot ulcers. A study published in 2001 noted that use of a total contact cast (TCC) healed a higher portion of wounds in a shorter time than a half shoe or removable cast walker (RCW).

More recently, investigators compared TCC use with that of a removable cast walker that was rendered irremovable (iTCC) by circumferential wrapping of an RCW with a single strip of fiberglass casting material. They concluded that the latter may be equally efficacious, faster to place, easier to use, and less expensive than TCC in the treatment of diabetic neuropathic plantar foot ulcers. The findings of this study and another study also suggest that modification of the RCW into an irremovable device may improve patient compliance, thereby increasing the proportion of healed ulcers and the rate of healing of diabetic neuropathic wounds. Regardless of the modality selected, no patient should return to an unmodified shoe until complete healing of the ulcer has occurred. Furthermore, any shoe that resulted in the formation of an ulcer should never again be worn by the patient.

Methods For Offloading Diabetic Wounds:

- Total Non-weight bearing: Crutches, wheelchair, bedrest
- Total Contact Casting
- Foot Casts or Boot (CAM Boot)
- Removable walking brace with rocker sole
- Total Contact Orthoses
- Patella Tendon Bearing Braces
- Half Shoes or Wedge Shoes
- Healing Sandal (Surgical Shoe)
- Accommodative Dressings (Felt, Foam, etc)
- Shoe Cutout
Assessment of Diabetic Foot Infections

When evaluating the patient with a diabetic foot infection, a problem-directed history and physical examination should be obtained. A systematic approach to the complete assessment of these patients is required, since there is evidence that they are often inadequately evaluated, even when hospitalized. The past medical history should assess the patient’s neurologic, cardiovascular, renal, and dermatologic status. Use of current medications as well as previous antibiotics may interfere with planned treatments or indicate that standard treatments will likely be ineffective. Pain should be considered an unreliable symptom in individuals with peripheral neuropathy.

The patient should be questioned regarding previous ulcerations, infections, trauma, and surgeries at the present site or at any other past location of infection. Constitutional symptoms (e.g., nausea, malaise, fatigue, vomiting, fever, chills) are important clinical clues when presented with an infected diabetic foot. Severe infection or sepsis must be considered when these symptoms are present. However, in about 50% of diabetic patients presenting with significant infection, systemic signs (fever and leukocytosis) are absent. Frequently, the only indication of infection is unexplained or recalcitrant hyperglycemia.

Laboratory testing might include a CBC with or without differential, blood cultures, glycosylated hemoglobin, fasting blood sugar, sedimentation rate, and urinalysis. Other tests should be performed as indicated by the patient’s condition or comorbidities.

The history of the wound or infection should include the onset, duration, and appearance before infection of the area. Depth or size of the ulcer, amount of drainage, swelling, color, odor, and extent of infection should be evaluated. The infection or ulcer should be probed to determine the presence of bone or joint involvement, sinus tracts, or extension into tendon sheaths. The latter are common routes for the spread of infection both distally and proximally. Reliable aerobic and anaerobic cultures should be obtained from purulent drainage or curettage of the ulcer base, since studies have shown good concordance with the true pathogen. Simple swab cultures of an ulcer surface are generally not advisable because they tend to be unreliable, especially in the presence of osteomyelitis or sinus tracts.

For patients with clinically uninfected or noninfamed neuropathic ulcers, the role of antibiotic therapy is still in question. Therefore, in these instances, wound culture is probably unnecessary. If osteomyelitis is suspected, bone cultures are necessary to make the definitive diagnosis and isolate the true pathogen. However, this must be balanced against the potential for contaminating non-infected bone in the presence of an active soft tissue infection. Intraoperative frozen section is also useful in assessing for deep infection. The presence of more than 5 to 10 neutrophils per high power field is suggestive of acute infection.

The majority of wounds are caused by Staphylococcus aureus, beta-hemolytic streptococci, and other gram positive cocci. Although community acquired cases of resistant bacterial infections have been reported, patients who have been previously hospitalized with an open wound are more likely to develop an infection from resistant bacteria such as methicillin-resistant S aureus (MRSA) and vancomycin-resistant enterococci (VRE). Chronic wounds may develop a more complex assortment of bacteria, including gram negative rods, obligate anaerobes, Pseudomonas aeruginosa, and enterococci.

Imaging studies are also important in the overall assessment of diabetic foot infections,
notwithstanding their hortcomings. Plain film x-rays may indicate the presence of bony erosions and/or gas in the soft tissues. It should be noted that the demonstration of osteomyelitis by plain radiographs lags the onset of bone involvement by 10 to 14 days. Radionucleotide bone scans such as Tc-99 may demonstrate abnormal uptake of the radionucleotide before changes are visible on radiographs. This may be less specific in patients with peripheral neuropathy or with any preexisting osseous condition that causes increased bone turnover (e.g., surgery, fracture, neuropathic arthropathy). A combination of scans such as the Tc-99m and an indium-labeled leukocyte scan or the Tc-99m HMPAO labeled leukocyte scan may aid the clinician in differentiating Charcot arthropathy and osteomyelitis with greater accuracy. MRI has generally supplanted the CT scan in the early diagnosis of osteomyelitis, due to its higher tissue contrast and ability to detect both soft tissue and marrow inflammation. Additionally, MRI can be used to follow the resolution of infection or as an aid in surgical planning. However, none of these imaging modalities are 100% sensitive and specific for diagnosing or ruling out bone infection. Furthermore, these tests are expensive and may not be readily available. Appropriate clinical assessment and diagnostic acumen should therefore remain the guiding principles to management.

Treatment of Diabetic Foot Infections

Diabetic foot infections should be managed through a multidisciplinary team approach utilizing appropriate consultations. Hospitalization of patients with limb-threatening infections is mandatory. All diabetic foot infections must be monitored closely. Equally important for the best possible outcome are patient compliance and education, especially in outpatient management.

Non-Limb Threatening Infections - Treatment of diabetic foot infections is guided by the severity of the infection. As previously discussed, non limb-threatening infections involve superficial ulcerations without significant ischemia and they do not involve bone or joint. Typically, cellulitis does not extend 2 cm beyond the ulcer margins and there is an absence of systemic symptoms (e.g. fever, chills, nausea, vomiting).

These less severe infections that frequently complicate diabetic foot ulcers, may be initially treated in an outpatient setting. Many mild or moderate infections are monomicrobial, with S aureus, S epidermidis, and streptococci the most common pathogens. Reliable specimens for cultures may be obtained through curettage of the infected ulcer. In addition to the standard treatment for ulcerations (i.e., non weight bearing and dressing changes), oral antibiotic therapy is usually sufficient as initial therapy. Antimicrobial treatment should be started as soon as possible with an agent providing adequate gram positive coverage, recognizing that gram negative organisms might also be involved. All antibiotic treatments should be monitored for development of resistance. Most cases of cellulitis respond within 3 to 5 days of initiation of appropriate antibiotics. If cellulitis is slow to respond, worsens, or recurs following several days of treatment, the ulceration should be reassessed and possibly recultured. Bacteria frequently develop resistance to an antimicrobial agent, especially with prolonged therapy.

Limb Threatening Infections - By definition, limb-threatening infections are much more serious and more often acute compared with the milder non limb-threatening infections. In the PEDIS system, limb-threatening infections are classified as grade 3 or 4, depending on severity and the presence of systemic manifestations. Neuropathy often predisposes such infections to progression to an emergent situation before the patient even becomes aware of the infection’s presence. Limb-threatening infections may have life-threatening complications, especially when left...
untreated. Because of diabetes-associated immunosuppression, up to 50% of patients with limb-threatening infections may exhibit no systemic symptoms or leukocytosis. However, other patients present with evidence of systemic toxicity, including fever, chills, loss of appetite, and malaise. Such findings in diabetic patients should alert clinicians to the severity of infection. Most will not be controllable hyperglycemia despite usual therapy and loss of appetite. Limb-threatening infections are recognized as having one or more of the following findings: greater than 2 cm of cellulitis around an ulcer, lymphangiitis, soft tissue necrosis, fluctuance, odor, gangrene, osteomyelitis. When such an infection is recognized, the patient requires emergent hospital admission for appropriate intervention. Upon admission, a complete history and physical examination are undertaken. The patient’s cardiovascular, renal, and neurologic risks should be evaluated to assess for secondary complications of diabetes and associated comorbidities. A thorough foot evaluation is undertaken to determine the clinical extent of the infectious process.

Vascular status must be assessed to ensure that appropriate arterial inflow is present. If perfusion is inadequate, this should be addressed prior to definitive reconstruction to enhance healing at a more distal level. Radiographs are necessary to evaluate for evidence of osteomyelitis or soft tissue gas.

If gas is identified in the ankle or hindfoot, radiographs of the lower leg should be obtained to assess the extent of the gas formation. Blood cultures are required if clinical findings indicate septicemia. Other appropriate laboratory studies, including CBC with differential and sedimentation rate, are obtained as warranted. Glucose management must be initiated to optimize metabolic perturbations and improve leukocyte function. The patient’s nutritional and metabolic status must be assessed and properly maintained, since relatively common nutritional and metabolic impairments in these patients can adversely affect wound healing and resolution of infection.

Consultations are typically required in the risk assessment and management of these complex cases. Medical, endocrinology, cardiology, nephrology, and diabetic teaching nurse consultations are often routinely needed to optimize patient care and fully assess surgical risks. Infectious disease and vascular surgery consultations are also obtained when complex infections or significant ischemia are identified, respectively. A multi-disciplinary approach to the management of these cases has been shown to significantly improve outcomes.

Early surgical treatment of the affected site is typically necessary as an integral part of infection management. This may include simple debridement of the soft tissues, wide incision and drainage of the pedal compartments, or open amputation to eliminate extensive areas of infection. At the time of debridement, aerobic, anaerobic, and fungal tissue cultures should be obtained from the depth of the wound to provide reliability.

Although many initial drainage procedures can be performed at the bedside for neuropathic patients, most require thorough debridement in the operating room. Anesthesia for such interventions may include local, regional, or general anesthetics.

However, spinal blocks are typically avoided in patients who may be septic. Even the sickest of patients should be considered for emergent incision, drainage, and debridement procedures, because their illness in this regard is directly attributable to the infection severity. Such life-threatening infections necessitate immediate surgical attention, without delay in obtaining radiologic or medical work-up of other comorbid
conditions. Polymicrobial infection should be anticipated in these patients, with a variety of gram positive cocci, gram negative rods, and anaerobic organisms predominating.

Accordingly, empirical antibiotic therapy typically includes broad-spectrum coverage for more common isolates from each of these three categories. Fully comprehensive empiric coverage is usually unnecessary unless the infection is life threatening.

Hospital therapies are usually initiated with intravenous medications, although most oral fluoroquinolones and oral linezolid have the same bioavailability as parenteral therapy. Once wound culture results become available, the initial antimicrobial therapy may require adjustment to provide more specific coverage or provide therapy against resistant organisms causing persisting infection. Recent evidence also supports the efficacy of initial parenteral therapy followed by the appropriate oral agent in the management of these patients. If the patient develops evidence of recurrent infection while receiving antibiotic therapy, repeat cultures should be obtained to assess for superinfection. Methicillin-resistant staphylococci, which have emerged as important pathogens in chronically-treated diabetic foot ulcer patients must be detected early and treated appropriately to avoid further tissue loss or extension of infection.

The surgical wound may require repeated surgical debridement to completely eradicate infection and soft tissue necrosis. Wound care is initiated on day 1 or day 2 postsurgery and may initially involve saline gauze dressing changes. Other dressings may be used to aid in healing. Negative pressure wound therapy (V.A.C.®, KCI, San Antonio, TX) has been found particularly useful in this regard. If the wound fails to show signs of healing, the patient’s vascularity, nutritional status, infection control, and wound off-loading must be re-evaluated.

Once soft tissue infection is under control and management of any osseous infection has been initiated, consideration may be given to wound closure or definitive amputation. Restoration and maintenance of function and independence is the ultimate goal for the patient. The residual extremity requires close follow-up, regular diabetic foot exams, periodic foot care, and appropriate footwear therapy.

Osteomyelitis and joint infection, when identified by clinical assessment or imaging studies, require a sampling of bone for microbiologic and histopathologic evaluation. If the patient’s soft tissue infection is controlled, consideration may be given to stopping antibiotic therapy 24 to 48 hours presurgery to improve culture accuracy. A diagnosis of osteomyelitis requires that both culture and biopsy studies reveal positive findings, including necrosis, chronic inflammatory infiltrates, and positive isolation of bacteria.

Resection of infected bone with or without local amputation and concurrent antimicrobial therapy is the most optimal management for osteomyelitis.

Antibiotics
1. The Penicillins
   a. Penicillin G: Parent compound introduced in the 1940's
      i. Good gram(+) and weak gram(-) coverage
      ii. Fallen out of favor since many resistant strains (Staph 100% Beta lactamase producing)
      iii. 1 mg PenG = 1667 units
   b. Penicillin VK:
      i. Used in severe erysipelas and rheumatic fever prophylaxis
   c. Methicillin:
i. For PCN-ase resistant organisms
  ii. IV form only
  iii. Can cause thrombophlebitis
d. Oxacillin/Dicloxicillin/Cloxacillin/Nafcillin: (PRPs)
  i. PCN-ase resistant
  ii. Good gram(+) coverage
  iii. Oral form can cause diarrhea
  iv. Requires frequent dosing, Q4-6 hours
e. Ampicillin: Increased gram(-) coverage
  i. Not PCN-ase resistant
  ii. Used with UTI, typhoid fever and salmonella infections
  iii. Used pre-op for endocarditis prophylaxis
  iv. Used in combination with aminoglycosides for gram(-) septicemia
f. Carbenicillin: The original anti-pseudomonal penicillin
  i. Can be combined with aminoglycoside for pseudomonas infection
  ii. Not used much now since has high sodium content, hepatotoxic, neurotoxic and causes bleeding disorders
  iii. Oral form: Geopen (UTI's only)
g. Ticarcillin: A 4th generation penicillin active against pseudomonas
  i. 2-4 times more potent than carbenicillin vs pseudomonas
  ii. Has increased anaerobic activity
  h. Piperacillin: As above, gram(+) 9 (-) activity
  i. Azlocillin: As above but superior to ticarcillin/piperacillin vs pseudomonas aeruginosa
  j. Neurotoxic/Hepatotoxic
  k. Mezlocillin (Mezlin): A 4th generation penicillin with good gram(-) and anaerobic activity
  i. Can be used for Pseudomonas/B.fragilis

2. The Clavulanates
a. Amoxicillin/clavulanate (Augmentin): Adds clavulanic acid to ampicillin which inactivates the beta-lactamase enzymes:
  i. PCN-ase resistant
  ii. Spectrum of activity increased vs gram (-) to include E.Coli & Klebsiella, also good Staph and Bacteroides coverage
  iii. The oral drug of choice for cat, dog and human bites
  iv. Dosed at 250-500 Q 8h (for other than endocard prophylaxis)
b. Ticarcillin/clavulanate (Timentin): Has greater gram(-) coverage than any 4th gen. penicillin
  i. Has good gram(+) coverage and covers anaerobes well (i.e. B. fragilis)
  ii. Good drug for initial therapy for moderate diabetic foot infections
  iii. Has high sodium load/use cautiously in hypertensive-renal pt's
  iv. Dosed at 3.1 Q 6-8h (3gm ticarcillin + 100mg clavulanate)

3. The Sulbactams
a. Ampicillin/ sulbactam (Unasyn):
  i. Similar to Timentin but has much lower sodium load
  ii. Adds sulbactam, a beta-lactam inhibitor
  iii. 99% coverage against B.fragilis/not good against pseudomonas and good against enterococcus
  iv. Dosed at 1.5-3g Q 4-6h.

4. The Tazobactams
a. Piperacillin/Tazobactam (Zosyn)
  i. Similar to Timentin in coverage and spectrum
  ii. Adds Tazobactum a Beta lactam inhibitor
  iii. Has greater activity than pipericillin
  iv. Dosed @ 3.375 gm Q 6 hrs

5. The Cephalosporins
Semi-synthetic compounds derived from the mold, cephalosporum acremonium. There is a cross reactivity with penicillin allergic patients from 5-20% depending upon the source. As a whole, these antibiotics are well tolerated, nontoxic and broad spectrum. They are categorized in generations, which define their spectrum.
a. 1st generation:
   i. Keflin (cephalothin), Keflex (cephalexin), Ancef (cefaclorin), Cefadyl (cephapirin), Anspor & Velocef (cephradine), Duricef (cephadroxil)
   ii. ACTIVITY vs gram (+) cocci: S. aureus and epidermidis, Strept pyogenes and pneumonia
   iii. Activity vs gram (-): Proteus mirabilis, E. coil, Klebsiella pneumonia (PECK)
b. 2nd generation:
   i. Mandol (cefamandole),
Mefoxin (cefoxitin), Ceclor (cefaclor), Zinacef (cefuroxime), Ceftin (cefuroxime axetil), Monocid (cefonicid), Cefotan (cefotetan), Lorabid (loracarbef), Cefzil (cefpodoxil)

ii. ACTIVITY vs gram (+): is variable to Staph, still OK to Strep
iii. Activity vs gram (-): as with 1st generation (PECK) plus H. flu, Enterobacter & Neisseria (HENPECK)

c. 3rd generation:
I. Claforan (cefotaxime), Cefobid (ceperazone), Cefizox (cefizoxime), Rocephin (ceftriaxone), Fortaz (ceftazidime), Suprax (cefixime), Vantin (cefpodoxime proxetil)
ii. ACTIVITY vs gram (+): variable to both Staph and Strep
iii. Activity vs gram (-): as with 2nd generation (HENPECK) plus Serratia, Morganella, Providencia, Citrobacter and Pseudomonas

6. Other Beta-Lactams:
a. Imipenem/Cilastatin (Primaxin): Is an extremely potent antibiotic with the broadest spectrum of an available beta lactam including anaerobic coverage/ most expensive antibiotic on the market. Cilastatin is added to prevent renal hydrolysis (destruction of imipenem)
I. May be the drug of choice in severe/limb threatening diabetic infections (as initial therapy) other than clinda/genta/ampi
ii. Major therapeutic use for Gram (+) cocci and aerobic gram (-) bacilli
iii. A 3% cross sensitivity with penicillin allergic patients
Dosed at 0.5-1 gram Q 6h IV up to 4gm/day
b. Azreomam (Azactam): Is ONLY effective against gram (-) aerobes, including P. aeruginosa
I. Can be combined with clindamycin in penicillin allergic patients when gram(+) and anaerobes are suspected

7. Quinolones:
a. Ciprofloxacin (Cipro):
  i. Its main benefit is it's p.o. gram (-) coverage
  ii. Can be used for methicillin resistant staph but should be combined with rifampin (300mg BID) in the treatment of these infections
  iii. Contraindicated in its use with children as it can cause cartilage degeneration
  iv. Can be combined with clindamycin (Cleocin) or metronidazole (Flagyl) in the treatment of diabetic foot infections
v. Oral therapy for osteomyelitis when caused by Pseudomonas
vi. Rarely a first line antibiotic
b. Ofloxacin (Floxin):
  i. As with the above, but with better gram (+) coverage
  ii. Dosed for soft-tissue infections at 400 mg Q 12 h

8. Aminoglycosides:
a. Streptomycin: (used in treatment of TB)
b. Kanamycin (used as an irrigant)
c. Gentamycin: used with methylmethacrylate beads (PMMA) for osteomyelitis and in triple antibiotic therapy for serious infections
d. Potentially ototoxic in patients with renal problems
d. Tobramycin (less ototoxic than gentamycin)
e. Amikacin (reserved for serious infections against aminoglycoside resistant organisms)
f. As a group these antibiotics have well documented toxicities (ototoxicity/hepatotoxicity). g. They are essentially anti-gram negative agents, but do have gram positive coverage. When using these antibiotics it is beneficial to, have an ID consult and you should perform peak/trough serum levels as well as creatinine clearance and BUN tests (if BUN elevated increase time span between doses or lower the dose)

9. Other antibiotics:
a. Vancomycin:
  i. Indicated in penicillin allergic patients or those patients needing coverage against gram (+) organisms, including methicillin resistant Staph
  ii. It is possibly nephrotoxic and should be monitored carefully
  iii. Red neck syndrome occurs if infused too quickly (not an allergy)/severe hypotension
can result
iv. Oral form is for pseudomembranous colitis only
b. Clindamycin:
i. It is used extensively for anaerobic infections and in the penicillin allergic patient for gram (+) coverage
ii. Can cause pseudomembranous colitis
c. Tetracycline:
i. A broad spectrum antibiotic used for rocky mountain spotted fever, Lyme disease, and H. pylori infection
ii. To be avoided in children and pregnant/nursing mothers (brown teeth)
d. Metronidazole (Flagyl):
i. An amebicidal drug also with excellent anaerobic coverage
ii. Can be combined with Cipro for more complete coverage
e. Erythromycin, clarithromycin (Biaxin), azithromycin (Zithromax) Specific Antimicrobial Therapy

1. Gram (+) cocci (Penicillinase resistant): When a gram stain report is received and initial therapy is to be started prior to receiving a C & S the following should be considered due to the increasing number of betalactamase organisms: Dicloxicillin 500 mg qid p.o., Nafcillin 1 gm IV Q6h, Ancef 1gm IV Q8h, Duricef 500mg bid, Timentin 3.1gm Q6h IV and Unasyn 1.5-3.0 gm Q6h IV
2. Gram (+) cocci + penicillin allergy: Vancomycin 500mg or 1gm Q6 and Q1 2 H IV or Clindamycin 300 Q 6h po, Erythromycin 500 mg Q6h po
3. Methicillin (nafcillin/oxacillin) resistant gram (+) Staph: Vancomycin 500 mg Q 6h IV or 1 gm Q12h, Cipro 500 Q12h po + Rifampin 300 mg Q 1h po (Rifampin synergizes the anti-gram (+) effect of Cipro when in combination with it), Minocycline, Trimethoprim/sulfa
4. Gram (-): When a gram stain is received and initial therapy is to be started prior to a C & S the following should be considered: Cipro 750 mg Q 1h, Azactam 1 gm Q8h IV, Gentamicin 3-5 mg/kg _IV following a loading dose, Timentin and Fortaz 1-2 gms Q8h IV, Zosyn (tazobactam/piperacillin) 3.375 gm Q6 IV
5. Anaerobic coverage: Flagyl 500 mg Q8h po, Clindamycin 600-800 mg Q8h IV or 300 mg bid-tid po, Primaxin, Timentin, and Unasyn
6. Antipseudomonal coverage: 4th generation penicillin (in combination with another antibiotic), Fortaz 1-2 gm Q 8h, Azactam 1 gm Q8h, Gentamycin 3-5 mg/kg IV following a loading dose, Cipro 750 mg Q 12h
7. Antifungal coverage: Diflucan 100 mg od, Amphotericin B (very severe side effects), Ketoconazole (Nizoral), Griseofulvin, and Sporanox 100mg
8. Antihelminthic Coverage: Thiabendazole (Mintezole) for cutaneous larva migrans and hook worms, and Gamma Benzene Hexachloride (Kwell) for parasitic skin infestations caused by scabies.
Module 8: Charcot Neuroarthropathy

Charcot Neuroarthropathy

A Slow to Rapidly PROGRESSIVE Noninfectious Destruction of One or More Bone(s) &/or Joint(s) Associated with Neuropathy.

A DIFFICULT AND CHALLENGING DIAGNOSTIC DILEMMA

Charcot Neuroarthropathy

- Often Misdiagnosed
- Males > Females
- Occurs B/L 6-40%
- Progressive
  - ulceration
  - infection
  - amputation
  - death
**Charcot - Etiology**

- Diabetes mellitus
  - most common
- Tabes Dorsalis
- Syringomyelia
- Leprosy
- Pernicious anemia
- Pott’s Disease
- Paraplegia
- Alcohol Abuse

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**French Theory - 1868 (Charcot)**

- Neurovascular Theory
  - Neurally initiated vascular reflex. “Autosympathectomy”
  - High rate of flow produces AV shunting with subsequent
  - bone demineralization and breakdown.
  - Bone resorption by osteoclasts.
  - Hyperemia with secondary pathologic fractures.
German Theory - 1936 (Jordan)

- Neurotraumatic theory
  - Bone and joint changes caused by repetitive foot trauma on an insensate foot.
  - Trauma (Minor/Major)
    - Repetitive moderate stress
    - Repetitive impulse loading
    - Trabecular microfractures
    - Inadequately protected fractures/sprains
    - Surgery

Likely a COMBINATION of the two

- There is a neurally initiated vascular reflex that causes increased blood flow to the foot and ankle. This increased blood flow causes bone demineralization and breakdown by osteoclasts.
- In addition there is continued weight bearing on an insensate foot that causes pathologic fractures and breakdown of bones and joints.
Factors Contributing to Charcot

- NEUROPATHY: Requirement
- Increased local blood flow
- Increased plantar foot pressures
- (Equinus)
- Continued repetitive stress/trauma

Pathogenesis

- Sensory - Motor Neuropathy
  — loss of protective sensation
  — absent deep tendon reflexes
  — muscle weakness
  — ankle equines
Pathogenesis

- Autonomic neuropathy
  - Sympathetic denervation
  - Loss of vasomotor control
  - Increased blood flow
  - Increased arterio-venous shunting
  - Increased blood flow to bone

Clinical Presentation

- Relatively Painless
- Neuropathy
- Bounding Pulses
- Loss of Protective Sensation
- Ligament Instability
- Warmth
- Edema
- Erythema
**Brodsky - Type 1**

tarsometatarsal and lesser tarsus
Brodsky - Type 2

peri-talar

Brodsky - Type 3

3a = ankle; 3b = posterior calcaneus
Brodsky - Type 5

Forefoot

Eichenholtz - Stage 1

Stage of Development

- **CLINICAL**
  - Edema & Erythema
  - Calor & Hyperemia
  - Altered Function

- **RADIOGRAPHIC**
  - Fragmentation / Debris
  - Dislocation
  - Destruction / Resorption
  - Capsular Distention
Eichenholtz - Stage 3

**Stage of Reconstruction**

**CLINICAL**
- Resolution of signs and symptoms
- Residual Deformity

**RADIOGRAPHIC**
- Reformation of Joint Architecture
- Rounding of bone
- Decreased Sclerosis
- Consolidates / Remodel

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Stage 0 Charcot Foot

**AT RISK FOOT**
- May have suffered injury or increased activity
- Localized warmth
- Erythema
- **NO CHANGES SEEN ON XRAY**
Non-Operative Treatment Charcot

- Strict Non weight bearing 8-12 weeks
- Brace, Total contact cast, Crow Walker
- Immobilization continued to consolidation stage.
- Treatment is prolonged and challenging
- New therapy: Bisphosphonates, calcitonin, anti-inflammatories
- Bone stimulators useful
- Midfoot charcot: Keep immobilized 4-6 months
- Ankle charcot: 9 months to 1 year

Goals of Treatment

- Arrest disease process
- Offload any pressure area or ulceration
- Stabilize foot and ankle - prevent further deformity
- Restore function
  - plantigrade, brace-able foot
- Prevent amputation
- Save a life
Module 9 Surgery in The Diabetic Foot

Surgical management of the diabetic lower extremity can be a daunting task, but with appropriate patient and procedural selection, successful resolution of ulceration and correction of inciting pathology may be achieved. Diabetic foot surgery performed in the absence of critical limb ischemia is based on three fundamental variables: presence or absence of neuropathy (LOPS), presence or absence of an open wound, and presence or absence of acute limb-threatening infection.

Classifications of Surgery Surgical intervention has previously been classified as curative, ablative, or elective. More recently, a modification of this scheme has been proposed that encompasses more procedures and a broader spectrum of patients, as follows:

Class I: Elective foot surgery (performed to treat a painful deformity in a patient without loss of protective sensation)
Class II: Prophylactic foot surgery (performed to reduce risk of ulceration or reulceration in patients with loss of protective sensation but without open wound)
Class III: Curative foot surgery (performed to assist in healing an open wound)
Class IV: Emergent foot surgery (performed to arrest or limit progression of acute infection). For any of these classes, the presence of critical ischemia should prompt a vascular surgical evaluation to consider the urgency of the procedure and possible revascularization prior to or subsequent to the procedure.

Elective Surgery. The goal of elective surgery is to relieve the pain associated with particular deformities such as hammertoes, bunions, and bone spurs in patients without peripheral sensory neuropathy and at low risk for ulceration. Essentially any type of reconstructive foot operation can fall into this category, including rearfoot and ankle arthrodeses as well as Achilles tendon lengthenings. However, amputations are generally not performed as elective procedures, except in cases of severe deformity or instability resulting from prior injury or neuromuscular diseases.

Prophylactic Surgery. Prophylactic procedures are indicated to prevent ulceration from occurring or recurring in patients with neuropathy, including those with a past history of ulceration (but without active ulceration). These procedures involve correcting an underlying tendon, bone, or joint deformity. Many reconstructive procedures in this category would be considered elective if the patient did not have sensory neuropathy and a higher risk for ulceration.

Curative Surgery. Curative procedures are performed to effect healing of a non-healing ulcer or a chronically recurring ulcer when off-loading and standard wound care techniques are not effective. These include multiple surgical procedures aimed at removing areas of chronically increased peak pressure as well as procedures for resecting infected bone or joints as an alternative to partial foot amputation. Operations frequently performed in this regard include exostectomy, digital arthroplasty, sesamoidectomy, single or multiple metatarsal head resection, joint resection (Fig 17), or partial calcaneectomy. Some surgeons have proposed the advantages of combining plastic surgical flaps and skin grafts with these procedures to expedite wound healing and provide for more durable soft tissue coverage.

Emergent Surgery. Emergent procedures are performed to stop the progression of infection. Such ablative surgical intervention, most often involving amputation, requires removal of all infected and necrotic tissue to the level of viable soft tissue and bone. When possible, they are also performed in a manner to allow for the maximum function from the remaining portion of the limb. Wounds may be closed primarily if the surgeon is confident no infection or ischemic tissue remains and if enough soft tissue is available. Other wounds may initially be packed open, requiring well controlled and frequently assessed wound care, with delayed primary closure or closure by secondary intention.

Another popular option is negative pressure wound therapy using a V.A.C.® device, which has been found to significantly expedite granulation tissue formation and healing of open partial-foot amputations. Mechanical assistance using a variety of skin stretching devices are the surgeon’s option and may help attain delayed primary closure for some wounds. More often, V.A.C.® therapy is used to manage large or deeper wounds until delayed primary closure can be
achieved. Other approaches include plastic surgical techniques utilizing split and full-thickness skin grafts and a variety of flaps.

Each patient must be assessed for the selection of the surgical management that best meets his or her needs. Secondary wound healing with or without adjunctive wound therapies may still be the best choice for some patients. Pathway 6 lists the various types of surgical procedures commonly used for managing diabetic foot complications. In the carefully selected patient, prophylactic or elective surgical correction of structural deformities that cannot be accommodated by therapeutic footwear can serve to reduce high pressure areas and ultimately prevent ulcer recurrence.

Many of the procedures mentioned in the discussion on curative surgery would also be indicated in the elective/prophylactic reconstruction of the nonulcerated foot. Common operations performed in this regard include the correction of hammertoes, bunions, and various exostoses of the foot. Tendon-achilles lengthening procedures are often performed as ancillary procedures to reduce pressures that contribute to recurrent ulcerations. Once healed, these surgical patients are at high risk for future ulceration and require appropriate ongoing care consistent with those prevention strategies already discussed.

Amputation Considerations
Amputation, a well recognized consequence in the management of the diabetic foot, is performed for a variety of reasons and can be characterized as curative or emergent.

Indications for amputation include removal of gangrenous or infected tissue, often to control or arrest the spread of infection; removal of portions of the foot that frequently ulcerate; and creation of a functional unit that can accommodate either normal or modified shoe gear. In general, the amputation should be performed at a level that balances preservation of limb length and function with the capacity for the surgical site to heal primarily.

Although this concept is intuitive, several factors may influence the selection of the level of amputation. It is well recognized that energy expenditure increases as the level of amputation becomes more proximal. Simple tasks such as ambulating to the bathroom or other activities of daily living become increasingly more difficult for the patient commensurate with the level of amputation. In addition, patients with more proximal amputations are far more difficult to rehabilitate to a functional community or household ambulation level.

Recent advances in vascular surgery have enabled the level of amputation to become more distal or “limb sparing”. The capacity to re-establish distal perfusion with endovascular techniques or bypass surgery to the distal tibial, peroneal, and pedal arteries has greatly enhanced the potential for more distal amputation. In most circumstances, patients should be given the opportunity for vascular surgical intervention prior to definitive amputation so that the most distal level of amputation can be successful.

Goals of Selection of Amputation Level
The selection of the level of amputation should incorporate the following goals:

- Creation of a distal stump that can be easily accommodated by a shoe insert, orthotic device, modified shoe gear, or prosthesis
- Creation of a distal stump that is durable and unlikely to break down from exogenous pressure
- Creation of a distal stump that will not cause muscle or other dynamic imbalances.
- Examples include medial migration of the lesser digits after 1st MTP joint disarticulation; varus deformity and lateral overload after 5th ray resection; and equinus contracture after transmetatarsal or Chopart amputation.
- Healing with primary intention. In most instances it is advisable to perform an amputation at the most distal level that would allow for primary healing. Unfortunately, there are few objective tests or strategies that can consistently and reliably predict healing potential.
MODULE 10: CARE OF THE FEET AND THERAPEUTIC FOOTWEAR

- INTRODUCTION
- DAILY FOOT CARE CHECK LIST
- CHOOSING A FOOT WEAR
- THERAPEUTIC FOOT WEAR
- CHOOSING SOCKS
- RECOGNISING THE FOOT AT RISK
- CARING FOR THE FOOT AT RISK

INTRODUCTION
Type 2 diabetes is a leading cause of non-traumatic lower limb amputation worldwide. It is therefore important to teach the person living with diabetes, and his immediate care givers the basics of preventive foot care. Prevention they say is better than cure.

DAILY FOOTCARE CHECK LIST
This starts with emphasizing the need for proper care of diabetes through ensuring blood glucose levels and blood pressure control are to the appropriate target for the individuals. Home blood glucose monitoring should be discussed and incorporated into routine self-care if not already being done. The cholesterol level should also be to target. Adopting a healthy lifestyle, with a healthy diet, regular exercise and weight management when needed are also important topics to be discussed with the person living with diabetes. Where available the services of a registered dietitian, and certified diabetes educator should be used.

- The person living with diabetes should be taught to inspect and examine the feet every day, carefully inspecting in between the toes; looking out for red spots, athletes’ foot, cuts, swelling, and blisters. If they cannot see the bottoms of their feet, they should be taught to use a mirror to check under their feet or ask someone for help.

- Feet should be kept clean with daily washing in clean water, after which they should be dried with a clean towel, especially in-between the toes. Skin should be kept soft and smooth by rubbing skin lotion over the tops and bottoms of the feet, but not between the toes.

- Toe nails should be trimmed when needed. Toenails should be trimmed straight across and the edges filed with an emery board or nail file.

- They must never walk barefoot, but wear comfortable shoes that fit well and protect their feet. The insides of shoes must be checked with shoes turned upside down and shaking off any foreign object in them before wearing them. They should ensure the inside lining is smooth and there are no objects inside.

- Feet should be protected from temperature extremes – either hot or cold; ensuring shoes are worn at the beach or on hot pavement.

- Persons living with diabetes should avoid thong slippers or sandals as they may cause injury to the feet if not well fitted. Avoid thong sandals and slippers.
• Soaking of feet in water or dipping of feet into hot water should be avoided. The water temperature should be tested with the elbow before putting their feet into water for cleaning feet.

• They must NEVER use hot water bottles, heating pads, or electric blankets; as they can burn feet without realizing it.

Daily feet exercises to keep the blood flowing to into their feet are recommended.

• They shouldn’t cross their legs for long periods of time.
   For individuals who are not ambulant, and maybe bed bound, general care of the feet to prevent pressure ulcers are recommended.

• Smoking of cigarettes and any other smokeable items should be totally stopped.
• Shoes should be bought late in the afternoons, avoiding shoes with narrow toe-boxes, instead buying shoes with a wide toe-box.

THERAPEUTIC FOOT WEAR
Everyone can benefit from a shoe that fits well.
People with foot deformities such as claw or hammer toes, bunions etc. will benefit from therapeutic shoes. Therapeutic shoes have special features. Below are features that qualify them as medical treatment for people at risk for foot problems or who already have some damage.

CHARACTERISTICS OF A THERAPEUTIC FOOT WEAR

1. Adjustable:
   An adjustable closure, such as shoelaces or no-tie straps, can allow for different foot needs, day to day and hour to hour. Persons who have difficulty tying laces (for example, because of nerve damage in their fingers or joint problems). A Velcro closure might be best.

2. Wide Toe Box: Pain from squeezing feet into too-small and too-narrow shoes can lead to feet or bruises and ulcers.

3. SUSTAINABLE MATERIAL: Leather and microfiber are two materials that expand, preventing irritating friction if your foot swells. A shoe without some give is a shoe that will cause a blister.

4. Special Foot Bed: Therapeutic shoes typically have a foot bed that is wider and made with shock-absorbing materials. The foot care Specialist might also suggest a custom insert, which can relieve heel or arch pain, and can take pressure off areas that might be prone to calluses.
5. Extra Deep: An extra-deep shoe cradles the foot. Support around the ankle gives more stability. The extra depth gives foot deformities such as bunions and hammer toes the space they need. A deeper shoe also gives room for an insert or orthotic.

Who Needs a Special Shoe?
1. People with existing foot problems; therapeutic shoes can help them prevent more complications.
2. People with a previous amputation, past ulcers, calluses that could lead to foot ulcers, nerve damage (neuropathy), poor circulation, or a foot deformity
3. People with plantar ulcers

Patients should be taught to avoid socks that have seams as they can cause rubbing or irritation that can lead to a blister or callus.

A breathable material, such as cotton, or a wicking material, such as microfiber, can keep bacteria from forming.
Persons with circulation problems may need a compression sock or stocking, depending on the circulation in the feet or legs.

RECOGNISING THE FOOT AT RISK
The following can be present in a foot at risk but this list is not an exhaustive list.
Feet with neuropathy
Feet with poor circulation
Feet with deformities; e.g. hammer toes, calluses, bunions etc

Previous Ulcer
Previous amputation
Feet of a person who is bed bound

CARING FOR THE FOOT AT RISK: This involves teaching the patient and/or care giver how to incorporate all the dos and don’ts as listed above into the patient’s daily routine foot care.