Diabetic Foot –The Impending Crisis

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1. Introduction
The world is currently experiencing an epidemic of Diabetes Mellitus, particularly Type II or Adult onset. (1) The United States has suffered a 30% increase in the prevalence of the disease in the last 10 years to 7.9% of the population. (2) Moreover, it is recognized that, in the future, the majority of this increasing number of patients will live in the developing countries of Asia, Africa and Latin America. Diabetic foot disease constitutes the most common cause for hospital admissions in diabetic patients. From 15-25% of diabetics will develop a foot ulcer during their lifetime. (3) Diabetics experience lower limb amputation at a rate 10-30 times that of the general population and 85% of these are preceded by foot ulcers. Mortality after amputation rises from a minimum of 15% after 1 year to 40-80% after 5 years. There is substantial evidence that this enormous burden of disease can be prevented with appropriate screening and management measures employed by a multidisciplinary team. (4) A number of very good reviews on this subject have been published including practice guidelines. (5-9) This Review will discuss the epidemiology, pathophysiology, diagnosis, prevention and practical management of the diabetic foot, from the context of the conditions in the developing world.

2. Definition
The definition of diabetes, taken from the American Diabetic Association is: 1. signs and symptoms of glycosuria or a random blood glucose greater than 11.1mmol/dL (200mg%); 2. a fasting blood glucose > 7mmol/dL (126mg%) on two occasions or 3. 2Hr BG after oral 75gm. load of 11.1mmol/dL (200mg%). (10)
3. Epidemiology

3.1. Diabetes in the developing world

There are well known variations in diabetes prevalence among different populations. The Diabetes Atlas (11) depicts these differences. The sharp increase in diabetes worldwide is felt to be due primarily to lifestyle changes: dietary changes and reduction in physical activity resulting in obesity and associated with urbanization. Age, obesity and a positive family history are associated with increased risk. The correlation with obesity and increased body mass index (BMI) is particularly strong in women. (12) The strong relationship between diabetes and accelerated atherosclerotic occlusive disease is a major source of increasing mortality rates. In India, death from diabetes is usually a result of infection or chronic renal failure as opposed to that in the developed world where myocardial infarction and stroke are the major causes of death. (13) Similar to global statistics, 80% of diabetes in Africa is type II and the greatest increase in prevalence is in adults as well. (14;15) While population data are limited, the increase is undeniable. The number of Africans expected to suffer from diabetes is expected to double in the next 25 years. This poses an enormous challenge to the already inadequate provision of medical care to the people of sub-Saharan Africa. (16;17) Aspray et al showed a marked difference between urban and rural diabetes rates in Tanzania. (18) In a small cluster sample study in Port Harcourt, Nigeria the standardized prevalence rate was estimated at 7.9%, which is high. (19) In Ghana the prevalence rate was determined to be 6.3%. (20) The disease affects communities and ethnicities differently; those from the Indian subcontinent are more severely affected as are Afro-Americans and Afro-Caribbean, as well as Native Americans. This has particular impact in certain countries of Central and South America.

3.2. Diabetic foot disease

In a study from Cameroon, diabetic foot ulceration DFU was the second most common cause of hospital admission and the main cause of prolonged hospital stay. (21) Morbach compared foot disease in Germany, India and Tanzania. (22) Abbas reviewed the epidemiology of diabetic foot disease in Africa. (23) Despite wide country variations of neuropathy and peripheral vascular disease, a common feature of inpatient admission with foot ulceration was high rates of amputation (33%) and death (50%) when amputation for severe neuro-ischemic lesions was not performed. Specific features include unrecognized foot trauma from walking barefoot or in flip-flops, poverty, lack of diabetes education and lack of access to medical care leading to delayed presentation. Tropical Diabetic Hand Syndrome is a specific clinical entity. (24) In a recent study from Nigeria, Ogbera et al found a case fatality rate of an astonishing 53% of admissions with DFU. (99) Amputation rates can be seen as a marker for the quality of foot care. (25) In the United States, blacks suffer increased rates of amputation compared to whites. This is not true in the United Kingdom. (26) It is clear, however, that once foot ulceration occurs, the risk of subsequent ulceration and amputation rises to between 10 and 25%. Thus solutions must include preventative measures as well as improved treatment. (27) Measures such as strict glycemic control as well as participation in multi-disciplinary diabetic clinics have been shown to significantly reduce complications (35% reduction for each 1% decrease in HbA1c) and amputation rates. (see Prevention)
4. Pathophysiology
The complications of diabetes result from a series of microvascular and macrovascular changes to the circulation. (28;29) Retinopathy, nephropathy and neuropathy are consequences of the microvascular changes. The diabetic foot, itself, results from a complex interplay of a number of pathologic factors. (30) Neuropathy resulting in abnormal biomechanics leads to unrecognized trauma and thus to ulceration. The impairment of wound healing in diabetics is only recently being recognized. (31;32) Vascular insufficiency, the consequence of macrovascular accelerated atherosclerosis, and infection are two additional factors which threaten the viability of the limb.

4.1. Neuropathy
The main pathologic process leading to the diabetic foot is a symmetrical distal neuropathy affecting sensory, motor and autonomic nerves. (33) The sensory abnormalities result in a stocking glove distribution with inability to perceive light touch, pressure and pain. This leads to loss of protective sensation (LOPS) and unrecognized trauma. The motor abnormalities result primarily in a paralysis of the intrinsic muscles of the feet and foot deformity. These deformities, including hammer and claw toes and rocker bottom abnormality of the sole, are the main precursors to the abnormal biomechanics of the foot. Finally the autonomic dysfunction causes decreased sweating and results in a dry, scaling skin susceptible to fungal and other superficial infections. 44% of patients attending a clinic in Tanzania, reported paresthesia. (34) Neuropathic osteoarthropathy or Charcot’s foot is a particular aggressive form of deforming arthritis resulting from micro- and macro-trauma to the articular surfaces of the tarsal and metatarsal bones and supporting structures. It is characterized by joint dislocation and pathologic fractures. The role of inflammatory cytokines has been suggested. (35) Recently the use of bisphosphonates has been recommended to reduce osteoclastic bone resorption. (36)

4.2. Vascular insufficiency
Diabetics suffer 3-4 times the risk of myocardial infarction and stroke, which together are the major causes of death in diabetics in North America. These result from an accelerated atherosclerosis whose causes are complex. (29) The additional risk factors of hypertension, hyperlipidemia, smoking and family history are also important and their effect cumulative. Peripheral vascular disease PVD is also more prevalent in diabetics and occurs at a younger age. (37;38) There is a specific pattern of infrapopliteal disease requiring more distal bypass in these patients. There is also a pattern of medial calcification in the vessels which MAY make noninvasive identification of insufficiency difficult. Ischemia should always be considered as a factor in the non-healing ulcer. In general however revascularization is only required in 20-25% of cases.

4.3. Infection
Infection of pre-existing lesions often precipitates medical attention. (39) The portal of entry may be small abrasions resulting from trauma, fungal infection or longstanding ulcers. The organisms are those associated with skin colonization including staphylococcus aureus and Group A and B streptococci. When the wound is more than one month in duration, gram negative aerobes such as pseudomonas, gram negative anaerobes such as bacteroides fragilis and enterococci are often found. In a report from Nigeria (40), staph aureus was isolated from 56% of patients and anaerobes were found in only 5%. The mean number of isolates/patient was 2.3. Concomitant neuropathy
decreases perception of infection and co-existing vascular insufficiency leads to spread of the infection in ischemic tissues.

4.4. Biomechanics

Diabetic neuropathy with LOPS and foot deformities result in abnormal plantar pressures which become the final common pathway to the development of the typical malperforans ulcer. (41;42) (Picture 1) Most ulcers occur on the sole of the foot. The malperforans ulcer on the plantar surface in a region of high pressure overlying the metatarsal head is the expression of these pathologic processes. Walking barefoot or wearing constricting shoes further exacerbates the abnormal biomechanics and has been shown to lead to ulceration. (43) While therapeutic shoes have been shown to lower plantar pressures, there is some doubt (44) whether their use can prevent ulceration. Prospective trials yielding scientific evidence are required here.

5. Diagnosis

Numerous guidelines argue the value of patient education and routine examination of the feet of diabetics within the context of the multi-disciplinary diabetic clinic. (7;45:46) Attempts have been made to ground these recommendations in evidence. (47)

5.1. History

Diabetics should be questioned about the duration of illness; type of medication used and compliance; diet; exercise; footwear and symptoms relating to eyesight, renal status and foot problems. Risk factors for atherosclerotic disease should be similarly enquired about. A history of prior foot ulceration is a significant risk factor for identification of the high risk patient. However since symptoms may be delayed in the neuropathic foot, every diabetic should have their feet examined at every clinic visit.

5.2. Physical exam

A rapid systematic examination of the foot can be developed. (48) The condition of the nails should be examined for paronychia, ingrown nails or onchomycosis; of the skin for fungal infection in the inter-digital areas, for edema, colour and temperature; for evidence of foot deformity; the range of motion of the various joints and presence or absence of lower extremity and pedal pulses. Finally the characteristics of any foot lesions, from calluses to ulcer or gangrene need to be documented. Ulcers which appear to be deep or are draining fluid should be probed to assess penetration to joint or bone.

The specific diagnosis of diabetic foot infection is crucial and may be difficult in the ischemic foot. (49) Cultures of draining fluid should be taken, but superficial cultures may be misleading and curetting or biopsy of deeper structures at the time of debridement is important. The presence or absence of osteomyelitis needs to be determined in every foot lesion and often requires radiology. (50)

5.3. Neurologic exam

Assessment of the presence of neuropathy is a vital tool in the identification of the high risk patient. The Semmes-Weinstein 5.07 monofilament (SWM) is an inexpensive and accurate measure of LOPS. (51) Appropriately used it measures 10 gm of pressure and is highly predictive of risk of developing ulceration. (52) Assessment of tendon reflexes and vibration sense with a 128 Hz tuning fork are also part of the neurologic assessment.

5.4. Noninvasive vascular assessment

Since vascular insufficiency is a strong risk factor for failure of ulcer healing and for amputation, every patient with absent pedal pulses should undergo non-invasive assessment. (53) Segmental pressures determined by hand held Doppler will be adequate in the majority of patients. An ankle/brachial index (ABI)
< 0.8 is indicative of vascular insufficiency consistent with claudication. Ankle pressure < 50 mmHg or ABI<0.4 is indicative of more severe disease associated with rest pain and limb threatening ischemia. The role of calcified vessels in causing falsely high ankle pressures is also important although its significance is controversial. Raising the leg relative to a pole and recording the height where the Doppler signal disappears is one method of eliminating this effect. The measurement of toe pressures circumvents the issue of arterial calcification and has been promoted as more accurate in diabetics but requires the use of photoplethysmography. Bonham recently showed that hand held Doppler measurements of toe pressures were not equivalent to those derived by photoplethysmography. Furthermore, Brooks has questioned the need for toe pressures unless the ABI >1.3, indicating incompressible vessels. The use of noninvasive tests to predict healing of foot lesions or levels of amputations is similarly controversial, although toe pressures by photoplethysmography is felt to be the most useful measurement. (See amputation) Transcutaneous oxygen measurement (TCOM) is recognized as an accurate predictor of healing with TPO2>25mmHg indicative of healing and < 25mmHg as predictive of failure to heal. However the equipment is expensive. Identification of severe PVD is an indication for arteriography and revascularization, if local capacity exists, or referral, if it does not.

5.5. Radiology Every foot harboring a diabetic ulcer should be xrayed to assess for the presence of osteomyelitis or other abnormalities such as Charcot’s foot. These two entities may be difficult to distinguish radiologically. Morrison gives an extensive discussion of the use of MRI in these patients and its ability to distinguish the two.

6. Classification A number of different types of classification systems have been developed to aid in the assessment of the high risk patient and management of the diabetic foot. The University of Texas Diabetic Foot Classification can be used in the diabetic clinic as a means of identifying the high risk patient and guiding intervention. The first 4 categories are entirely lacking foot lesions and intervention here is preventative.

Category 0: the protective sensation is intact. Patients in this category have 1/5 the risk of developing foot ulcer than subsequent categories. Education on footcare is given and the patient may be seen annually.

Category 1: LOPS is found but without history of previous ulceration. Footcare education and prescription of therapeutic shoes is recommended and closer follow-up every 6 months.

Category 2: LOPS and deformity mandates closer follow-up every 3 months and possible elective corrective surgery.

Category 3: LOPS, deformity and a history of previous ulceration indicates the highest level of risk and mandates monthly follow-up.

Category 4: The first category with actual ulceration is divided into A – non-infected, non-ischemic neuropathic ulcer – all U of Texas Stage A ulcers (see below) and B– acute Charcot’s foot. Treatment of 4A is primarily debridement and orthotic device, such as total contact casting. Treatment of Charcot’s foot is primarily directed at immobilization and stress reduction in the acute phase.

Category 5: Infected diabetic foot – treatment directed towards control of infection as well as underlying abnormalities.
**Category 6:** Ischemic foot – ABI<0.6; Toe pressure<40mmHg; TCO2<30mmHg. A referral to a vascular surgeon is indicated.

While the above classification is used to direct action in the outpatient clinic, the University of Texas Wound Classification system focuses on ulcers themselves.

**Grade 0:** Pre or post ulcer – skin epithelialized; **Grade 1:** Superficial ulceration; **Grade 2:** Wound penetrating to tendon or capsule; **Grade 3:** Wound penetrating joint or bone. The wound is then staged: **Stage A:** Not infected or ischemic; **Stage B:** Infected; **Stage C:** Ischemic; **Stage D:** infected and ischemic.

There are a number of other classifications systems – Wagners (45) and Pedis. Strauss (60) evaluates these and proposes his own system.

### 7. Prevention

Because the presence of an ulcer is itself a major risk factor for repeat ulceration, an important focus of the management of this problem is preventative.(3) This can be classified as primary to prevent the diabetic state itself and secondary to prevent foot ulcers in diabetics.

#### 7.1. Primary

Education, aimed at the general population in order to forestall the development of diabetes, should be directed towards issues of diet, exercise and lifestyle. However various impediments to this activity exist. In many countries diabetes carries a considerable stigma and fear of the diagnosis prevents people from altering their behaviour and seeking early medical attention. Many countries with limited resources do not at present consider chronic non-communicable disease as a priority. (61) The World Health Organization and the International Diabetic Federation have both raised the alarm concerning the epidemic of diabetes in the developing world. The significance of diabetic foot disease was recognized in the World Diabetes Day 2005 which focused on diabetic footcare. [http://www.diabetic-foot-consensus.com/index.php](http://www.diabetic-foot-consensus.com/index.php)

#### 7.2. Secondary

There is evidence that 3 types of interventions can reduce the risk of developing foot ulcers in diabetic patients.(62;63)

**7.2.1. Glycemic control** While a complete description of the medical management of diabetes is beyond the scope of this review, the interested reader is referred to “Approach to the Management of Diabetes Mellitus.”(64) Hyperglycemia results in glycolation of the haemoglobin molecule into HbA1c, which becomes a marker for chronic hyperglycemia. Normal value is < 6 %. This should be measured every 3 months. A reduction of HbA1c levels from 9.0 to 8.0 was associated with a 25% reduction in microvascular complications and a smaller reduction in myocardial infarcts. (65)

**7.2.2. The Diabetic clinic** The role of the multi-disciplinary diabetic clinic, with regard to the diabetic foot, is to identify high-risk patients, educate them in footcare, encourage the use of appropriate footwear and diagnose and treat ulceration at an earlier stage. The core of the team includes diabetic nurse clinicians and educators with access to podiatrists, internists, ophthalmologists, nephrologists, vascular and orthopedic surgeons. While some clinics have reported decreased amputation rates of more than 50%, a systematic review of studies for the Cochrane database concluded that most studies examining the value of education interventions were of poor quality. (66) The reference clinic in the “Save the Diabetic Foot” project in Brazil reduced amputations by 77%.
Hunt (4) believes there is good evidence for these interventions. The Registered Nurses Association of Ontario has published a set of guidelines for assessments and interventions in the diabetic clinic and the level of evidence for each of these. (67) Assessment of sensation with SWM, history of foot ulceration, foot deformity, evidence of impaired circulation and deficits in self-care are all used to identify the high risk patient. Education concerning the need for daily inspection of the feet and the wearing of footwear can take the form of visual aids in illiterate patients.

7.2.3. Orthotics Since it is well recognized that elevated plantar pressures can be reduced while wearing therapeutic shoes with increased depth, soft insoles and rocker bottoms, prescription of these shoes has been standard for the high risk patient. Sadly the evidence for the value of therapeutic shoes to prevent ulcers in high risk patients is equivocal. (3;68) The quality of evidence assessing their preventative value in patients with prior ulceration is also inconclusive (44:68), although Hunt (4) believes there is good evidence to recommend therapeutic footwear. Given the fact that many patients in the tropical, developing world walk without any footwear whatsoever or with only sandals, exposing their insensate feet to unrecognized trauma, or wear constricting footwear which actually causes ulcerations; the recommendation to use therapeutic footwear in the high risk patients seems sensible. However expensive or “ugly” footwear will not be purchased or worn. In this regard, it has been shown that high quality off the shelf running shoes may do as well as therapeutic shoes in lowering plantar pressures with better compliance. (43)

8. Management
Like prevention, management of the diabetic foot ulcer requires a multi-disciplinary approach. (47;69;70) Stabilization of the diabetes and associated medical problems, antibiotics, surgery, and orthotic devices all play their role. As with preventative care, the evidence base for management practice is not strong. (71)

8.1. Medical control
Glycemic control in the patient with an infected foot may be very difficult. The diabetic requiring surgical intervention is at increased risk of peri-operative MI, stoke and renal failure. Proper identification and management of the medical risk factors is important. (64) Adequate hydration, aspirin, beta-blockers, anti-lipid agents and DVT prophylaxis should be considered.

8.2. Antibiotics and infection
Diabetic foot infection is a common consequence of ulceration and a major risk factor for amputation. Neuropathy and arterial insufficiency may diminish the normal clinical signs of diabetic foot infection. (72) Resistant hyperglycemia may be an important clue to occult infection. Edmonds recommends antibiotic therapy for all ulcers. (73) Infection itself may be difficult to diagnose. (74)

With regard to infection the foot can be classified as Grade 1 – no infection; Grade 2 - involving only skin and superficial subcutaneous tissue with cellulitis extending less than 2 cm from the lesion; Grade 3 - limb threatening with > 2 cm. cellulitis or involvement of tendon, joint or bone and Grade 4 - life threatening infections with a systemic inflammatory response syndrome causing significant temperature, tachycardia, hypotension, leukocytosis, toxicity and/or confusion. (75) While Grade 2 infections may respond to antibiotics alone, deeper infections associated with abscess, gangrene or osteomyelitis (see below) require surgical incision, debridement or amputation to control
infection.
Initial antibiotic therapy will be empirical and based on knowledge of the usual infecting organisms. Oral B-lactamase resistant antibiotics are usually prescribed for Grade 2 infections: cloxacillin, amoxicillin/clavulanate or clindamycin in those with penicillin allergy. Recommendations require alteration in the presence of MRSA. Older wounds require the addition of agents against gram negative aerobes such as ciprofloxacin and anaerobes such as metronidazole. With Grade 3 and 4 infections, antibiotic therapy should be broader based and parenteral. Coverage should include gram positive aerobes, gram negative aerobes and anaerobes. Third generation cephalosporins + metronidazole; clindamycin + aminoglycosides are both acceptable regimes. There is no evidence suggesting the value of one regime over another. (76) Local cost and availability are factors in choice.
The identity of the infecting organism(s) should always be determined by culture. Superficial swabbing may not yield a correct identification and deeper infected material such as pus, curettings, and biopsy of bone or other material at debridement should be cultured. Two systematic reviews conclude that the evidence is too weak to choose one strategy over another. (77;78)

8.2.1 Osteomyelitis can be diagnosed by probing ulcers (positive if bone is found at the base) or by xray or other imaging. However, this is not foolproof. Bonham reviews the available evidence in the diagnosis and treatment of osteomyelitis in diabetic feet and determines it is inconclusive. (50;79) Once diagnosed, osteomyelitis and septic arthritis almost always mandate surgical excision, either with or without amputation.

8.3. Surgery
The various surgical approaches to the diabetic foot extend from simple debridement, to incision and drainage of abscesses and bone excision for osteomyelitis, to local and major amputation for gangrene, to angioplasty and open revascularization for ischemic limbs and finally reconstructive surgery to prevent ulceration and correct deformities. (80)

8.3.1. Debridement Virtually every ulcer requires debridement. All macerated, hyperkeratotic or infected material needs to be excised making the ulcer as flat as possible. Measurement of the dimensions should be taken for monitoring. (Pictures 2-5) Grade 1-2 Stage A ulcers can be debrided of callus and necrotic material and then a pressure off-loading device such as a total contact cast can be applied. This is the accepted and proven outpatient therapy for these ulcers. More aggressive disease requires more aggressive debridement. The basic principle is that all infected, necrotic tissue must be removed; until well vascularized tissue remains. The reality is that debridement is usually inadequate or delayed. Repeated debridements are often necessary. Ankle blocks should provide adequate analgesia.

8.3.2. Amputation
8.3.2.1. Local amputation may be a simple extension of debridement. (81) Transphalangeal amputation is indicated when the disease process affects the deep tissues of the toe, such as tendon, bone or joint precluding soft tissue coverage and with normal skin and soft tissue at the region of the proximal phalanx. Ray amputation, taking the metatarsal head, is an extension of this for more proximal disease, often excising the originating ulcer on the plantar surface. Transmetatarsal amputation is reserved for cases of more major gangrene or infection involving multiple digits. Symes foot disarticulation should be reserved for cases with excellent posterior tibial pulses where forefoot
amputations will not suffice. Improvisation of standard incisions may be necessary to insure soft tissue coverage. The wounds of infected feet should be left open or closed using delayed primary closure.

Adequate vascular supply can be assured if ABI > .5, ankle pressures > 70mmHg, toe pressures >30-55mmHg or TPO2 >30. In the presence of palpable pedal pulses all foot lesions should heal. A foot lesion with an ankle pressure < 55mmHg is unlikely to heal. In general, tests of distal circulation are more accurate in predicting healing than in predicting failure to heal. The International Consensus on the Diabetic Foot has published a useful graph showing the probability of healing a foot ulcer with different indicators of vascular status. (see figure) A transmetatarsal signal or digital signal with the handheld doppler is encouraging. A liberal policy of revascularization can be applied if local capacity exists.

8.3.2.2. Major Amputation may be required to control sepsis or excise gangrene when minor amputations will not suffice or where revascularization is unavailable or unsuccessful. The below knee amputation BKA affords considerable rehabilitation potential in comparison to the above knee amputation and should be chosen wherever possible. However vascular supply needs to be assured. A popliteal pulse is an obvious favourable indicator but calf pressures >65 and ankle pressures >30 provide assurance for primary healing of BKA. 90% of BKAs will heal if the TPO2 >35mmHg 10cm below the knee. A minimum below which BKAs fail is less easily determined. Standard recommendations are for a long posterior flap. There is no evidence favoring one surgical approach over another. (82)

8.3.3. Revascularization While the need for revascularization will vary with the age and demographics of differing diabetic populations, the capacity to improve arterial supply will save limbs. In the developed world a sophisticated vascular surgical capability has developed which may not exist in most developing countries. However vascular injuries and diabetic feet are two conditions whose frequency warrants such development. Arterial reconstruction in the diabetic proceeds using the standard assessment and operative techniques, with endovascular or open approaches both being used. (83) Since the disease in diabetics has an infrainguinal and infrapopliteal predilection, more distal bypasses will be required using autologous vein grafts, either reversed or after valvulotomy. (84)

8.3.4. Reconstruction surgery in the diabetic foot extends from wound closure techniques of flap and skin graft to surgery to prevent ulcers by correcting foot deformities and removing bony pressure points to reconstruction for Charcot’s foot. Wieman describes the technique of excision of the metatarsal heads for the purpose of offloading pressure or to treat infection. (85)

8.4 Wound Care
The healing of diabetic wounds requires the excision of necrotic material, the elimination of infection, the adequacy of circulation and the offloading of pressure. (86) Even when these conditions are ensured, healing of wound may be delayed. The principles of wound care are to fill the cavity, absorb exudates, maintain a moist environment, provide thermal insulation (in cold climates) and promote healing. Expense and ease of nursing care are important considerations. A wide variety of different agents are available. Uninfected ulcers may be treated with off-loading techniques alone (see below) such as felted-foam or total contact casts. Occlusive hydrocolloid dressings are useful in areas of low to
moderate exudate. (87) Alginates are used where there is high volume of exudate. (88) Silver sulfadiazine dressings are currently popular for infected wounds, but they have not been submitted to scientific study. (89) Other agents such as hyperbaric oxygen (90), negative pressure wound therapy (91), and granulocyte stimulating factor (92) have all been promoted.

8.5. Orthotics
Despite the relative lack of scientific evidence (68), the use of orthotic devices is one of the mainstays of diabetic foot management. Pressure off loading can be obtained by bedrest and elevation of the leg or wheelchairs, however these are impractical and dangerous for the long periods required for ulcer healing. Diabetics generally do not have the upper body strength to use crutches. A wide variety of orthotic devices have been developed. (93;94) For non-infected ulcers a felted foam dressing (95) or total contact casting may be used. The latter has proven to be better than dressing alone in randomized control trials. (88) It is contraindicated in infected or ischemic ulcers. While prefabricated diabetic orthotic devices are widely used in North America, being removable they are often not used at home. Rendering them irremovable significantly improves healing time and compares them favourably to total contact casting. (96;97) Rearfoot problems are particularly difficult to manage and orthoses, which off load pressures in this area, have been designed. (98)

9. Recommendations
Practical recommendations for the development of an integrated Diabetic foot program can be divided into preventative and management phases of care.

9.1. Prevention, High Risk Assessment and Early Intervention
1. All diabetics should be regularly assessed in an outpatient multi-disciplinary diabetic clinic.
2. Education concerning footcare and footwear should be offered to all diabetics.
3. Systematic, routine examination of the feet of all diabetics should be offered at each visit including neurologic testing with SWM and assessment of vascular status.
4. The University of Texas Diabetic Foot Classification should be used to identify patients at high risk for ulceration and appropriate monitoring and footwear should be prescribed.
5. Glycolated Hb should be measured 3 monthly on all diabetics and every attempt made to lower HbA1c<9%.
6. Patients with impalpable pedal pulses should be referred for noninvasive vascular assessment.
7. Patients with early signs of ulceration and infection should be promptly treated with antibiotics, debridement and off-loading devices.

9.2. Inpatient Management of the Diabetic Foot
1. All ulcers should be classified into the University of Texas Diabetic Wound Classification. The healing of wounds should be charted by measurement.
2. Infected wounds should be classified as Grade 2–4 and antibiotic treatment appropriate for Grade instituted. Presence or absence of osteomyelitis should be determined.
3. The medical condition of the patient should be assessed and hyperglycemia controlled with insulin.
4. Vascular status should be assessed non-invasively and vascular consultation sought if
foot lesions or forefoot amputations are considered unlikely to heal due to arterial insufficiency.

5. All infected, gangrenous or necrotic tissue should be excised leaving a well vascularized wound.

6. Appropriate wound dressings should be used which absorb exudates but keep the wound moist.

7. An appropriate orthotic device which off-loads pressure should be applied when the patient is stable and the patient discharged using this.

8. All patients with healed ulcers should be closely monitored and instructed to wear therapeutic footwear.

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Reference List

(2) Veves AGJMLFW. The Diabetic Foot Medical and Surgical Management. Humana Press, 2002.
(5) Cavanagh PR, Ulbrecht JS, Caputo GM. New developments in the biomechanics of the diabetic foot. [Review] [47 refs]. Diabetes/Metabolism Research Reviews 16 Suppl 1:S6-S10, 2000; Oct. 66 kb
Ankle Surgeons. [Review] [248 refs]. Journal of Foot & Ankle Surgery 2006; 45(Suppl):S1-S68. 4445 kb


(10) WHO/IDF. Definitions and Diagnosis of Diabetes and Intermediate Hyperglycemia. 2006. World Health Organization. Ref Type: Pamphlet 1572 kb


(23) Abbas ZG, Archibald LK. Epidemiology of the diabetic foot in Africa. [Review] [80 refs]. Medical Science Monitor 11(8):RA262-70, 2005, 324 kb
(28) Bloomgarden ZT. Diabetes complications. [Review] [57 refs]. Diabetes Care 27(6):1506-14, 2004, 104 kb
(40) Unachukwu CN, Obunge OK, Odia OJ. The bacteriology of diabetic foot ulcers in
(48) Boike AM, Hall JO. A practical guide for examining and treating the diabetic foot. [Review] [1 refs]. Cleveland Clinic Journal of Medicine 69(4):342-8, 2002. 371 kb
(56) Brooks Beal. TBI or not TBI: That is the question. Diabetic Medicine 2001; 18:528-532. 100 kb


(61) Boutayeb A. The double burden of communicable and non-communicable diseases in developing countries. [Review] [56 refs]. Transactions of the Royal Society of Tropical Medicine & Hygiene 100(3):191-9, 2006. 168 kb


(64) Diabetes Care and Education Committee. Approach to the Management of Diabetes Mellitus. 6th. 2005. Toronto, Banting and Best Diabetes Centre. Ref Type: Pamphlet 1346 kb

(65) Bloomgarden ZT. Glycemic treatment: Control of glycemia. [Review] [40 refs]. Diabetes Care 27(5):1227-34, 2004. 100 kb


(68) Spencer S. Pressure relieving interventions for preventing and treating diabetic foot ulcers. [Review] [30 refs]. Cochrane Database of Systematic Reviews (3):CD002302, 2000. 291 kb


(70) Patout CA, Jr., Birke JA, Wilbright WA, Coleman WC, Mathews RE. A decision pathway for the staged management of foot problems in diabetes mellitus. [Review] [42 refs]. Archives of Physical Medicine & Rehabilitation 82(12):1724-8, 2001. 100 kb


(82) Tisi PV, Callam MJ. Type of incision for below knee amputation. [Review] [22 refs]. Cochrane Database of Systematic Reviews (1):CD003749, 2004. 571 kb


(87) Smith J. Debridement of diabetic foot ulcers. [see comment]. [Review] [48 refs]. Cochrane Database of Systematic Reviews (4):CD003556, 2002. 415 kb


(94) McGuire JB. Pressure redistribution strategies for the diabetic or at-risk foot: Part II. [Review] [12 refs]. Advances in Skin & Wound Care 1919;quiz-9. 3571 kb
(95) Birke JA. The Effectiveness of an Accomodative Dressing in Off-loading Pressure Over Areas of Previous Metatarsal Head Ulceration. Wounds 2003; 15(2):33-39. 726 kb
(99) Ogbera AO, Fasanmade O, Ohwovoriole AE, Adediran O. An Assessment of the Disease Burden of Foot Ulcers in Patients With Diabetes Mellitus Attending a Teaching Hospital in Lagos, Nigeria. Lower Extremity Wounds 5(4);2006 pp. 244-249. 68 kb

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