DIABETIC FOOT SEPSIS

DR LYNNE TUDHOPE
NUMBER OF PEOPLE WITH DIABETES GLOBALLY

2000
171 million

2030
366 million

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of People</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000</td>
<td>171 million</td>
</tr>
<tr>
<td>2010</td>
<td>285 million</td>
</tr>
<tr>
<td>2011</td>
<td>366 million</td>
</tr>
<tr>
<td>2030</td>
<td>366 million</td>
</tr>
</tbody>
</table>

**2030** 552 million

The evolution of mankind...

Inactivity
4.8 million people died and 471 billion USD were spent due to diabetes in 2012.

HEALTHCARE EXPENDITURES AND DEATHS PER 1,000 DUE TO DIABETES BY INCOME GROUP
ADA DATA - MARCH 2013

• 41% increase in total cost of diabetes mellitus
  – $245 billion
  – 1 in $5 spent on health care goes to diabetes

• 43% on hospital inpatient costs

33% of direct cost burden of diabetes is in the lower extremity

(only 0.17% of research funding in the USA is spent on the diabetic foot!)
SOUTH AFRICA AND THE STATE OF DIABETES CARE

• Estimated population of 52.98 million
• Exact prevalence of diabetes is unknown
  – Estimated to be 5 – 7% of the population
    • Indians 11 – 13%
    • Coloureds 8 – 10%
    • Blacks 5 – 8%
    • Whites 4%
• ± 85% of these receive public (Government) sector medical care
  – Overburdened and inefficient
• ± 15% receive medical care in the private sector – paid for either by themselves or by medical insurance schemes
MEAN DIABETES-RELATED EXPENDITURE PER PERSON WITH DIABETES (USD) 2011

Botswana $816
South Africa $695
Namibia $468
Zimbabwe $56
Mozambique $37
Zambia $125
Tanzania $40
DRC $25
Malawi $31
Brazil $1038
USA $8468
UK $4267
Australia $4878
Canada $5106
Luxembourg $9341

http://www.idf.org/diabetesatlas
DIABETES MANAGEMENT IS GETTING MORE COMPLEX AND COSTLY

Urine Sugar → Blood Sugar → MDI /Analogs → CGM → closed loop
Pens, Pumps


Glargine, Detemir, Lispro, Aspart, Glulisine
THE DIABETIC FOOT

• Nearly 80% of all non traumatic amputations occur in diabetics
• 85% of these begin with a foot ulcer
• 1 in 4 people with diabetes will have an ulcer in their lifetime
• 50% of these will become infected
• 50% of patients who have a foot ulcer die within 5 year

• Diabetic foot sepsis = amputation = loss of bipedalism
CAUSES OF PREVALENCE OF DIABETIC FOOT PROBLEMS IN SOUTH AFRICA

Health Care Related

- Lack of Podiatrists (even in best hospitals)
- Insufficient experience of those undertaking foot care (surgeons, diabetologists, dermatologists)
- Central Distribution of “Good” health care services.
- Shortage of finances
CAUSES OF PREVALENCE OF DIABETIC FOOT PROBLEMS IN SOUTH AFRICA

Health Care Related

- Lack of health insurance of thousands of patients
- Lack of health education (busy clinics, few national programs, media, ....)
- Insufficient national data about different health problems
- Setting priorities (underestimation of foot problems)
CAUSES OF PREVALENCE OF DIABETIC FOOT PROBLEMS IN SOUTH AFRICA

Patients related Factors

- High Prevalence of DM
- Walking barefoot
- Illiteracy
- Associated comorbidities
- Poor compliance of patients
THE AMPUTATION RATE IN SOUTH AFRICA?

- **Published data** shows a 60.2% rate of non-traumatic lower limb amputation accountable to diabetes in public hospitals.
- **Unpublished data** from two separate public hospitals showed an amputation rate of 78.5% however.
- **Limb salvage rate** in a multidisciplinary clinic in a **private hospital** by contrast is 85% over a three year period.
CONSIDER INFECTION IN ANY FOOT WOUND IN A DIABETIC PATIENT

• Evaluate at 3 levels
  – patient as a whole
  – affected foot
    • arterial ischemia
    • venous insufficiency
    • presence of protective sensation
    • biomechanical problems
  – infected wound
EURODIALE

• PAD and peripheral neuropathy are both well known risk factors for diabetic foot ulceration and foot infection

• PAD and infection
  – PAD present in diabetes = X 5.5 increased risk for diabetic foot infection
  – 3x increase risk of amputation
SYSTEMIC EVALUATION

- ECG, stress test and even coronary angiography with intervention may be required
- Renal status and creatinine clearance
- Pulmonary function and chest preparation
- Control of diabetic status
- Identification and control of active infection
PHYSICAL EXAMINATION OF THE FOOT

- Presence of peripheral pulses
- Bruit
- Skin temperature and colour
- Hair loss, muscle and skin atrophy
- Dependant hyperaemia
- Skin ulceration or tissue loss
INFECTION?

- Erythema
- Swelling
- Induration
- Tenderness
- Malodor
Factors associated with increased risk of infection

• Positive probe to bone test
• Ulcer duration more than 30 days
• Traumatic wound
• Presence of PAD and PND
• Previous amputation
• Renal insufficiency
• History of walking barefoot
SIGNS OF POSSIBLE IMMINENT LIMB-THREATENING INFECTION

• Systemic inflammatory response
• Rapid progression of infection
• Extensive necrosis or gangrene
• Crepitus on examination or tissue gas on imaging
• Extensive ecchymoses or petechiae
• Bullae, especially hemorrhagic
• Pain out of proportion to clinical findings
• Recent loss of neurologic function
• Critical limb ischemia
• Extensive soft tissue loss
• Extensive boney destruction, especially midfoot/hindfoot
• Failure of infection to improve with appropriate Rx
INFECTIONS

• Mild
• Moderate
• Severe
## Texas Classification

<table>
<thead>
<tr>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Lesion completely epithelialised</td>
<td>Superficial wound, not involving tendon capsule or bone</td>
<td>Wound penetrating to capsule or tendon</td>
<td>Wound penetrating to joint or bone</td>
</tr>
<tr>
<td>0%</td>
<td>0%</td>
<td>0%</td>
<td>0%</td>
</tr>
<tr>
<td>Infection</td>
<td>Infection</td>
<td>Infection</td>
<td>Infection</td>
</tr>
<tr>
<td>12.5%</td>
<td>8.5%</td>
<td>28.5%</td>
<td>92%</td>
</tr>
<tr>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
<td>Ischemia</td>
</tr>
<tr>
<td>25%</td>
<td>25%</td>
<td>25%</td>
<td>100%</td>
</tr>
<tr>
<td>Infection and ischemia</td>
<td>Infection and ischemia</td>
<td>Infection and ischemia</td>
<td>Infection and ischemia</td>
</tr>
<tr>
<td>50%</td>
<td>50%</td>
<td>100%</td>
<td>100%</td>
</tr>
<tr>
<td>Clinical manifestation of infection</td>
<td>PEDIS grade</td>
<td>IDSA infection severity</td>
<td></td>
</tr>
<tr>
<td>-----------------------------------</td>
<td>-------------</td>
<td>------------------------</td>
<td></td>
</tr>
<tr>
<td>No symptoms or signs of infection</td>
<td>1</td>
<td>Uninfected</td>
<td></td>
</tr>
<tr>
<td>Infection present, as defined by the presence of at least 2 of the following: • Local swelling or induration • Erythema • Local tenderness or pain • Local warmth • Purulent discharge</td>
<td>2</td>
<td>Mild</td>
<td></td>
</tr>
<tr>
<td>Local infection involving skin and subcutaneous tissue without involvement of deeper tissue and no systemic signs. Erythema &gt;0,5 cm to ≤2cm around the ulcer. Exclude other causes of inflammatory response (acute Charcot, trauma, gout fracture)</td>
<td>3</td>
<td>Moderate</td>
<td></td>
</tr>
<tr>
<td>Local infection with erythema &gt;2cm or involving structures deeper then skin and subcutaneous tissues and no SIRS</td>
<td>4</td>
<td>Severe</td>
<td></td>
</tr>
<tr>
<td>Local infection with signs of SIRS with ≥2 of following • Temperature &gt;38° or &lt;36°C • Heart rate &gt;90 beats/min • Respiratory rate &gt;20 breaths/min or PaCO2 &lt;32 mmHg • WBS &gt;12 000 or &lt; 4000 or ≥10% immature forms</td>
<td>4</td>
<td>Severe</td>
<td></td>
</tr>
</tbody>
</table>
INFECTION

• Polymicrobial  
  – Aerobic gram+ cocci       → staphylococci, beta-hemolytic streptococci
  – Warm climates and exposure to water, gram-bacilli       → pseudomonas, E. coli
  – + aerobic bacilli in chronic wounds
  – Obligate anaerobes in ischemic / necrotic wounds
  – MRSA in diabetic foot wounds ranges from 5 to 30%
    • usually if previously hospitalised
Antibiotic regimens

• Clinically uninfected?
  – No antimicrobial therapy

• Clinically infected?
  – Select antibiotic targeting likely pathogen
  – As narrow spectrum as possible
  – Empirical choice should cover
    • Staphylococcus aureus
    • Gram + aerobic streptococci

• Only severe infections require IV Rx
  – Mild to moderate – 1 to 2 weeks Rx
  – Serious – 4 weeks of Rx
# Suggested Route, Setting and Duration of Antibiotic Rx

<table>
<thead>
<tr>
<th>Site of infection, by severity or extent</th>
<th>Route of administration</th>
<th>Setting</th>
<th>Duration of therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOFT TISSUE ONLY</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild</td>
<td>Topical or oral</td>
<td>Outpatient</td>
<td>1-2 weeks</td>
</tr>
<tr>
<td>Moderate</td>
<td>Oral (or initial IV)</td>
<td>Outpatient/inpatient</td>
<td>1-3 weeks</td>
</tr>
<tr>
<td>Severe</td>
<td>Initial IV, switch to oral when possible</td>
<td>Inpatient, then outpatient</td>
<td>2 – 4 weeks</td>
</tr>
<tr>
<td>BONE OR JOINT</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No residual infected tissue</td>
<td>IV or oral</td>
<td>…</td>
<td>2-5 days</td>
</tr>
<tr>
<td>Residual infected soft tissue (not bone)</td>
<td>IV or oral</td>
<td>…</td>
<td>1-3 weeks</td>
</tr>
<tr>
<td>Residual infected (but viable) bone</td>
<td>Initial IV, consider oral switch</td>
<td>…</td>
<td>4-6 weeks</td>
</tr>
<tr>
<td>No surgery or residual dead bone</td>
<td>Initial IV then oral switch</td>
<td></td>
<td>≥3 months</td>
</tr>
</tbody>
</table>
**SUGGESTED EMPIRIC ANTIBIOTIC REGIMENS BASED ON CLINICAL SEVERITY FOR DIABETIC FOOT INFECTIONS**

<table>
<thead>
<tr>
<th>Infection Severity</th>
<th>Probable Pathogen(s)</th>
<th>Antibiotic Agent</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild (usually treated with oral agent(s)) or severe (usually treated with parenteral agent(s))</td>
<td>Staphylococcus aureus (MSSA), Streptococcus spp.</td>
<td>Doxycycline</td>
<td>Requires QID dosing; narrow-spectrum; inexpensive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clindamycin¹</td>
<td>Usually active against community-associated MRSA; but check macrolide sensitivity and consider ordering a &quot;D-test&quot; before using for MRSA; inhibits protein synthesis of some bacterial toxins.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cefalexin²</td>
<td>Requires QID dosing; inexpensive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Levofloxacin³</td>
<td>Once-daily dosing; suboptimal against S. aureus</td>
</tr>
<tr>
<td></td>
<td>Methicillin-resistant S. aureus (MRSA)</td>
<td>Daptomycin</td>
<td>Active against many MRSA &amp; some gram-negatives; uncertain against streptococcal species.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Trimeprinö/iflunoxazole</td>
<td>Active against many MRSA &amp; some gram-negatives; uncertain activity against streptococcal.</td>
</tr>
<tr>
<td>Moderate (may be treated with oral or initial parenteral agent(s)) or severe (usually treated with parenteral agent(s))</td>
<td>MSSA, Streptococcus spp; Enterobacteriaceae; obligate anaerobes</td>
<td>Levofloxacin³</td>
<td>Once-daily dosing; suboptimal against S. aureus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cefotaxin⁵</td>
<td>Second-generation cephalosporin with anaerobic coverage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ceftaroline</td>
<td>Once-daily dosing; third-generation cephalosporin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ampicillin-sulbactam⁶</td>
<td>Adequate if low suspicion of P. aeruginosa</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Meropenem⁷</td>
<td>Once-daily oral dosing; Generally broad-spectrum, including most obligate anaerobic organisms.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tigecycline⁸</td>
<td>Active against MRSA. Spectrum may be excessively broad. High rates of nausea and vomiting and increased mortality warrant. Noninferior to etepenetin + vancomycin in 1 randomized clinical trial.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Levofloxacin³ or ciprofloxacin³ with clindamycin³</td>
<td>Limited evidence supporting clindamycin for severe S. aureus infections; PO &amp; IV formulations for both drugs.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Imipenemcilastatin⁹</td>
<td>Very broad-spectrum (but not against MRSA); use only when this is required. Consider when ESBL-producing pathogens suspected.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Linezolid⁰</td>
<td>Expensive; increased risk of toxicities when used &gt;2 wk.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Daptomycin⁹</td>
<td>Once-daily dosing. Requires serial monitoring of CPK.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Vancomycin⁰</td>
<td>Vancomycin MICs for MRSA are gradually increasing.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pseudomonas aeruginosa</td>
<td>Piperacillin-tazobactam¹</td>
</tr>
<tr>
<td>MRSA, Enterobacteriaceae, Pseudomonas, and obligate anaerobes</td>
<td>Vancomycin⁰, ceftazidime, cefepime, piperacillin-tazobactam³, aztreonam,¹ or a carbapenem³</td>
<td>Very broad-spectrum coverage; usually only used for empiric therapy of severe infection. Consider addition of obligate anaerobic coverage if cefotaxime, cefepime, or aztreonam selected.</td>
<td></td>
</tr>
</tbody>
</table>
Non infected wound

- Should not be Rx with topical or systemic antibiotics
- After open bypass surgery
  - wait 4 to 8 days before definitive debridement
- After endovascular intervention
  - wait 3 to 4 weeks
OSTEOMYELITIS

• Present in up to 20% of mild to moderate infections
• Present in 50% to 60% of severely infected wounds
• Consider in ulcers that are deep, large chronic or over a boney prominence
• Charcot difficult to distinguish from osteomyelitis and can co exist
• Radiographic changes of osteomyelitis may lag clinical disease by up to a month
• Antibiotic Rx based on culture results of bone
• Prolonged course of antibiotics (3-6 months) has a clinical success rate of 65%-80% in diabetic foot osteomyelitis
Wound

• Surgical debridement of dead tissue
• Appropriate antibiotic Rx
• Removing pressure off the wound
• Improve blood flow to the infected area

• Deep tissue specimen the best
• Superficial wound swabs often contaminated
• 50% volume decrease in 4 weeks or 10 to 15% decrease each week
• 40% of amputations are preventable with appropriate wound care
Schematic diagram of cross-section of the foot. Numbers 1 to 5 indicate metatarsal bones. A, central plantar space; B, deep interosseous space; C, lateral plantar space; D, medial plantar space.
BASIC TOOLS OF DEBRIDEMENT

- Blades
- Forceps
- Scissors
- Curettes
- Rongeurs
OFFICE DEBRIDEMENT: CURETTE

remove biofilm on top of wound
OFFICE DEBRIDEMENT:
SURGICAL BLADE OR NIPPERS

No 15 blade!
IDENTIFYING DEAD TISSUE

COLOUR CODING FOR IDIOTS

- Good
- Bad

EXCEPT VEIN
IDENTIFYING DEAD TISSUE: CLOTTED VEINS
DEBRIDEMENT OF NECROTIC MUSCLE
DEBRIDEMENT OF NECROTIC MUSCLE
DEBRIDEMENT OF NECROTIC MUSCLE
IDENTIFYING DEAD TISSUE: LIQUIFIED FASCIA
DEBRIDING FASCIA / TENDON:
DEAD TENDON = INFECTION
DEBRIDING BONE: PUNCTATE BLEEDING

“paprika sign”
Debridement

stop when....

only normal tissue remains & odour gone
WHEN IS OBTAINING A BONE SPECIMEN FOR CULTURE AND HISTOLOGY JUSTIFIED?

• When there is uncertainty regarding the diagnosis of osteomyelitis despite clinical and imaging evaluations
• An absence (or confusing mix) of culture data from soft tissue specimens
• Failure of the patient to respond to empiric AB Rx
RECOMMENDATIONS FOR COLLECTION OF SPECIMENS FOR CULTURE FROM DIABETIC FOOT WOUNDS

• **Do**
  – Obtain an appropriate specimen for culture from almost all infected wounds
  – Cleans and debride the wound before obtaining specimen for culture
  – Obtain a tissue specimen for culture by scraping with a sterile scalpel or dermal curette or biopsy from the base of a debrided ulcer
  – Aspirate any purulent secretions using a sterile needle and syringe
  – Promptly send specimens in a sterile container for aerobic and anaerobic culture

• **Do not**
  – Culture a clinically uninfected lesion, unless for specific epidemiological purposes
  – Obtain a specimen for culture without first cleansing or debriding the wound
  – Obtain a specimen for culture by swabbing the wound or wound drainage
Entropy

• Common to all systems
• Tend from a state of order to a state of chaos
• Young patient..... held in check by the body’s intrinsic system of repair and regeneration
• Older and immune suppressed patient.... This process is abandoned by the body
• It does not cause disease but it leaves an organism vulnerable
• Disease takes hold where the system is weakest
• In humans???
• The cardiovascular system
Diabetic Vascular Disease

- **Hyperglycemia**
  - Retina, Kidney, Nerves, Myocardium
  - Microvascular disease

- **Hyperglycemia**
  - Hypertension
  - Hyperlipidemia

- **Large Arteries**
  - Increased Rate of Atherosclerosis
Sustained decrease of major amputation in diabetic patients- an analysis of a 20 year period in a defined population

- When vascular evaluations and interventions were increased during a 20 year population based study on diabetics there was a 57% relative decrease in major amputations
INDICATION FOR RX

Any diabetic in whom pulses are not easily palpable, has a critically ischemic limb until proven otherwise.
Factors related to outcome of neuroischemic/ischemic foot ulcer in diabetic patients

- 1151 diabetic patients with neuro ischemic ulceration
- < 50% considered ischemic prior to non-invasive testing
DIAGNOSTIC MODALITIES

• Noninvasive Investigation
  – ABI ankle/brachial index (Doppler)
  – Segmental arterial pressures
  – Toe pressure and toe/brachial index
  – Spectral waveform analysis
  – Percutaneous $pO_2$ measurement

• Imaging Studies
  • Duplex Doppler with colour flow
    – Conventional angiography (Gold Standard)
    – MRI angiography
    – CT angiography
NON INVASIVE INVESTIGATION

- Ankle pressure < 50 (-70) mmHg
- Toe pressure < 30 (-50) mmHg
- TepO2 < 20-30 mmHg
  < 60 mmHg (sitting/O2)
ABPI – CONSENSUS STATEMENT

- Diabetic > 50yrs
  - Screening ABPI in all diabetics
  - Repeat every 5 years if test is normal
- Diabetic < 50 yrs
  - Screening ABPI if other PAD risk factors present
    - Smoking, hypertension, hyperlipidemia, duration of diabetes > 10yrs
- Diagnostic ABPI in any diabetic with PAD symptoms; together with treadmill testing if ABPI and clinical symptoms do not correlate
IDEAL NON-INVASIVE TEST = TOE PRESSURES

- Should be approximately 60% of brachial pressure
- Toe pressures
  - > 45 mmHg = 85% primary healing
  - < 45 mmHg = 36% healing without amputation
TcPO2 levels

< 34 mmHg (amputation 85%) indicates the need for revascularization

> 34 < 40 mmHg (amputation 20%) less pressing, although there remains a considerable probability of amputation

> 40 mmHg revascularization is dependent on the severity of tissue loss and possible morbidity caused by the procedure

SOUNDS

• Doppler sounds are described as either tri-, bi-, or monophasic.
• Triphasic flow indicates normal arterial flow; the pulse curve has three components.
• Biphasic flow implies a loss of one component and mildly compromised flow.
• Monophasic flow, in contrast, indicates arterial compromise either due to a significant stenosis or narrowing or occlusion of the artery.
Fig 7: **Arterial↔arterial connections:**
By occluding arteries proximal or distal to the arterial signal, it is possible to assess the direction of arterial flow.
Direct vascular connections:
arterial ↔ arterial connections

All the main arteries of the foot and ankle are directly connected to one another
Doppler: peroneal artery: anterior perforating branch
Doppler anterior perforating branch of the peroneal artery just medial to distal fibula
Doppler: Anterior tibial artery → dorsalis pedis
Doppler:

✓ antegrade flow posterior tibial artery

Locate artery

✓ for antegrade flow
THE IMPORTANCE OF ANGIOSOMES IN HEALING FOOT ULCERS

• Taylor describes at least 40 angiosomes in the body, of which 5 are found in the foot and ankle.
• These originate from the three main arteries in the lower extremity, the posterior tibial artery, the anterior tibial artery and the peroneal artery.
• The posterior tibial artery supplies the sole of the foot via the calcaneal branch, the medial plantar branch and the lateral plantar branch.
THE IMPORTANCE OF ANGIOSOMES IN HEALING FOOT ULCERS

• The anterior tibial artery supplies the anterior ankle and as the dorsali pedis artery, it also supplies the dorsum of the foot.

• The peroneal artery supplies the lateral anterior upper ankle via its anterior perforating branch and also supplies the plantar heel area via a calcaneal branch.

• Arterial to arterial connections are important because, despite the occlusion of one or more arteries to the foot, these connections provide an uninterrupted blood flow to the entire foot.
The importance of angiosomes in healing foot ulcers

• The treatment of ischemia in the diabetic foot should be aimed at the restoration of maximum blood flow to the foot with the restoration of pulsatile palpable foot pulses whenever possible.
• This pulsatile flow increases the chance of wound healing and diminishes future skin breakdown and ulcer formation.
• In planning any surgical procedure on the foot, or when embarking on any course of wound care treatment, it is essential that optimum blood flow is obtained in the area of tissue breakdown.
• By understanding the principle of angiosomes and the vascular anatomy of the foot, wound healing and foot salvage will be easier to predict.
• It has been reported that up to 15% of bypasses to the foot fail to heal wounds on the foot, in spite of remaining patent, simply because these bypasses failed to revascularize the affected angiosome.

• It is, therefore, crucial that bypass procedures are done to the right blood vessel, if existent ischemic ulcers are to be healed.
VASCULAR SUPPLY

• Regulation of vasculature
  – Arterial supply maximization
  – Periwound edema minimization
PERIPHERAL NEUROPATHY

• Affects sensory, motor and autonomic innervation
• Loss of sensation – wounds go unnoticed
  – Reduction of pain and tenderness.... No early warning system
• Motor nerve damage – foot deformity, abnormal pressure points, callus, ulceration.
• Autonomic neuropathy – dry skin, heel cracks, tearing and infection
BARRIERS TO EFFECTIVE MANAGEMENT

• Importance of foot care not recognised
• Ignorance of improved patient outcomes with better foot care
• Non existent podiatry services
• Team approach lacking
• Routine referral for amputation
• Limited training programs for healthcare providers
BARRIERS TO EFFECTIVE MANAGEMENT

• Services run by non specialist nurses not foot care specialists
• Barefoot walking common
• Faith healers, herbalists and home therapies
• Unaffordable footwear
• Poverty; limited access to care
THE DIABETIC FOOT

- Establish contacts in healthcare
- Raise funds
- Foot clinics beginning with a diabetes centre for excellence
- Multidisciplinary educational approach
- Establish attainable goals
- Recruit, train and retain
- Motivate healthcare professionals
SERVICES NEEDED

- Foot Screening
- Nail Care
- Ulcer Care
- Debridement
- Offloading Devices
- Education
In summary

• non infected wound?
  – no antibiotics
  – specialised wound care
  – off loading

• Infected wound?
  – stage wound
  – chemical or mechanical debridement
  – tissue and/or bone biopsy
  – oral or IV antibiotics
  – off loading
THE DIABETIC FOOT: Two decades of “progress”

1986: First Malvern Diabetic Foot Meeting
1987: Foot Council of ADA formed
1991: First International Diabetic Foot Meeting
1998: Diabetic Foot Study Group of EASD founded
1999: International Consensus group publishes “Guidelines on management”
2001: Formation of DFSIs
2002: First DFCon meeting, Universal City, Los Angeles
2004: Formation of GLEPED
2005: IDF designated year of the Diabetic Foot
2007: Formation of DFWG, South Africa
2007: Fifth International Diabetic Foot meeting
2009: Opening of first foot clinic in Colombia
2010: 13th biennial Malvern Diabetic Foot meeting
2010: First Pan-African Diabetic Foot meeting, Spier
2014: 2nd Pan-African Diabetic Foot meeting, Dar es Salaam
Laughter the best medicine?

• 5 year study – Leeds School of Healthcare
  – “A hearty chuckle stimulates the diaphragm which in turn plays a vital role in moving blood around the body and speeds recovery from leg ulcers”