

Atlas of the Diabetic Foot.
N. Katsilambros, E. Dounis, P. Tsapogas and N. Tentolouris
Copyright © 2003 John Wiley & Sons, Ltd.
ISBN: 0-471-48673-6

Atlas of the Diabetic Foot

Atlas of the Diabetic Foot

Professor Nicholas Katsilambros, MD

*Director of the 1st Department of Propaedeutic Medicine and the Diabetic Centre
Athens University Medical School
Laiko General Hospital
Athens, Greece*

Eleftherios Dounis, MD, FACS

*Director of the Orthopedic Department
Laiko General Hospital
Athens, Greece*

Panagiotis Tsapogas, MD

*Senior Registrar in Internal Medicine and Diabetes
Laiko General Hospital
Athens, Greece*

Nicholas Tentolouris, MD

*Senior Registrar in Internal Medicine and Diabetes
Laiko General Hospital
Athens, Greece*



JOHN WILEY & SONS, LTD

Copyright © 2003

John Wiley & Sons Ltd, The Atrium, Southern Gate, Chichester,
West Sussex PO19 8SQ, England

Telephone (+44) 1243 779777

Email (for orders and customer service enquiries): cs-books@wiley.co.uk

Visit our Home Page on www.wileyeurope.com or www.wiley.com

All Rights Reserved. No part of this publication may be reproduced, stored in a retrieval system or transmitted in any form or by any means, electronic, mechanical, photocopying, recording, scanning or otherwise, except under the terms of the Copyright, Designs and Patents Act 1988 or under the terms of a licence issued by the Copyright Licensing Agency Ltd, 90 Tottenham Court Road, London W1T 4LP, UK, without the permission in writing of the Publisher. Requests to the Publisher should be addressed to the Permissions Department, John Wiley & Sons Ltd, The Atrium, Southern Gate, Chichester, West Sussex PO19 8SQ, England, or emailed to permreq@wiley.co.uk, or faxed to (+44) 1243 770620.

This publication is designed to provide accurate and authoritative information in regard to the subject matter covered. It is sold on the understanding that the Publisher is not engaged in rendering professional services. If professional advice or other expert assistance is required, the services of a competent professional should be sought.

Other Wiley Editorial Offices

John Wiley & Sons Inc., 111 River Street, Hoboken, NJ 07030, USA

Jossey-Bass, 989 Market Street, San Francisco, CA 94103-1741, USA

Wiley-VCH Verlag GmbH, Boschstr. 12, D-69469 Weinheim, Germany

John Wiley & Sons Australia Ltd, 33 Park Road, Milton, Queensland 4064, Australia

John Wiley & Sons (Asia) Pte Ltd, 2 Clementi Loop #02-01, Jin Xing Distripark,
Singapore 129809

John Wiley & Sons Canada Ltd, 22 Worcester Road, Etobicoke, Ontario, Canada M9W 1L1

Wiley also publishes its books in a variety of electronic formats. Some content that appears in print may not be available in electronic books.

British Library Cataloguing in Publication Data

A catalogue record for this book is available from the British Library

ISBN 0-471-486736

Typeset in 10/12pt Times by Laserwords Private Limited, Chennai, India
Printed and bound in Italy

This book is printed on acid-free paper responsibly manufactured from sustainable forestry in which at least two trees are planted for each one used for paper production.

Contents

<i>Preface</i>	<i>vii</i>
<i>Acknowledgments</i>	<i>ix</i>
Chapter I Who is the Patient at Risk for Foot Ulceration?	1
Chapter II Classification, Prevention and Treatment of Foot Ulcers	23
Chapter III Anatomical Risk Factors for Diabetic Foot Ulceration	41
Chapter IV Some Uncommon Conditions	73
Chapter V Neuropathic Ulcers at Various Sites	85
Chapter VI Neuro-Ischemic Ulcers at Various Sites	105
Chapter VII Gangrene	125
Chapter VIII Infections	151
Chapter IX Neuro-Osteoarthropathy. The Charcot Foot	185
Appendix 1 Anatomy of the Foot	213
Appendix 2 Manufacturers of Preventive and Therapeutic Footwear	217
<i>Index</i>	<i>221</i>

Preface

Diabetes mellitus is a common disease all over the world and its frequency is steadily increasing. The availability of a wide variety of treatment options results in improvement or even normalization of hyperglycemia as well as of the accompanying metabolic disorders. However people with diabetes continue to suffer from the complications of the disease.

Diabetic foot-related problems occur frequently and may have serious consequences. Amputations at different anatomical levels are the most serious of them.

The present *Atlas* represents a systematic description of the many different foot lesions, which are often seen in diabetic patients. Each figure corresponds to a case treated in our Diabetes Centre at the Athens University Medical School. Our patients are evaluated and treated in collaboration with the Orthopedic Department as well as with other specialists depending upon individual needs. A short text, which follows each illustration, describes the history of the patient, the physical signs observed, the approach to treatment, and is followed by a short comment.

It is hoped that this *Atlas* will be of assistance, as a reference guide and a teaching instrument, not only to diabetologists and surgeons, but also to all doctors involved in the treatment of diabetic patients. This book may help them not only to recognize and to treat the diabetic foot lesions, but also to prevent them.

On behalf of the authors
N. Katsilambros

Acknowledgments

The authors of this *Atlas* would like to express their thanks and gratitude to Elias Bastounis, Professor of Surgery and Christos Liapis, Associate Professor, both of whom are vascular surgeons, as well as to Othon Papadopoulos, Assistant Professor, who is a plastic surgeon and to all the academics in the University of Athens for their help with certain cases in which they are specialists. The help of Constantine Revenas, radiologist and Associate Director in Laiko General Hospital, in the field of ultrasonography is also gratefully acknowledged.

The authors would also like to express their sincere gratitude to nurse Georgia Markou, who is indispensable to the Outpatient Diabetic Foot Clinic, for her meticulous attention to the efficient functioning of the clinic and to the upkeep of patient records.

Thanks are also due the numerous doctors who have assisted the Outpatient Diabetic Foot Clinic either as specialists in infectious diseases or orthopedics or as scholars in the field of diabetes and the diabetic foot.

Chapter I

WHO IS THE PATIENT AT RISK FOR FOOT ULCERATION?

- INTRODUCTION
- WHICH PATIENTS ARE AT RISK
FOR FOOT ULCERATION?
- DIABETIC NEUROPATHY
- PERIPHERAL VASCULAR DISEASE
- BIBLIOGRAPHY

INTRODUCTION

The prevalence of foot ulceration in the general diabetic population is 4–10%, being lower (1.5–3.5%) in young and highest (5–10%) in older patients. The lifetime risk for foot ulcers in diabetic patients is about 15%. The major adverse outcome of foot ulceration is amputation. Data from several studies have documented that foot ulcers precede approximately 85% of all amputations performed in patients with diabetes. Risk of ulceration and amputation increases 2- to 4-fold with both age and duration of diabetes. According to one report, prevalence of amputations in diabetic patients is 1.6% in the age range 18–44 years, 3.4% among those aged 45–64 years, and 3.6% in patients older than 65 years. Incidence of lower extremity amputations in the United States was 9.8 per 1000 patients with diabetes in 1996, increasing by 26% from 1990, despite efforts to reduce these rates. Data from other countries confirm the increase of amputation rates worldwide. This may be due to aging of the diabetic population, and better reporting. As the diabetic population increases, more amputations are expected in the future.

Foot ulceration and amputation affect the quality of life for patients and create an economic burden for both the patient and the health care system. Therefore, efforts to identify the patient who is at risk for foot ulceration, prevention and appropriate treatment must, of necessity, become a major priority for healthcare providers.

WHICH PATIENTS ARE AT RISK FOR FOOT ULCERATION?

Risk factors for foot ulceration are as follows.

- History of previous foot ulceration or amputation
- Peripheral neuropathy
- Peripheral vascular disease
- Trauma (poor footwear, walking barefoot, objects inside the shoes)
- Foot deformities (prominent metatarsal heads, claw toe, hammer toe, pes cavus, nail deformities, deformities related to previous trauma and surgery, bony prominences, etc.)
- Callus formation
- Neuro-osteoarthropathy
- Limited joint mobility
- Long duration of diabetes
- Poor diabetes control

In addition to these well-recognized risk factors for foot ulceration, several—but not all—studies have shown that foot ulcers are more common in male patients. In addition, social factors including low social status, poor access to healthcare services, poor education and a solitary lifestyle have all been associated with foot ulceration. Another important factor for foot ulceration is poor compliance by the patient with medical instructions and neglecting to follow procedures. Edema may impair blood supply to the foot, particularly in patients with peripheral vascular disease. Inhibition of sweating (anhidrosis)—due to peripheral neuropathy—may cause dry skin and fissures. Dry skin together with limited joint mobility and high plantar pressures contribute to callus formation.

Peripheral neuropathy and vascular disease alone do not cause foot ulceration. It is the combination of the factors mentioned above, that act together in the vast majority of cases. Trauma from either the patient's shoes or from external causes, and loss of protective sensation and peripheral vascular disease are among the major contributors to foot ulceration. Diabetic neuropathy is

the common denominator in almost 90% of diabetic foot ulcers. Trauma initially causes minor injuries, which are not perceived by the patient with loss of protective sensation. As the patient continues his activities, a small injury enlarges and may be complicated by infection. The pathway to foot ulceration in diabetes is depicted in Figure 1.1.

DIABETIC NEUROPATHY

Diabetic neuropathy is defined—according to the International Consensus Group on Neuropathy—as ‘the presence of symptoms and/or signs of peripheral nerve dysfunction in people with diabetes, after exclusion of other causes’. The prevalence

of peripheral neuropathy in diabetes is 23–42% and is higher (50–60%) among older type 2 diabetic patients. It should be mentioned that the prevalence of symptomatic peripheral neuropathy (burning sensation, pins and needles or allodynia in the feet, shooting, sharp and stabbing pain or muscle cramps at the legs) is only 15–20% and the majority of the patients with neuropathy are free of symptoms. Often, the first sign of peripheral neuropathy is a neuropathic ulcer. Other patients have neuropathic pain and on examination are found to have severe loss of sensation. This combination is described as ‘painful-painless legs’ and these patients are at increased risk for foot ulceration.

All patients with diabetes should be examined annually for peripheral neuropathy,

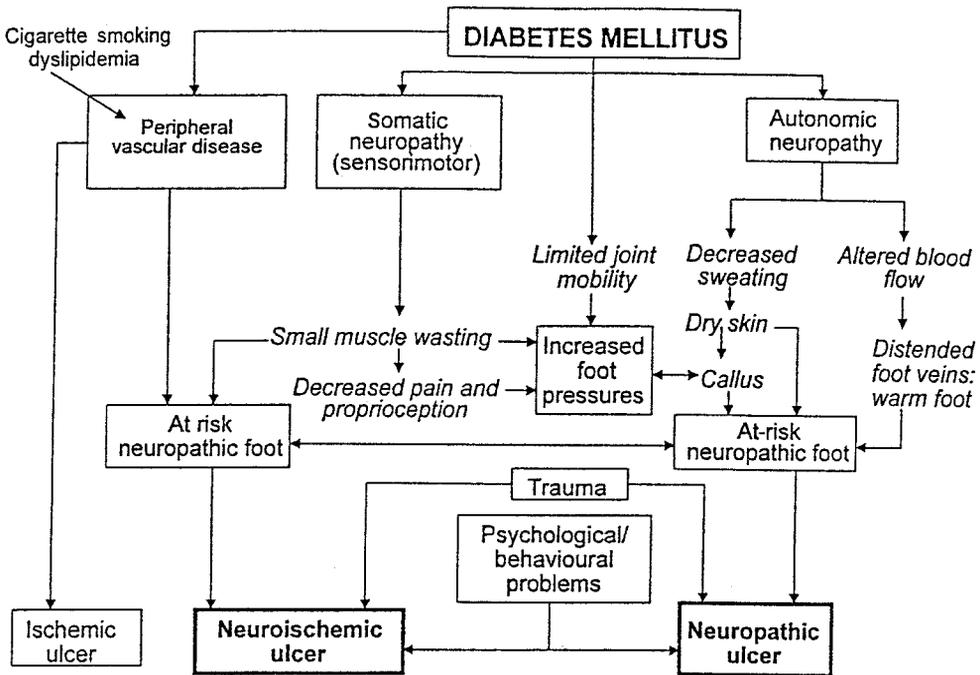


Figure 1.1 Pathways to foot ulceration in diabetic patients. (From Boulton AJM. The pathway to ulceration: Aetiopathogenesis. In Boulton AJM, Connor H, Cavanagh PR (Eds), *The Foot in Diabetes* (3rd edn). Chichester: Wiley, 2000; 61–72, with permission)

so that those at risk for ulceration can be identified. The tests for peripheral neuropathy are many and some of them are quite sophisticated, and are undertaken only in specialist centers. However, the tests that are used to characterize the patient with loss of protective sensation are simple, fast and easily carried out at the outpatient clinic. These tests are as follows.

1. Questioning the patient to ascertain whether symptoms of peripheral neuropathy, as described above, are present. Typically neuropathic symptoms are worse during the night and may wake the patient, who finds relief on walking.
2. Loss of sensation of (a) *pain* (using a disposable pin; this test is carried out only when the skin is intact), (b) *light touch* (using a cotton wisp), and (c) *temperature* (using two metal rods, one at a temperature of 4°C and the other at 40°C) on the dorsum of the feet. Typically, in diabetic peripheral neuropathy the sensory deficit is pronounced at the periphery of the extremities (in a 'glove and stocking distribution'). A zone of hypoesthesia is found between the area of loss of sensation and a more central area of normal sensation. Achilles tendon reflexes may be reduced or absent. *Wasting of small muscles* of the feet results in toe deformities (claw, hammer, curly toes) and prominent metatarsal heads. *Vibration perception* is tested using a 128-Hz tuning fork on the dorsal side of the distal phalanx of the great toes (Figure 1.2). A tuning fork should be placed perpendicular to the foot at a constant pressure. During examination the patient is prevented from seeing where the examiner has placed the tuning fork. Examination is repeated twice and there is at least one 'sham' application in which the tuning fork is not vibrating. The patient has



Figure 1.2 Examination of vibration perception by the use of tuning fork

normal sensation when his reactions are correct in two out of three tests, but is at risk for ulceration when they are incorrect in two out of the three tests.

3. Pressure perception is tested with Semmes–Weinstein monofilaments. Many studies have shown that inability to perceive pressure is related to a several-fold increase in the risk for foot ulceration. The filaments are available in large sets with varying levels of force required to bend them. Diabetic neuropathy can be detected using the 5.07 monofilament (this filament bends with the application



Figure 1.3 Semmes–Weinstein (5.07) monofilament examination

of a 10-g force). Monofilament should be applied perpendicular to the skin surface and with sufficient force so that it bends or buckles (Figure 1.3). Total duration of skin contact of the filament should be approximately 2 s. During examination the patient is prevented from seeing if and where the examiner applies the filament. The patient is asked to say whether he can feel the pressure applied (yes/no) and in which foot (right/left foot). Examination is repeated twice at the same site and there is at least one ‘sham’ application, in which no filament is applied (a total of three questions per site). The patient has normal protective sensation when the correct answer is given for two out of the three tests and is at risk for ulceration when they are not. The International Consensus on the Diabetic Foot suggested three sites to be tested on both feet: the plantar aspect of the great toe, the first and the fifth metatarsal heads. The filament must be applied at the perimeter and not at an ulcer site, callus, scar or site of necrotic tissue.

4. Determination of vibration perception thresholds using a biothesiometer or a neurothesiometer. Vibration perception threshold is measured at the tip of the great toes with the vibrating head of the device balanced under its own weight (Figure 1.4). The vibrating stimulus is increased until the patient feels it, the stimulus is then withdrawn and the test repeated. This test is usually carried out three times at each site and the mean value is calculated. Several studies have shown that a vibration perception threshold over 25 V is associated with a 4- to 7-fold increase in risk for foot ulceration.

PERIPHERAL VASCULAR DISEASE

ASSESSMENT OF THE VASCULAR STATUS IN PATIENTS WITH DIABETES

The prevalence of peripheral vascular disease in diabetic patients is 15–30%. The

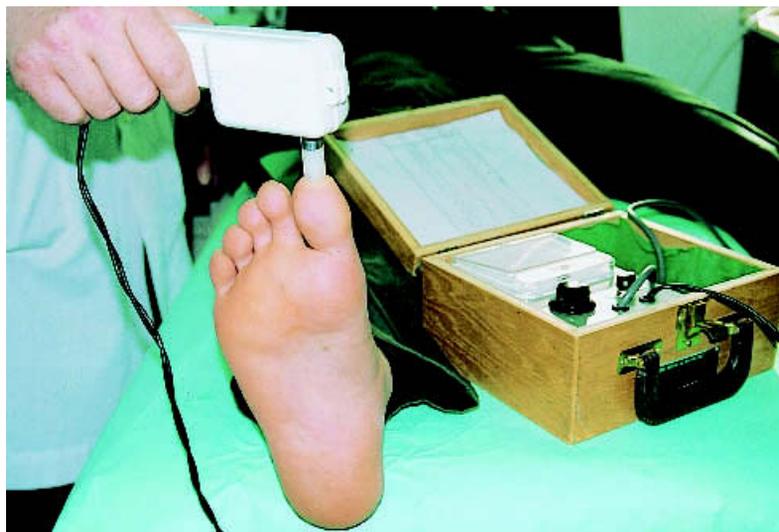


Figure 1.4 Examination of vibration perception by the use of a biothesiometer

disease progresses with both duration of diabetes and age. A diagnostic work-up of the peripheral extremities is based on clinical examination (history of intermittent claudication, rest pain, walking distance, palpation of leg pulses, and measurement of ankle brachial index). Co-existence of neuropathy in diabetic patients might reduce the pain of intermittent claudication or even ischemic rest pain. Palpation of feet pulses remains the cornerstone of screening for peripheral vascular disease. The absence of two or more pulses on both feet is diagnostic of peripheral vascular disease. Based on the results of clinical examination, a decision must be made as to whether the doctor will proceed with more sophisticated methods of examination of the lower extremities in order to determine the exact level and degree of the arterial obstruction.

Fontaine Clinical Staging

Fontaine clinical staging of peripheral arterial disease includes four stages:

Stage I is asymptomatic; patients may complain of numbness or that their legs get easily tired, but they do not seek medical help. Usually the superficial femoral artery is stenosed at the level of the Hunterian duct; lateral circulation of the deep femoral artery is adequate for the needs of the limb.

Stage II in which patients suffer from intermittent claudication; they are subclassified as *Stage IIa*, if they can walk without symptoms for more than 250 m; or *Stage IIb*, if they have to stop earlier. If patients feel pain in the leg, it is usually due to occlusion of the femoral artery, while an occlusion of the iliac artery causes pain in the thigh.

Stage III patients suffer from rest pain of the limb, which may become constant and very intense, usually during the night; the pain is often resistant to analgesics. The prognosis is not good; half of these patients will have an amputation within the next 5 years.

Stage IV patients have gangrene. Minor trauma, ulcers or paronychias may evolve

into gangrene when stage III peripheral artery disease is present. The patient feels pain at rest unless diabetic neuropathy is also present.

Noninvasive Vascular Testing

Calculation of the Ankle Brachial Index (ABI). The ankle brachial index (ankle arm index) is widely used and can easily be measured in the outpatient clinic. Measurements are made with the use of a pocket-size continuous-wave Doppler probe operating at 4 or 10 MHz. The brachial systolic pressure on both sides is determined first. Then the ankle systolic pressure on each side is determined with the Doppler probe by applying a blood pressure cuff around the ankle, just above the malleolus. Ankle pressure is measured at both posterior tibial (behind the medial malleolus) and dorsal pedal arteries. No pressure is applied on the probe. Pressures are determined at a beam-vessel angle of approximately 60°. After measuring the systolic pressures, the highest ankle pressure is divided by the highest brachial pressure; this ratio is called the *ankle brachial index* (ABI). Occasionally, no audible signal can be obtained from the foot arteries. In these cases, a careful search often reveals a peroneal collateral signal anteriorly, next to the lateral malleolus. Normally, systolic ankle pressure exceeds systolic arm pressure by 12–24 mmHg. The normal value of the ABI is 1 to 1.2. A level of less than 0.9 is usually taken as indicative of occlusive arterial disease. An ankle systolic pressure of <50 mmHg or an ABI <0.3 in the presence of rest pain or tissue damage denotes critical limb ischemia. The equivalent toe systolic pressure is 30 mmHg or less.

A change of >0.15 in the ABI during follow-up suggests significant narrowing and it is an indication for further study with

angiography. A spontaneous rise in the ABI is usually attributable to the development of collateral circulation.

Medial calcification, which is very common in diabetes (Figure 1.5), renders the underlying arteries incompressible, resulting in spuriously high ABI values (more than 1.2). In these cases, the severity of arterial occlusive disease can be assessed by toe pressure measurements. Other causes of inaccurately high ABI values include too high positioning of the upper body, chronic venous insufficiency and significant ankle edema. Spurious low ABI values can result from the rapid deflation of the cuff, excessive probe pressure, and an insufficient rest period.

Despite these limitations, the ankle brachial index is a useful screening tool for the assessment of presence and severity of peripheral vascular disease and it remains the basic examination suggested by an international panel on the assessment of peripheral vascular disease in diabetes (see below).

Toe Pressures. Toe pressures are measured by a pneumatic cuff with a diameter which



Figure 1.5 Extensive calcification of the posterior tibial artery

is about 1.2 times that of the digit, wrapped around the proximal phalanx, with a flow sensor (usually a photoplethysmograph) located distally (Figure 1.6). In addition, toe pressures can also be measured using a digital strain gauge. Normal toe pressures average 24–40 mmHg or less compared to ankle pressure. Rest pain, skin lesions, or both are present in approximately 50% of limbs with toe pressures ≤ 30 mmHg, and in a much lower proportion of patients with toe pressures above this level. Toe pressures do not differ between patients with and without diabetes. Spuriously high

toe pressures due to arterial calcification seldom occur at the toe level. For this reason, toe pressure determination is valuable in diabetic patients when an ankle pressure is abnormally high.

Transcutaneous Oximetry. Transcutaneous oximetry (measurement of transcutaneous oxygen pressure, TcPO₂) is used for the assessment of severe peripheral vascular disease. It is usually measured at the dorsum of the feet with the patient in the supine position (Figure 1.7). With increasing age, the TcPO₂ tends to decrease,

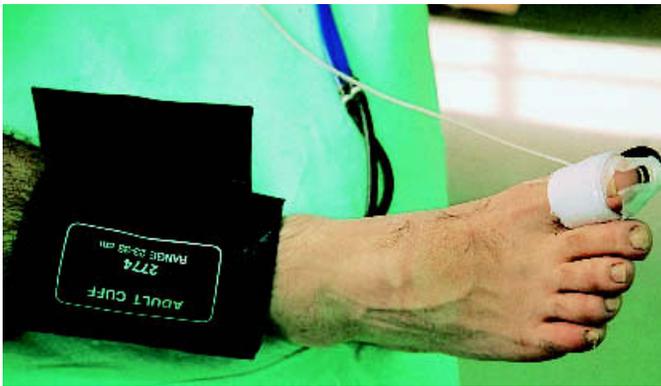


Figure 1.6 Toe and ankle pressure measurement

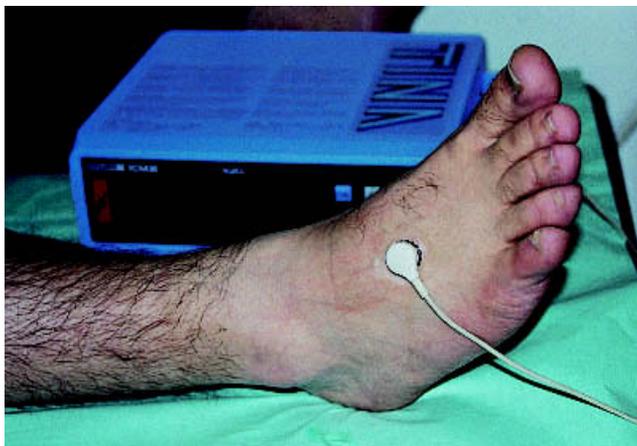


Figure 1.7 Transcutaneous oximetry

paralleling a similar decline in arterial PO_2 . Normal subjects have values of 40 to 70 mmHg. In general, a resting $TcPO_2$ greater than 55 mmHg may be considered normal, regardless of age. Patients with anemia may also have lower values. Patients with rest pain or gangrene have values between 0 and 30 mmHg. In diabetes, $TcPO_2$ is lower than in age-matched arteriopathic patients. A $TcPO_2 < 40$ mmHg is associated with failure of wound healing, while an increase after angioplasty or bypass surgery predicts success of the intervention. Because the results are not affected by arterial calcification, this method is particularly valuable for evaluating diabetic vascular disease.

Segmental Pressures Measurement. Abnormal blood pressure values found by any of the above methods indicate that arterial occlusive disease is present, but they do not identify the specific segments involved. Further diagnostic information can be obtained by measuring pressure gradients in the legs. However, only rarely do these measurements need to be made when the ABI is normal. To determine segmental pressure in the legs, pressure cuffs 10–12 cm wide are applied around the thigh at the groin level, above the knee, below the knee and at the ankle level (Figure 1.8). By listening with a Doppler probe over the pedal arteries (posterior tibial or dorsal pedal), the pressure at the level of the inflated cuff can be measured. A pressure index can be obtained by dividing the segmental systolic pressure by the brachial pressure. The pressure index should be equal to 1.0 or slightly higher. Normal pressure index at the high thigh level is 1.3. The pressure gradient between any two adjacent levels in the normal leg is $< 20\text{--}30$ mmHg. Gradients > 30 mmHg suggest that a significant stenosis is present

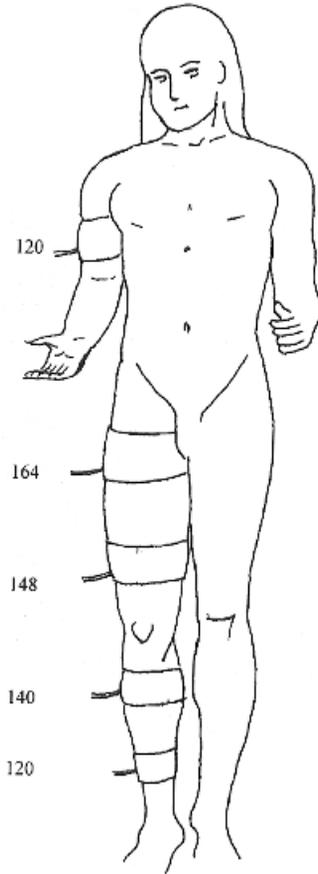


Figure 1.8 Normal segmental pressures. The pressure gradient between any two adjacent levels in the normal leg is less than 20–30 mmHg. Gradients greater than 30 mmHg suggest that a significant stenosis is present at the intervening arterial segment. When the gradient exceeds 40 mmHg the artery is occluded

at the intervening arterial segment. When the gradient exceeds 40 mmHg the artery is occluded.

It should be taken into account that patients with severe stenosis at a proximal level (e.g. aortoiliac disease) may have spuriously normal pressure gradients between high thigh and low thigh in the presence of severe superficial femoral artery stenosis.

In addition, obstructions below the knee may not be diagnosed, unless the stenosis is sufficiently severe to involve all three tibial arteries.

Segmental Plethysmography. Plethysmography is a useful technique for the assessment of peripheral arteries. There are several types of plethysmographs (air, mercury, indium–gallium and strain gauge plethysmographs) and all measure the same parameter: the momentary change in the volume of the soft tissues when a pulse wave fills the arteries of the area of the leg which is being examined. Photoplethysmography measures blood concentration in the cutaneous microcirculation by detecting the reflection of the applied infrared light. Air plethysmographs are the standard instruments for segmental plethysmography. Pressure cuffs are applied at different levels of the leg as in segmental pressure measurement. A plethysmograph records the change in volume as a wave, which reflects the intra-arterial changes. The normal segmental volume pulse contour is characterized by a steep, almost vertical

upstroke, a sharp systolic peak, and a down slope that bows towards baseline during diastole. In the middle of the down slope there is a prominent dicrotic wave. Distal to a stenosis, the upslope is less steep, the peak becomes rounded, the down slope bows away from the baseline, and the dicrotic wave disappears. Examples of various degree of arterial stenosis are shown in [Figure 1.9](#). A plethysmography record is not affected by the presence of arterial calcification; for this reason it is a valuable method for the assessment of peripheral vascular disease in diabetes.

Ultrasonography. Arterial ultrasound examination has become very popular in recent years. It is a simple, low cost and valid method for determination of the site and degree of obstructive lesions, and of the patency of a vessel after revascularization. The site of an arterial stenosis can be identified by serial placements of the Doppler probe along the extremities. However, there is no justification for its use as a routine screening procedure. The exact site of arterial disease is located by the

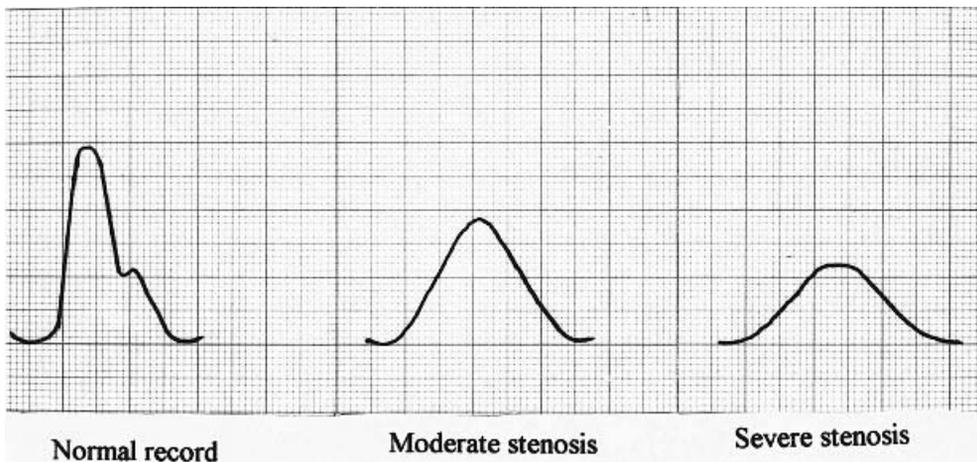


Figure 1.9 Plethysmography pulse volume waveforms associated with different degrees of peripheral vascular disease

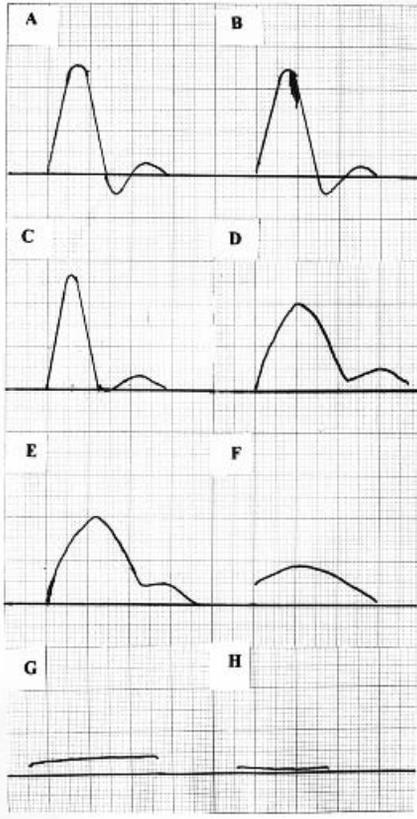


Figure 1.10 Qualitative analysis of spectral waveforms proximal to the site of the probe. (A) Normal. (B) Mild arterial stenosis causing turbulence during systole. (C and D) Loss of reverse flow due to more severe stenosis. (E and F) As the degree of stenosis increases, the rate of acceleration of the upstroke decreases, the peak becomes rounded (E) and the wave becomes continuous and less pulsatile (F–H). Completely damped waveforms (F–H) in the pedal arteries are compatible with multilevel vessel disease and indicate the presence of blood flow due to the development of collateral circulation

use of duplex scanning. Duplex scanners use the combination of real time B-mode ultrasound imaging of the arterial wall, together with pulsed Doppler imaging and examine flow patterns in a defined area

within the artery lumen. Pulsed Doppler imaging produces spectral analysis of the pulse wave which delineates the complete spectrum of frequencies (that is, blood flow velocities) found in the arterial waveform during a single cardiac cycle. Tissues are displayed in varying shades of gray (duplex) on the screen. The addition of color frequency mapping (color duplex or triplex) makes identification of arterial stenosis easier and allows a better description of the atheromatous plaques on the arterial wall. A normal spectrum shows a typical triphasic flow pattern, consisting of a steep systolic upstroke, a systolic peak, a reverse flow component in early diastole and a pre-systolic zero flow (Figure 1.10). A clear spectral window under the systolic peak is a normal finding, signaling the absence of slow turbulent flow components. If a stenosis is present the window becomes occluded. The degree of stenosis can be quantified by analyzing the spectral waveform, and by determining the peak systolic velocity ratio (PSV ratio). In general, a cross-sectional reduction of at least 30% must be present to produce a detectable spectral change. The flow velocities may vary, but peak systolic velocities in the arteries above knee are about 50–100 cm/s, while below knee they are approximately 50 cm/s.

Qualitative analysis of the waveform. Inspecting the contour of the spectral waveform is of considerable diagnostic value.

Table 1.1 Peak systolic velocity ratio (PSV ratio) for the determination of the degree of stenosis

PSV ratio	Reduction in cross-sectional area
<2.5	0–49%
>2.5	50–74%
>5.5	75–99%

Atherosclerotic disease proximal to the site of the probe produces a subtle change in the contour of the systolic peak or in the early deceleration phase (Figure 1.10). With increasing proximal stenosis, the

reverse flow component is damped and then disappears entirely.

Quantitative analysis of the waveform.

The most widely used criterion for

Table 1.2 Criteria for lower limb arterial stenosis in spectral analysis

Percentage stenosis	Pre-stenotic spectrum	Intra-stenotic spectrum	Spectrum just past the stenosis
0–50%	Normal: — Triphasic or biphasic — Narrow frequency band — Clear spectral window	Increase in PSV (by <100% and/or <180 cm/s)	No significant turbulence Possible flow reversal
51–75%	Normal	Increase in PSV (by >100% and/or >180 cm/s)	Flow reversal Possible slight turbulence
76–99%	Normal or slightly reduced velocity	Increase in PSV (by >250% and/or >180 cm/s)	Significant turbulence Complete occlusion of spectral window

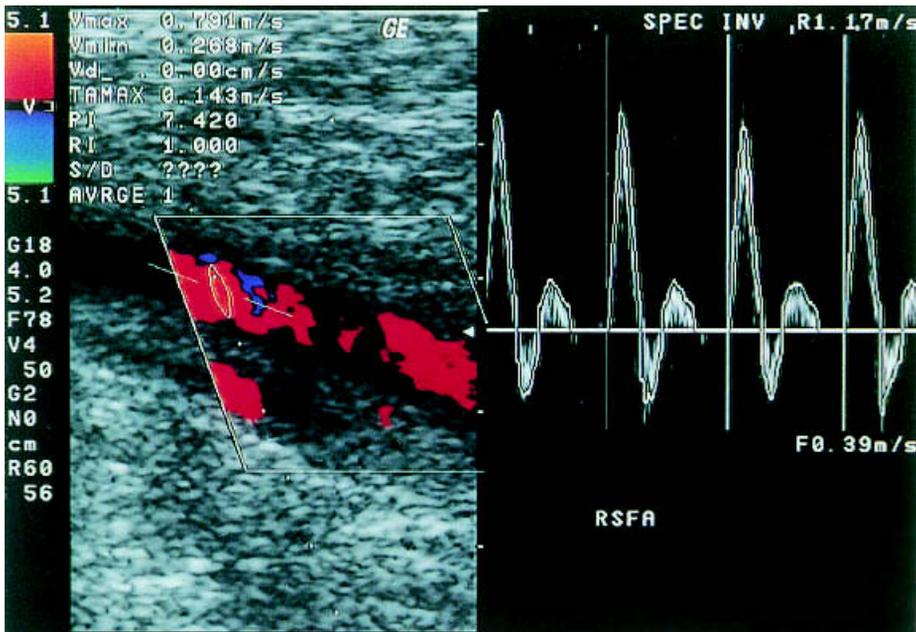


Figure 1.11 Normal triphasic spectral waveform from the right superficial femoral artery. Note the narrow, steep increase and decrease of the waveform. Peak systolic velocity is 79.1 cm/s (normal peak systolic velocities in the arteries above knee are 50–100 cm/s). (Courtesy of C. Revenas)

diagnosis of peripheral arterial stenosis is the peak systolic velocity ratio. This ratio expresses the relationship of the intra-stenotic peak systolic velocity to the lowest post-stenotic or pre-stenotic peak systolic velocity. The PSV ratio allows estimation of the degree of a stenosis without distortion by a second stenosis located at a more distal or a proximal site (Table 1.1). Other criteria used for the estimation of arterial stenosis are presented in Table 1.2.

Duplex ultrasonography has a sensitivity of 80% and specificity above 90% for detecting femoral and popliteal stenosis compared with angiography, but it is less reliable for the assessment of the severity of stenosis in the tibial and peroneal arteries.

Normal and abnormal spectral waveform recordings are shown in Figures 1.11–1.18.

Other Methods. Modern methods for the assessment of peripheral arteries include helical or spiral computed tomography (CT) and magnetic resonance angiography. Spiral CT has the ability to generate three-dimensional images and is most useful in the evaluation of large arteries (e.g. thoracic or abdominal aorta). Disadvantages include intravascular administration of iodinated contrast material and the inability to assess small vessel disease. Magnetic resonance angiography (MRA) is mainly used for examining the cerebral vessels and the carotid arteries. Recent data suggest that this method might replace angiography as a primary imaging examination for

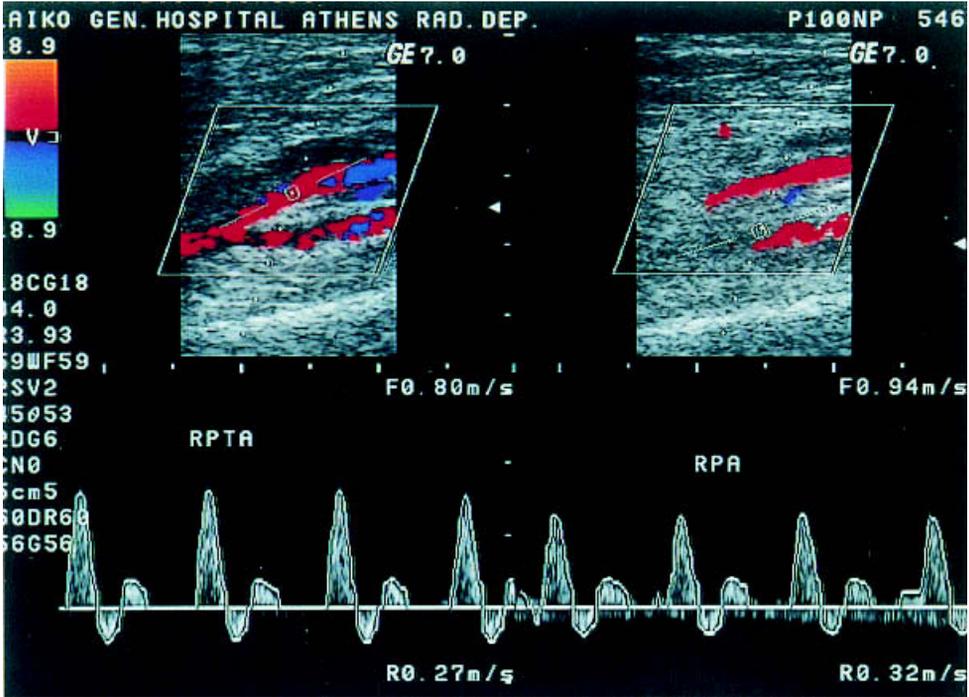


Figure 1.12 Normal triphasic spectral waveform from the right posterior tibial artery. At the top of the figure the duplex scan of the artery is seen. Peak systolic velocity is 49 cm/s. (Courtesy of C. Revenas)

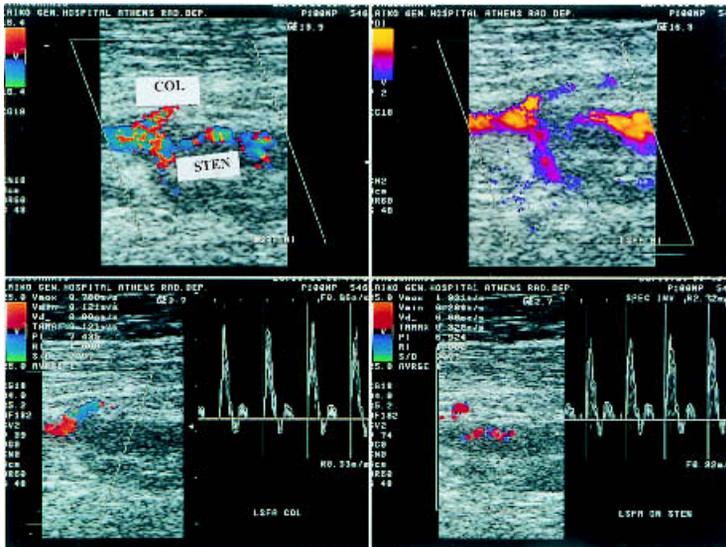


Figure 1.13 In the left upper panel a significant stenosis (STEN) of the left superficial femoral artery with collateral circulation development (COL) is shown. Note (left lower panel) the triphasic spectral waveform in the collateral vessel and that the peak systolic velocity is 78 cm/s, which is too high for such a vessel. In the area of the femoral artery stenosis, the peak systolic velocity is high (193 cm/s) and the waveform is triphasic, but blood flow during diastole is low, as seen from the short duration of the reverse flow (right lower panel). Adjacent to the spectral waveform, a color duplex scan of the artery with the stenosis is shown. These findings suggest the presence of stenosis of approximately 50–80%. In the upper right panel a dynamic Doppler recording is shown, which gives a clearer image of the collateral vessels. (Courtesy of C. Revenas)

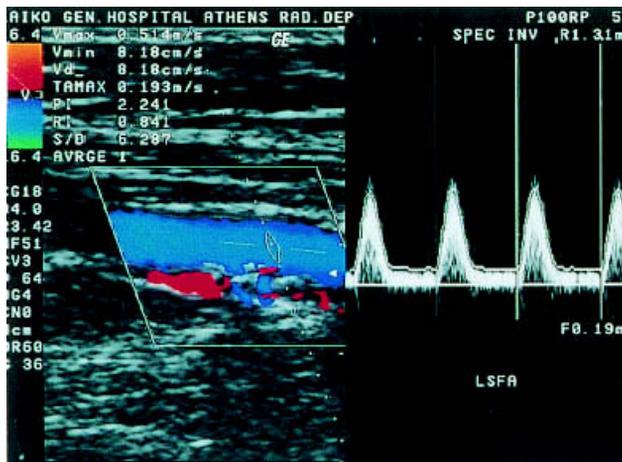


Figure 1.14 Biphasic spectral waveforms obtained from the left superficial femoral artery. The spectral window is widened and is filled in, although not completely. Peak systolic velocity is low (51.4 cm/s). These findings indicate the presence of significant proximal stenosis at one or multiple levels. (Courtesy of C. Revenas)

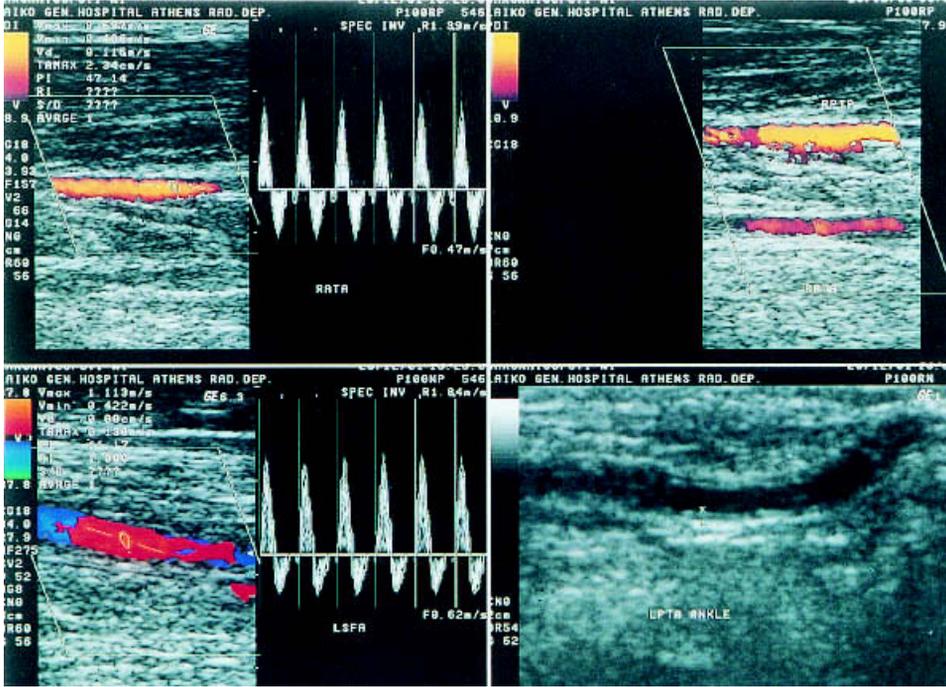


Figure 1.15 The spectral waveform from the right anterior tibial artery in an area of stenosis is seen in the upper left panel. Peak systolic velocity is high (69.7 cm/s)—peak systolic velocities in the arteries below knee are normally ~50 cm/s—and there is mild widening of the spectrum during both systole and diastole. This record corresponds to a stenosis of about 30%. The spectral waveform from the left superficial femoral artery is shown in the lower left panel. There is mild spectral widening, and loss of pre-systolic flow. The color duplex image of the right tibial arteries is shown in the right upper panel. A duplex scan of the left posterior tibial artery is shown in the lower right panel. (Courtesy of C. Revenas)

peripheral vascular disease. Angiography may be reserved for percutaneous interventions and in cases of equivocal findings only. In addition, an MRA is a simple, non-toxic and relatively inexpensive method.

An International Meeting on the Assessment of Peripheral Vascular Disease in Diabetes was held in 1993 and made the following recommendations for the detection and follow-up of peripheral vascular disease.

- All adults (age \geq 18 years) with diabetes should be asked whether they suffer from intermittent claudication. Presence

of claudication is an indication for ankle brachial index (ABI) determination on an annual basis. If the ABI is less than 0.9, the patient needs intensive management of cardiovascular risk factors. All patients with lifestyle-limiting claudication should be referred for specialist vascular assessment. Intensive management of cardiovascular risk factors includes reduction of lipid levels, smoking cessation, control of blood pressure, weight and glucose levels and the use of aspirin as in coronary heart disease.

- All adults (age \geq 18 years) with diabetes should be examined annually for signs of

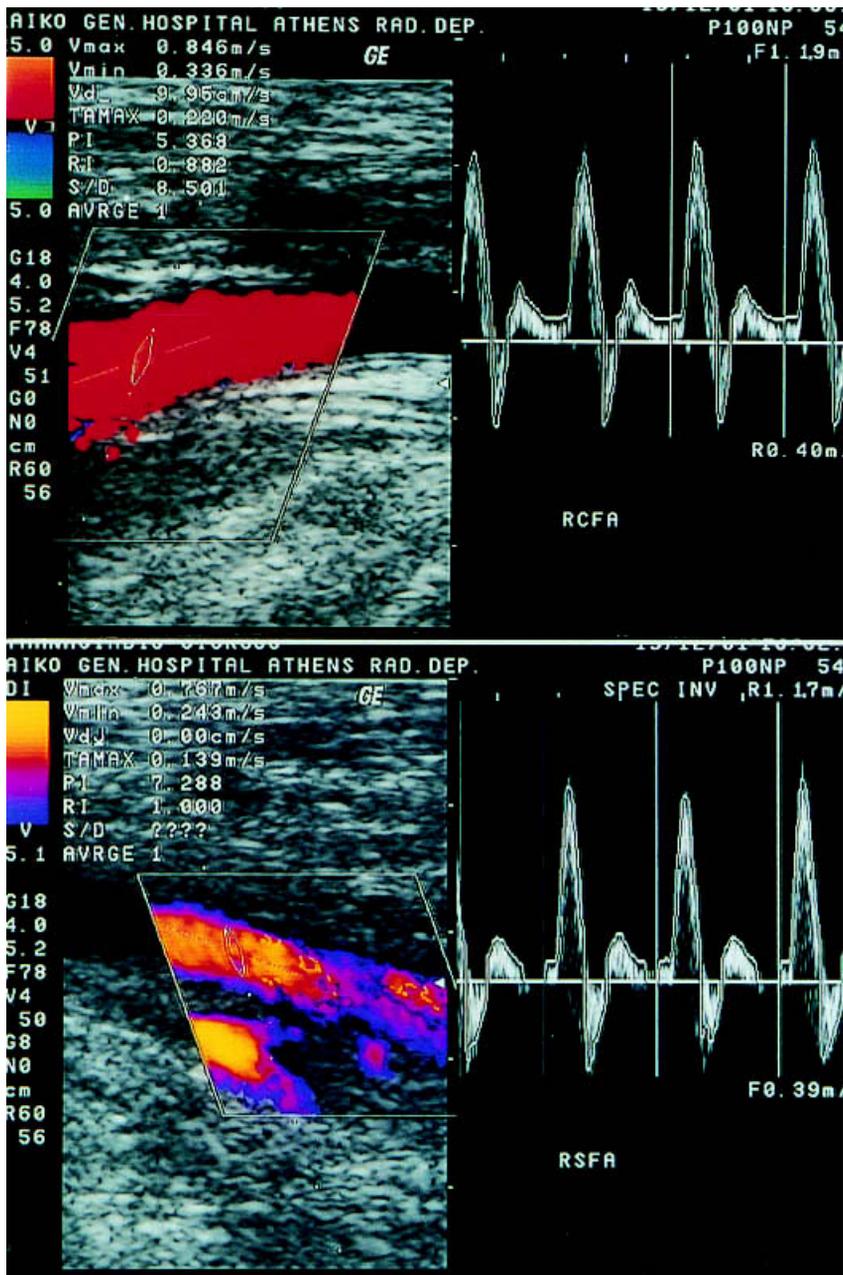


Figure 1.16 Near normal spectral waveforms obtained from the right common (upper panel) and right superficial (lower panel) femoral arteries. The peak systolic velocity is reduced slightly; the waveform is triphasic and there is minimal widening of the spectral window. These findings suggest the presence of a mild proximal stenosis. (Courtesy of C. Revenas)

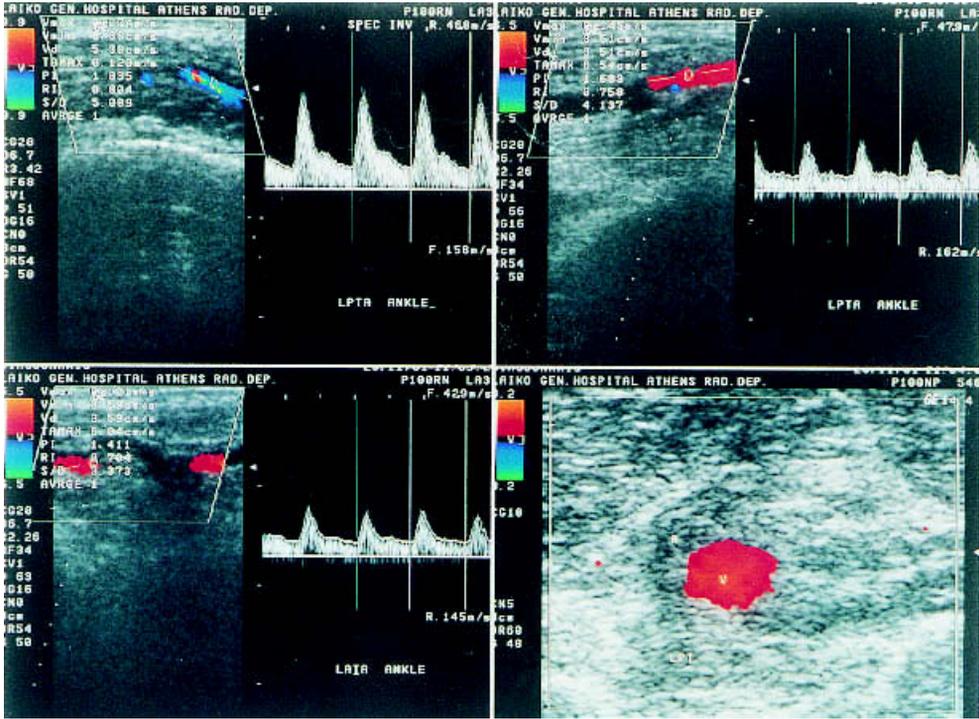


Figure 1.17 Upper left panel: a biphasic waveform of the left posterior tibial artery at ankle level. The peak systolic velocity is reduced (27.4 cm/s) and there is widening of the spectral window during systole, while velocity is high during diastole. The artery diameter is normal as seen in a color duplex image on the left of the spectral waveform. These findings suggest the presence of a proximal stenosis of about 40%. Right upper panel: the same artery at another site after a stenosis. The low peak systolic velocity (14.5 cm/s), biphasic waveform, and spectral widening during systole, as well as the high velocity during diastole are notable features. These findings suggest the presence of a proximal stenosis of more than 50%. Left lower panel: duplex scan of the left anterior tibial artery from the same patient and the recorded spectral waveform. An even lower peak systolic velocity (12.1 cm/s), significant widening of the systolic spectral window and high diastolic velocity are shown. The diameter of the artery is normal (lower right panel). The above findings signify the presence of a proximal stenosis of about 50–60%. (Courtesy of C. Revenas)

critical limb ischemia (gangrene, ulcer, skin changes, or ischemic rest pain). If such signs are present, the patient should be referred for specialist vascular assessment. In addition, intensive management of co-existent cardiovascular risk factors should be initiated.

- Palpation of the dorsalis pedis and posterior tibial artery as well as auscultation for femoral artery bruits should be

performed on an annual basis for all adults with diabetes. If one pedal artery is absent or diminished or if bruits are audible, ABI determinations should be carried out annually. If the ABI value is below 0.9, intensive management of co-existent cardiovascular risk factors should be initiated.

- Patients for whom ABI monitoring is recommended: (a) all those with type 1

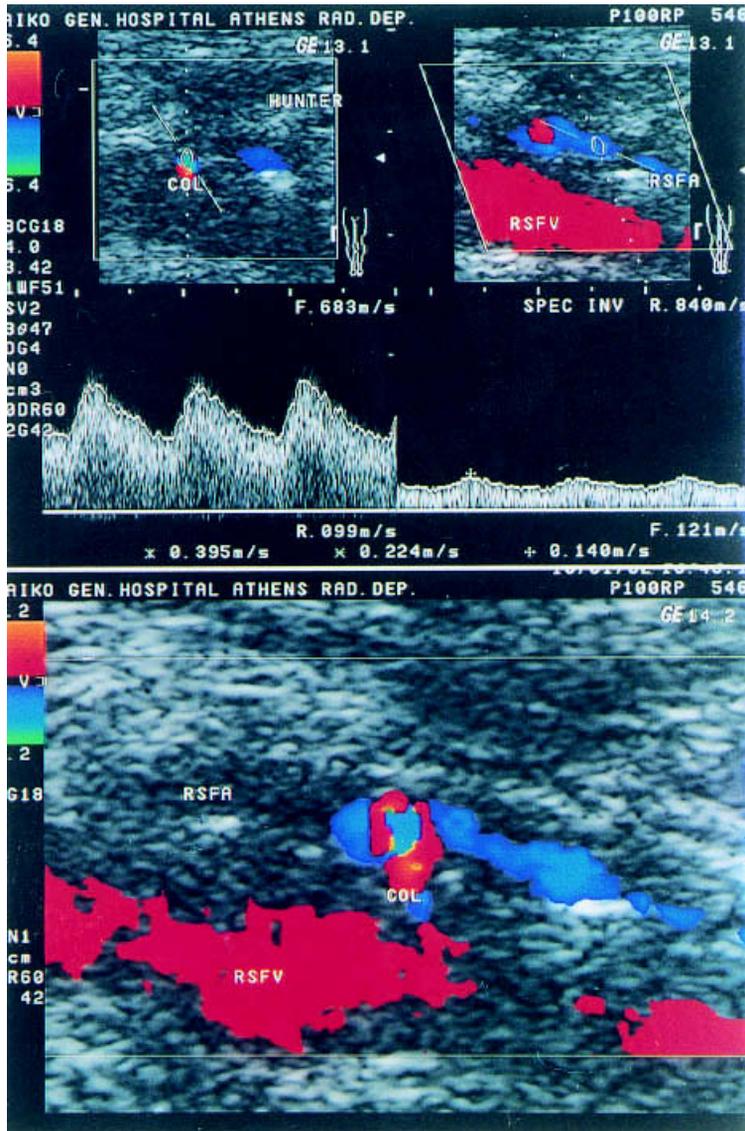


Figure 1.18 Lower panel: complete obstruction of the right superficial femoral artery (RSFA) at the canal of Hunter. A collateral vessel (COL) is seen proximal to the stenosis. Distal to the site of the obstruction there is blood flow in the superficial femoral artery from collateral vessels. Upper panel: the spectral waveform obtained from the collateral vessel shown in the lower panel of the figure. The waveform is biphasic, both peak systolic and diastolic velocities are high and there is widening of the systolic spectral window. The waveform obtained from the right superficial femoral artery distal to the site of the complete obstruction is shown. Notice the low peak systolic and the high diastolic velocity. This waveform is called *tardus parvus*. This type of spectral waveform is similar to that obtained from the venous circulation, and signifies blood flow in an artery resulting from the development of collateral circulation. As more collateral vessels fill the artery, the spectral waveform may be triphasic, but the peak systolic velocity will be reduced. (Courtesy of C. Revenas)

diabetes older than 35 years, or who have had diabetes for over 20 years at baseline; (b) all patients older than 40 years at baseline with type 2 diabetes; (c) any diabetic patient who has newly detected diminished pulses, femoral bruits, or a foot ulcer; (d) any diabetic patient with leg pain of unknown etiology.

- Based on the results of the ABI, the following recommendations are suggested:
 - If the ABI is above 0.9, measurement should be repeated every 2–3 years.
 - If the ABI is 0.50–0.89, measurement should be repeated within 3 months and intensive management of co-existent cardiovascular risk factors should be initiated.
 - If the ABI is below 0.5, the patient should be referred for specialist vascular assessment and intensive management of co-existent cardiovascular risk factors should be initiated.
- If an incompressible artery with an ankle pressure above 300 mmHg or an ankle pressure 75 mmHg above arm pressure is found, these measurements should be repeated in 3 months. If still present, these patients should be referred for vascular assessment and intensive management of co-existent cardiovascular risk factors should be undertaken.

Invasive Vascular Testing — Arteriography

Arteriography remains the definitive diagnostic procedure before any form of surgical intervention. It should not be used as a diagnostic procedure to establish the presence of arterial disease. Contrast material may exaggerate any preexisting renal disease and for this reason the contrast material used should be limited as much as possible. In addition, the International Meeting on the Assessment of Peripheral

Vascular Disease in Diabetes strongly recommended that in diabetic patients arteriography should be carried out before any decision regarding an amputation is made, in order to assess the exact status of the vascular tree, particularly when the ankle brachial index and toe systolic pressure indicate that arterial disease is present.

Keywords: Etiopathogenesis of foot ulceration; diabetic neuropathy, diagnosis; symptoms of peripheral neuropathy; vibration perception threshold; Semmes–Weinstein monofilaments; assessment of vascular status; ankle brachial index; medial arterial calcification; toe pressure; transcutaneous oximetry; segmental pressures measurement; segmental plethysmography; ultrasonography; duplex; triplex; waveforms, quantitative analysis; waveforms, qualitative analysis; peak systolic velocity ratio; spiral computed tomography; magnetic resonance angiography; invasive vascular testing; angiography; Fontaine stage

BIBLIOGRAPHY

1. The International Working Group on the Diabetic Foot. *International Consensus on the Diabetic Foot*. Amsterdam, The Netherlands, 1999.
2. Boulton AJM, Greis FA, Jervell JA. Guidelines for the diagnosis and outpatient management of diabetic peripheral neuropathy. *Diabet Med* 1998; **15**: 508–514.
3. Boulton AJM. The pathway to ulceration: Aetiopathogenesis. In Boulton AJM, Connor H, Cavanagh PR (Eds), *The Foot in Diabetes* (3rd edn). Chichester: Wiley, 2000; 61–72.
4. Veves A, Uccioli L, Manes C, Van Acker K, Komninou H, Philippides P, Katsilambros N, De Leeuw I, Menzinger G, Boulton AJ. Comparison of risk factors for foot problems in diabetic patients attending teaching outpatient clinics in four different

- European states. *Diabet Med* 1994; **11**: 709–713.
5. Ziegler RE, Summer DS. Physiologic assessment of peripheral arterial occlusive disease. In Rutherford RB (Ed.), *Vascular Surgery* (5th edn). Philadelphia: Saunders, 2000; 140–165.
 6. Katsilambros N, Hatzakis A, Perdikaris G, Pefanis A, Papazachos G, Papadoyannis D, Balas P. Peripheral occlusive arterial disease in longstanding diabetes mellitus. A population study. *Int Angiol* 1989; **8**: 36–40.
 7. Kasilambros NL, Tsapogas PC, Arvanitis MP, Tritos NA, Alexiou ZP, Rigas KL. Risk factors for lower extremity arterial disease in non-insulin-dependent diabetic persons. *Diabet Med* 1996; **13**: 243–246.
 8. Donnelly R, Hinwood D, London NJM. Non-invasive methods of arterial and venous assessment. In Donnelly R, London NJM (Eds), *ABC of Arterial and Venous Disease*. London: BMJ Books, 2000; 1–4.
 9. Orchard TJ, Strandness DE. Assessment of peripheral vascular disease in diabetes. *Diabetes Care* 1993; **16**: 1199–1209.
 10. Veves A, Giurini JM, LoGerfo FW. *The Diabetic Foot: Medical and Surgical Management*. Totowa, NJ: Humana Press, 2002.

Chapter II

CLASSIFICATION, PREVENTION AND TREATMENT OF FOOT ULCERS

- CLASSIFICATION SYSTEMS
- CLINICAL PRESENTATION OF NEUROPATHIC, ISCHEMIC AND NEURO-ISCHEMIC ULCERS
- PREVENTION OF FOOT ULCERS
- METHODS FOR OFFLOADING PRESSURE ON THE FOOT
- DRESSINGS
- NEW TREATMENTS
- BIBLIOGRAPHY

CLASSIFICATION SYSTEMS

- *The Meggitt–Wagner classification* is the most well-known and validated system for foot ulcers, and is shown in [Table 2.1](#). The advantages and disadvantages of this classification system are described in [Table 2.2](#).
- ‘*The University of Texas classification system for diabetic foot wounds*’,

Table 2.1 Meggitt–Wagner classification of foot ulcers

Grade	Description of the ulcer
Grade 0	Pre- or post-ulcerative lesion completely epithelialized
Grade 1	Superficial, full thickness ulcer limited to the dermis, not extending to the subcutis
Grade 2	Ulcer of the skin extending through the subcutis with exposed tendon or bone and without osteomyelitis or abscess formation
Grade 3	Deep ulcers with osteomyelitis or abscess formation
Grade 4	Localized gangrene of the toes or the forefoot
Grade 5	Foot with extensive gangrene

Table 2.2 Advantages and disadvantages of the Meggitt–Wagner classification system

Advantages
<ul style="list-style-type: none"> • It is simple to use and has been validated in a number of studies • Higher grades are directly related to increased risk for lower limb amputation • It provides a guide for planning treatment • It is considered the gold-standard, against which other systems should be validated
Disadvantages
<ul style="list-style-type: none"> • Although the presence of infection and ischemia are related to poor outcome, ischemia in patients classified into grades 1–3 and infection in grade 1, 2 and 4 patients is not taken into account • The location of the ulcer is not described • Patient-related factors (poor foot care, emotional upset, denial) and foot deformities are not evaluated

([Table 2.3](#)) has recently been proposed and validated by the University of Texas. This system evaluates both depth of the ulcer—as in Meggitt–Wagner classification system—and presence of infection and ischemia. Uncomplicated ulcers are classified as stage A, infected ulcers as stage B, ulcers with ischemia as

Table 2.3 ‘The University of Texas classification system for diabetic foot wounds’

Stage	Grade			
	0	1	2	3
A	Pre- or post-ulcerative lesion completely epithelialized	Superficial wound not involving tendon, capsule or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	With infection	With infection	With infection	With infection
C	With ischemia	With ischemia	With ischemia	With ischemia
D	With infection and ischemia	With infection and ischemia	With infection and ischemia	With infection and ischemia

Table 2.4 Advantages and disadvantages of 'The University of Texas classification system'**Advantages**

- It is simple to use and more descriptive
- It has been evaluated and shown to predict more accurately the outcome of an ulcer (healing or amputation) than the Meggitt–Wagner classification.
- Cases with infection and/or ischemia are taken into account in this system
- It provides a guide for planning treatment

Disadvantages

- Patient-related factors (poor foot care, emotional upset, denial) and foot deformities are not evaluated
- The location of the ulcer is not described

stage C and ulcers with both infection and ischemia as stage D. Grades 1 and 2 are similar to the Meggitt–Wagner classification. Grade 3 ulcers are ulcers penetrating the bone or joint. The higher the grade, and the stage of an ulcer, the greater the risk for non-healing or amputation. The advantages and disadvantages of 'The University of Texas classification system' are described in [Table 2.4](#).

In addition to these two classification systems, other systems have been proposed:

- Edmonds and Foster have proposed a simpler classification. According to this system, based on clinical tests and determination of the ankle brachial pressure index, foot ulcers are classified into neuropathic and neuro-ischemic.
- Brodsky suggested the '*depth-ischemia*' classification, which is a modification of the Meggitt–Wagner classification. According to this proposal, ulcers are classified into four subgroups (A, not ischemic; B, ischemic without gangrene; C, partial gangrene of the foot; and

D, complete foot gangrene) with grades 0–3 (similar to the Meggitt–Wagner classification).

- Macfarlane and Jeffcoate proposed the *S(AD)SAD classification* for diabetic foot ulcers. According to this system, ulcers are classified on the basis of size (area and depth), presence of sepsis, arteriopathy, and denervation. This system awaits clinical validation.

Any valid classification system of foot ulcers should facilitate appropriate treatment, simplify monitoring of healing progress and serve as a communication code across specialties in standardized terms. Despite its disadvantages, the 'University of Texas classification system' offers many advantages over the Meggitt–Wagner system and is the most appropriate system devised to date. In addition, inclusion in a classification system of other parameters such as location of the ulcer, foot deformities and other factors which may be related to the outcome of an ulcer, makes the system more complex and cumbersome. 'The University of Texas classification system' is expected to be widely adopted in the future.

CLINICAL PRESENTATION OF NEUROPATHIC, ISCHEMIC AND NEURO-ISCHEMIC ULCERS

- Neuropathy is present in about 85–90% of foot ulcers in patients with diabetes.
- Ischemia is a major factor in 38–52% of cases of foot ulcers.

NEUROPATHIC ULCERS ([FIGURES 2.1–2.3](#))

- Develop at areas of high plantar pressures (metatarsal heads, plantar aspect of



Figure 2.1 Typical neuropathic ulcer with callus formation on the first metatarsal head before debridement



Figure 2.3 Neuropathic ulcer on the first metatarsal head with healthy granulating tissue on its bed and callus formation



Figure 2.2 Neuropathic ulcer on the first metatarsal head with healthy granulating tissue on its bed

the great toe, heel or over bony prominences in a Charcot-type foot).

- Are painless, unless they are complicated by infection.
- There is callus formation at the borders of the ulcer.
- Its base is red, with a healthy granular appearance.
- On examination evidence of peripheral neuropathy (hypoesthesia or complete loss of sensation of light touch, pain, temperature, and vibration in response to 5.07 monofilaments, atrophy of the small muscles of the feet, dry skin and distended dorsal foot veins) is present. However, the pattern of sensory loss may vary considerably from patient to patient.



Figure 2.4 Ischemic ulcer under the heel in a patient with severe peripheral vascular disease

- The foot has normal temperature or may be warm.
- Peripheral pulses are present and the ankle brachial pressure index is normal or above 1.3.

ISCHEMIC ULCERS (FIGURES 2.4–2.8)

- Develop on the borders or the dorsal aspect of the feet and toes or between toes.
- They are usually painful.



Figure 2.5 Ischemic ulcer on the dorsum of the second toe in a patient with critical limb ischemia. Case discussed in Chapter 7



Figure 2.6 Dry gangrene of the fifth right toe. Redness, and edema, which are typical signs of infection involving the forefoot, are present

- There is usually redness at the borders of the ulcer.
- Its base is yellowish or necrotic (black).
- There is a history of intermittent claudication.
- On examination indications of peripheral vascular disease (skin is cool, pale or cyanosed, shiny and thin, with loss of hair, and onychodystrophy; peripheral pulses are absent or weak; the ankle brachial index is <0.9) are present.
- Non-invasive vascular testing (duplex or triplex ultrasound examination,



Figure 2.7 Ischemic ulcer after sharp debridement of the gangrene shown in [Figure 2.6](#)



Figure 2.8 Ischemic ulcer on the tip of the third right toe, with necrotic center

segmental pressures measurement, plethysmography), and angiography confirm peripheral vascular disease.

- There are no findings of peripheral neuropathy.

MIXED ETIOLOGY ULCERS (NEURO-ISCHEMIC ULCERS) ([FIGURES 2.9 AND 2.10](#))

Neuro-ischemic ulcers have a mixed etiology, i.e. neuropathy and ischemia, and a mixed appearance.

PREVENTION OF FOOT ULCERS

Based on the results of clinical examination, and/or laboratory testing and imaging studies, every patient with diabetes may be classified on the basis of the risk for foot problems ([Table 2.5](#)). This classification helps as a guide for patient management. Patients with active foot ulcers are not included in this classification.

Inappropriate footwear is a major cause of ulceration. The aim of providing special shoes and insoles (preventive footwear) to diabetic patients at risk for foot ulceration, is to reduce peak plantar pressure over areas 'at risk', and to protect their feet against injuries from friction. Although there is limited scientific information about shoe selection, recommendations can be made in this regard, based on risk



Figure 2.9 Neuro-ischemic ulcer on heel. This was a painless ulcer due to severe diabetic peripheral neuropathy. Another neuro-ischemic ulcer is seen under the first metatarsal head. Claw toes and lateral plantar cracks on the midfoot are also evident

Table 2.5 Classification of categories of diabetic patients based on the risk for ulceration

Risk category	
0	Protective sensation is intact; the patient may have foot deformity
1	Loss of protective sensation
2	Loss of protective sensation and high plantar pressure, or callosities, or history of foot ulcer
3	Loss of protective sensation and history of ulcer, and severe foot or toe deformity and/or limited joint mobility; significant peripheral vascular disease

(Modified from Chantelau E. Footwear for the high-risk patient. In Boulton AJM, Connor H, Cavanagh PR (Eds), *The Foot in Diabetes* (3rd edn). Chichester: Wiley, 2000; 131–142, with permission).

stratification studies. Shoes for the patient at risk for ulceration should have certain characteristics. High heel shoes are completely inappropriate, as they shift body weight towards the forefoot, and increase pressure under the metatarsal heads. Patients with toe deformities need shoes with sufficient room in the toe box to prevent



Figure 2.10 Neuro-ischemic ulcer in the medial aspect of the right first metatarsal head with fibrous tissue and necrosis on its bed

friction and pressure on the dorsum of the toes.

A recent study from the UK estimated that providing preventive footwear for 700 patients at risk for foot ulceration per year (with an average total cost of €179,000), would only need to prevent two below-knee amputations per year in order to be cost-effective, since the total cost of an amputation procedure is about €88,000.

Foot deformity is defined according to the 'International Consensus on the Diabetic Foot' as 'the presence of structural abnormalities of the foot such as presence of hammer toes, claw toes, hallux valgus, prominent metatarsal heads, status after neuro-osteoarthropathy, amputations or other foot surgery'. Additional foot deformities which can also lead to foot ulceration are described in other chapters of this book.

RISK CATEGORY 0

Patients in this category are characterized by preserved protective sensation and normal blood supply to their feet. These patients should have their feet examined on an annual basis, as asymptomatic nerve or vascular damage may develop. There is no need for special footwear. Patients should be instructed to choose shoes of proper style and fit, which pose no risk to their feet should they develop loss of sensation or inadequate blood supply to the feet. Athletic footwear is a good choice.

RISK CATEGORY 1

Correct foot care should be explained to all patients classified in categories 1–3, and these patients should be examined in the outpatient diabetes clinic every 4 months. Loss of protective sensation should be 'replaced' by increased awareness of situations which threaten the foot. Patients in

category 1 are at twice the risk of developing foot ulcers than those in category 0. Particular care should be taken when these patients buy new shoes. Patients with loss of protective sensation tend to select shoes which are too small because they are more able to feel a tight shoe. Shoes should not be too loose either. The inside of the shoe should be 1–2 cm longer than the foot itself. The internal width should be equal to the width of the foot at the metatarsophalangeal joints. The fitting must be carried out with the patient in the standing position and preferably at the end of the day.

All patients with loss of protective sensation should have soft, shock-absorbing stock insoles in all shoes they wear. Such insoles are usually made of open cell urethane foam, microcellular rubber or polyethylene foam (plastazote). According to the design of the insole and the material used, peak plantar pressure reduction during walking may range from 5 to 40%. As insoles may take up considerable space inside the shoe, care should be taken to allow sufficient room for the dorsum of the foot (by the use of extra depth stock shoes) otherwise ulceration may develop in this area. Many materials used in footwear lose their effectiveness in a relatively short time, depending on the patient's degree of activity. Therefore, regular replacement of the insoles is necessary at least three times a year. Shoes should also be changed at least once a year. Some specifically designed socks (padded socks) may also be used, since these reduce peak plantar pressures during walking by up to 30%.

EDUCATING PATIENTS IN APPROPRIATE FOOT CARE

Education of patients who are at risk of developing foot ulceration is the cornerstone of disease management. Patients

should fully understand the risks posed by the loss of protective sensation or an inadequate blood supply to their feet. Education of the patient at risk may reduce the incidence of foot ulcers and subsequently amputations.

The patient at risk for foot ulceration should:

- Inspect his or her feet every day, including areas between toes. Inspection of the sole may be accomplished using a mirror.
- Let someone else inspect his or her feet in cases where the patient is unable to do it.
- Avoid walking barefoot any time, in- or outdoors.
- Avoid wearing shoes without socks, even for short periods.
- Buy shoes of the correct size.
- Avoid wearing new shoes for more than 1 h per day; feet should be inspected after taking off new shoes; in the case of foot irritation the patient should inform the healthcare provider.
- Change shoes at noon, and, if possible, again in the evening; this prevents high pressures remaining on the same area of the foot for a prolonged period.
- Inspect and palpate the inside of his or her shoes before wearing them.
- Wash his or her feet every day, taking care to dry them, especially the web spaces.
- Avoid putting his or her feet onto heaters.
- Test the water temperature before bathing using his or her elbow; the temperature of the water should be less than 37°C.
- Avoid the use of chemical agents or plasters and razors for the removal of corns and calluses; they must be treated by a health care provider.
- Cut the nails straight across.
- Wear socks with seams inside out, or preferably without any seams at all.

- Use lubricating oils or creams for dry skin, but not between toes.
- Inspect his or her feet after prolonged walking.
- Notify his or her healthcare provider at once, if a blister, cut, scratch, sore, redness or black area develops, or if any discharge appears on socks.

RISK CATEGORY 2

Patients in this category do not usually need custom-made shoes. The use of appropriate *insoles*, which reduce peak plantar pressures under specific areas, is usually enough; these are inserted in commercially available extra-depth shoes. Insoles must be custom-molded and shock-absorbing. The idea is to redistribute plantar pressures by the use of such insoles, that is, to decrease the load from regions ‘at-risk’ to ‘safe’ regions. In addition, insoles reduce shear stress since total contact minimizes the horizontal and vertical foot movement. These insoles have two or three layers and are made of materials of different density. A thin layer of the material with the lowest density (the most potent shock-absorbing material, usually cross-linked polyethylene foams) is placed at the foot–insole interface; the firmest material (acrylic plastics, thermoplastic polymers or cork) is placed at the shoe–insole interface. A soft, shock-absorbing, durable material (closed cell neoprene, rubber or urethane polymer) is placed between them (Figures 2.11 and 2.12). Appropriate insoles for the patient at risk for ulceration should have a minimum thickness of 6.25 mm. Patients at high risk require thicker (12.5 mm) insoles.

RISK CATEGORY 3

These patients need the greatest help to remain free of foot ulceration. Patients in this



Figure 2.11 Upper side of a three-layer custom-made insole used to offload pressure on the forefoot. The upper layer is composed of cross-linked polyethylene foam, the middle layer of polyurethane, and the lower layer of cork

category are 12–36 times more likely to develop foot ulcers than patients in category 0. Severe foot deformities and limited mobility of the foot joints are associated with high plantar pressures.

Limited joint mobility is defined as a limitation in dorsiflexion of the first metatarsophalangeal joint of more than 50° when the patient is seated (hallux rigidus).

Patients with severe peripheral vascular disease are also included in this category. Inadequate circulation makes the thin skin vulnerable to ulceration.

In addition to custom-molded insoles, custom-made and extra depth-shoes are

often necessary. Patients with recurrent foot ulcerations, or an active lifestyle, often need modifications of the outsole. In the *rocker* style shoe the rigid outsole rotates over a ridge (fulcrum) as the patient walks; this ridge is located 1 cm behind the metatarsal heads (see [Figure 5.2](#)). The rocker outsole allows the shoe to ‘rock’ forward during propulsion before the metatarsophalangeal joints are allowed to flex, thereby reducing the pressure applied to the forefoot. In a *roller* style shoe the contour of the outsole is a continuous curve without the ridge used in the rocker style. During walking, as the person lifts the heel, the shoe rolls forward on the curved outsole. This prevents the pressure from remaining in one region. Rocker style shoes are more effective in reducing forefoot plantar pressure than the roller style shoes.

METHODS FOR OFFLOADING PRESSURE ON THE FOOT

The mainstay in the management of an active plantar foot ulcer is the effective offloading of the ulcer area. Once an ulcer is

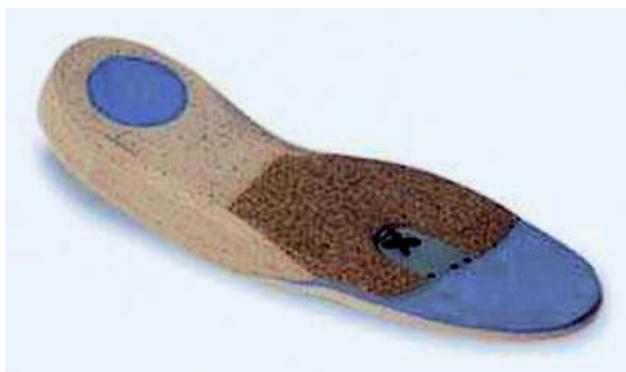


Figure 2.12 Lower side of insole illustrated in [Figure 2.11](#)

present, it will not heal unless the mechanical load on it is removed. Among the methods used for this purpose are complete bed rest, crutch-assisted gait, wheelchair, and prosthetics. However, these methods are impractical for the majority of patients to use for a period of several weeks while the ulcer heals. Common approaches for reducing the load on the ulcerated area include the use of a total-contact cast or other commercially-available casts, and therapeutic footwear.

TOTAL-CONTACT CAST

A total-contact cast (Figure 2.13) is a plaster of Paris cast, which extends from knee to toes. This is the method of choice for the treatment of grades 1 and 2 (according to the Meggitt–Wagner classification) diabetic foot ulcers which are located on the forefoot



Figure 2.13 Total-contact cast

and midfoot; the cast reduces peak plantar pressures in these areas by almost 40–80%, but is less effective with ulcers located on the hindfoot. In one study, the use of a total-contact cast resulted in almost 90% of plantar ulcers healing within an average of 6–7 weeks. This method permits walking while uniformly decreasing the pressure on the sole of the foot.

The ulcerated area should be debrided and covered with a thin dry dressing. A total-contact cast is applied with the patient in the prone position and the foot and ankle in a neutral position (i.e. with the foot flexed at a 90°-angle to the ankle). A layer of fiberglass tape is usually applied over the plaster, to strengthen the cast and allow early ambulation. A small rubber rocker is added for walking. A plywood board is inserted between the rubber rocker and the cast in order to minimize the possibility of the sole of the cast becoming cracked. The cast should be changed every 3–7 days. The use of a total-contact cast is contraindicated when infection or gangrene (Meggitt–Wagner stages 3–5) is present. Skin atrophy and an ankle brachial index below 0.4 are considered to be relative contraindications to the use of a total-contact cast. Although a total-contact cast permits walking, patients are instructed to minimize their activity in order to reduce the pressure on their soles. Instability and the risk of falls are disadvantages of this cast. Both in- and outdoor compliance is another advantage, especially for the non-compliant patient, since this cast is not easily removed.

OTHER COMMERCIALY- AVAILABLE CASTS

Removable Cast Walkers

Prefabricated walkers function on a similar principle to the total-contact cast and

are removable, commercially available, lightweight casts (see [Figure 9.11](#)). They are not designed to provide total contact, and the addition of inflatable or adjustable pads reduces movement of the limb within the cast. A custom-molded removable insole is adjusted to reduce plantar pressure. Use of removable cast walkers allows inspection and dressing of the wound on a daily basis. They may be used in patients with infected and ischemic ulcers. In addition, patients can bathe and sleep more comfortably. The rocker shape of the outsole reduces further pressure on the forefoot while standing and walking. In addition, these casts are ideal for clinics, which do not have personnel with experience in plastering.

Scotch-Cast Boot

This is a lightweight, well-padded fiberglass cast, extending from just below the toes to the ankle, and it is worn with a cast sandal ([Figure 2.14](#)). It may be fabricated as a removable or non-removable cast. With appropriate modifications of the pads, the scotch-cast boot reduces pressure on any region of the sole when needed. Removable scotch-cast boots can be used in cases of both ischemic and infected ulcers, since drainage and wound dressings are easily applied. As with the total-contact cast, experience in plastering is required.

PRESSURE RELIEF SHOES (THERAPEUTIC FOOTWEAR)

These are temporary shoes which allow some level of ambulation, while at the same time offloading pressure on the ulcerated area. These shoes are easy to use and are of low cost and since they enable the patient to walk quite normally, they lead



Figure 2.14 Scotch-cast boot

to a better quality of life. A rigid rocker sole is incorporated in order to reduce the weight-bearing load in the forefoot by up to 40% during walking. The appropriate choice of insole may reduce plantar pressure by an additional 20%. Half shoes (see [Figure 3.36](#)) are indicated for ulcers located on the forefoot (almost 90% of diabetic foot ulcers are located in this area). They offload pressure on the entire forefoot, while increasing pressure on the midfoot and heel, permitting the patient to engage in limited walking activities. Instability is a problem, and the patient needs to use crutches. With the use of half shoes the mean time to ulcer healing was reported to be 7–10 weeks in two studies. Patients are instructed to walk on their heel and avoid forefoot contact with the ground at the end of the stance phase. A sole lift

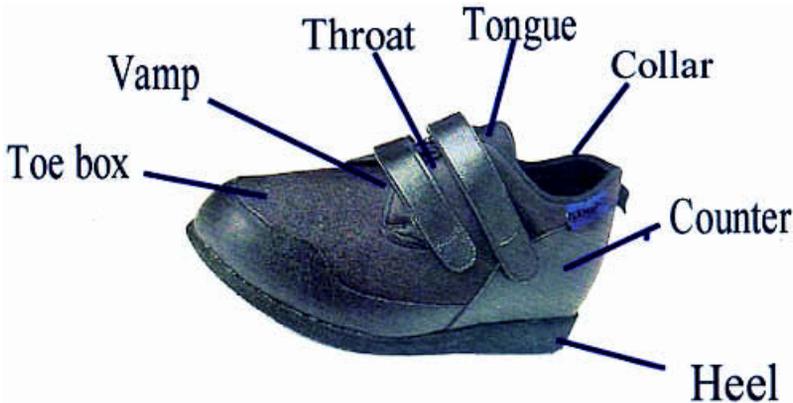


Figure 2.15 Shoe terms

on the opposite shoe may be necessary to equalize the limb length. These shoes are easily removed for dressing changes.

Heel-free shoes (see [Figure 5.18](#)) reduce peak plantar pressure on the heel by transferring pressures to the midfoot and forefoot. They have the same advantages and disadvantages as half shoes. Both half and heel-free shoes are commercially available.

Ulcers located on midfoot (mainly over bony prominences due to neuro-osteoarthropathy) are best treated with the use of customized insoles with windows under the ulcerated area.

Shoe terms are shown in [Figure 2.15](#).

DRESSINGS

The characteristics required for optimal wound dressings have been described as follows. They should

- be free from particulate or toxic contaminants
- remove excess exudates and toxic components

- maintain a moist environment at the wound/dressing interface
- be impermeable to microorganisms, thus protecting against secondary infection
- allow gaseous exchange
- be easily removal without trauma
- be transparent, or changed frequently, thus allowing monitoring of the wound
- be acceptable to the patient, conformable and occupy a minimum of space in the shoe
- be cost-effective
- be available in hospitals and community health care centers

There is a broad spectrum of wound dressing materials currently available. Their particular properties and indications are described in [Table 2.6](#) and the advantages and disadvantages of the available types of dressings are described in [Table 2.7](#).

NEW TREATMENTS

HYPERBARIC OXYGEN

There have been no controlled trials comparing the use of hyperbaric oxygen therapy

Table 2.6 Properties, and indications of available dressings

Type of dressing	Necrosis/slough	Gangrenous	Infection	Low exudate	High exudate	Flat wound with low exudate	Flat wound with high exudates	Cavity without sinus	Cavity with sinus tract
Dry		+	+	+		+			
Enzymatic debrider	+								
Films				+		+			
Foams			+	+	+		+		
Hydrogels	+							+	+
Hydrocolloids				+		+		+	
Alginates			+		+	Alginate rope		Alginate rope	Alginate rope

Table 2.7 Advantages and disadvantages of available types of dressings

Type of dressing	Advantages	Disadvantages
Traditional dressings (gauze and absorbent cellulose)	Cheap and widely available. Appropriate for gangrenous lesions	Adhere to the wound bed and may cause bleeding on removal. Provide little protection against bacterial contamination
Films	Semi-permeable. Form bacterial barrier. Durable. Require changing every 4–5 days. Cheap	Useful on flat or superficial wounds only. Some patients are allergic to the adhesive in the dressing
Foams	Appropriate for ulcers with high production of exudates. Provide thermal insulation. Easily conformable. May be used to fill cavities without sinus tracts	Variability in absorbency of different foams. Limited published data
Hydrogels	Effective, versatile and easy to apply. Very selective, with no damage to surrounding skin. Safe process, using the body’s own defense mechanisms. Promote autolysis and healing. Decrease risk of infection. Useful in removing slough from wounds. May be used to fill cavities with sinus tracts	Effect difficult to quantify. Not as effective and rapid as surgical debridement. Not appropriate for neuro-ischemic ulcers, which produce minimal exudates. Wound must be monitored closely for signs of infection
Hydrocolloids	Safe and selective, using the body’s own defense mechanisms. Good for necrotic lesions, with light to moderate exudates. May be used to fill cavities without sinus tracts. Can be easily used with a shoe. Adhesive surface prevents slippage. Do not require daily dressing changes. Cost-effective	Their occlusive and opaque nature prevents daily observation of the wound. Wound must be monitored closely for signs of infection. May promote anaerobic growth and mask a secondary infection

(continued overleaf)

Table 2.7 (continued)

Type of dressing	Advantages	Disadvantages
Alginates	Useful as absorbents of exudates. Good for infected ulcers. Some products have hemostatic properties	Not appropriate for neuro-ischemic ulcers, which produce minimal exudates. Some researchers think they may traumatize the wound bed and predispose to infections. May dry out and form a plug within the wound bed. Requires painstaking removal with the use of large amounts of saline
Enzymatic debriders	Good for any wound with a large amount of necrotic debris, and for eschar formation. Promote autolysis and fast healing. Decrease maceration of the skin, and risk of infection	Costly. Must be applied carefully only to the necrotic tissue. May require a specific secondary dressing. Irritation and discomfort may occur
Medicated dressings		Data based on animal models and cell cultures only

in the treatment of neuropathic ulcers. At the present time it is only used to treat patients with severe foot infections which have not responded to other treatments. Hyperbaric oxygen is particularly effective in patients with foot ischemia.

FACTORS ACCELERATING WOUND HEALING

Platelet-Derived Growth Factor- β

Platelet-derived growth factor- β (PDGF- β , becaplermin, Regranex®, Janssen-Cilag) has been developed as a topical, effective and safe therapy for the treatment of diabetic foot ulcers and has also been found to be effective and safe as local therapy for the treatment of non-infected diabetic foot ulcers. It is applied as a gel on the ulcer surface once daily by the patient, while the ulcer is debrided on a weekly basis. A dose of 100 $\mu\text{g/g}$ has been demonstrated to be the most effective. Compared to standard treatment, more ulcers treated with becaplermin heal completely and in a

shorter time. The maximum time required to achieve has been reported as 20 weeks.

Dermagraf®

Dermagraf® (Smith & Nephew) is a bio-engineered 'human dermis' designed to replace the patient's own damaged dermis. It is applied to the ulcerated area on a weekly basis. Preliminary results show that it is an effective and safe treatment. According to a controlled trial, 50% of diabetic foot ulcers healed within 8 weeks when treated with Dermagraf, compared to 8% of ulcers treated with standard methods. Dermagraf should be stored at -70°C and must be thawed, rinsed and cut to the size of the ulcer prior to implantation. As with becaplermin, the presence of infection is a contraindication to its use.

Graftskin

Graftskin (Apligraf®, Novartis) consists of an epidermal layer formed by human keratinocytes and a dermal layer, composed

of human fibroblasts derived from neonatal foreskin in a bovine collagen matrix. Studies have shown that treatment with Apligraf resulted in a higher percentage of diabetic foot ulcers healing completely and in a shorter time (56% of the ulcers healed in 65 days), compared to placebo (39% of the ulcers healed in 90 days). Apligraf has been shown to be safe and, in addition, its use was found to lead to a reduction in the incidence of osteomyelitis and amputations.

Granulocyte-Colony Stimulating Factor (GCSF)

Subcutaneous administration of GCSF once daily for 1 week in patients with infected foot ulcers resulted in a faster resolution of the infection, earlier eradication of bacterial pathogens isolated from wound swabs, shorter duration of i.v. antibiotic administration and shorter duration of hospital stay in a double-blind placebo-controlled study. Larger controlled studies are needed to evaluate the efficacy and safety of GCSF in the treatment of the infected foot ulcers.

Hyaff®

Hyaff® (Convatec, Bristol-Myers-Squibb) is a semi-synthetic ester of hyaluronic acid. Serum or wound exudates, when in contact with Hyaff, form a moist environment which promotes granulation and healing. So far it has been used in the treatment of neuropathic ulcers with promising results.

Keywords: Classification of foot ulcers; Meggitt-Wagner classification of foot ulcers; 'The University of Texas classification system for diabetic foot wounds'; neuro-ischemic ulcers, characteristics; ischemic ulcers, characteristics; neuropathic

ulcers, characteristics; prevention of foot ulcers; risk category for foot ulcers; education in foot care; insoles; limited joint mobility; methods for offloading pressure on the foot; total-contact cast; manufactured casts; removable cast walkers; scotch-cast boot; therapeutic footwear; heel-free shoes; half shoes; shoe terms; hyperbaric oxygen; platelet-derived growth factor- β ; Dermagraf®; Graftskin; Apligraf®; granulocyte-colony stimulating factor; Hyaff®; dressings; dressings, advantages and disadvantages

BIBLIOGRAPHY

1. Meggitt B. Surgical management of the diabetic foot. *Br J Hosp Med* 1976; **16**: 227-232.
2. Wagner FW. The dysvascular foot: a system for diagnosis and treatment. *Foot Ankle* 1981; **2**: 64.
3. Lavery LA, Armstrong DG, Harkless LB. Classification of diabetic foot wounds. *J Foot Ankle Surg* 1996; **35**: 528-531.
4. Armstrong DG, Lavery LA, Harkless LB. Validation of a diabetic wound classification system. *Diabetes Care* 1998; **21**: 855-861.
5. Consensus development conference on diabetic foot wound care. *Diabetes Care* 1999; **22**: 1354-1360.
6. Young MJ. Classification of ulcers and its relevance to management. In Boulton AJM, Connor H, Cavanagh PR (Eds), *The Foot in Diabetes* (3rd edn). Chichester: Wiley, 2000; 61-72.
7. Edmonds ME, Foster AVM. Classification and management of neuropathic and neuroischaemic ulcers. In Boulton AJM, Connor H, Cavanagh PR (Eds), *The Foot in Diabetes* (2nd edn). Chichester: Wiley, 1994; 109-120.
8. Brodsky JW. The diabetic foot. In Bowker JH, Pfeifer MA (Eds), *Levin and O'Neal's The Diabetic Foot* (6th edn). St Louis: Mosby, 2001; 273-282.
9. Macfarlane RF, Jeffcoate WJ. Classification of foot ulcers: The S(SAD)SAD system. *Diabetic Foot* 1999; **2**: 123-131.

10. Shaw JE, Boulton JE. The pathogenesis of diabetic foot problems. *Diabetes* 1997; **46**(Suppl. 2): S58–S61.
11. Edmonds ME, Bates M, Doxford M, Gough A, Foster A. New treatments in ulcer healing and wound infection. *Diabetes Metab Res Rev* 2000; **16**(Suppl. 1): S51–S54.
12. Dinh T, Pham H, Veves A. Emerging treatments in diabetic wound care. *Wounds* 2002; **14**: 2–10.
13. Veves A, Falanga V, Armstrong DG, Sabolinski ML. Graftskin, a human skin equivalent, is effective in the management of noninfected neuropathic diabetic foot ulcers. *Diabetes Care* 2001; **24**: 290–295.
14. Jones V. Selecting a dressing for the diabetic foot: factors to consider. *Diabetic Foot* 1998; **1**: 48–52.
15. Harding KG, Jones V, Price P. Topical treatment: which dressing to choose. *Diabetes Metab Res Rev* 2000; **16**(Suppl. 1): S47–S50.

Chapter III

ANATOMICAL RISK FACTORS FOR DIABETIC FOOT ULCERATION

- PES PLANUS
- PES PLANUS DEFORMITY — BUNIONETTE
- PES CAVUS
- BUNIONETTE (TAILOR'S BUNION)
- CLAW TOES
- CLAW AND CURLY TOE DEFORMITIES
- VARUS DEFORMITY OF TOES
- HELOMA DURUM, BUNION, BURSITIS, CLAW TOE
- HELOMA MOLLE
- HALLUX VALGUS WITH OVERRIDING TOE
- CONVEX TRIANGULAR FOOT (HALLUX VALGUS AND QUINTUS VARGUS)
- HALLUX VALGUS, OVERRIDING TOE, CLAW TOES, EDEMA
- ONYCHOMYCOSIS: HALLUX VALGUS AND HAMMER TOE DEFORMITY
- MALLEET TOE
- PROMINENT METATARSAL HEADS AND CLAW TOES

- POSTOPERATIVE HALLUX VALGUS AFTER SECOND TOE REMOVAL
- FIRST RAY AMPUTATION
- CALLUS UNDER BONE PROMINENCE
- CALLUS OVER PROMINENT METATARSAL HEADS
- HEMORRHAGIC CALLUS
- ULCER UNDER A CALLUS AREA
- ULCER UNDER HALLUX
- HEEL CRACKS
- BILATERAL CHOPART DISARTICULATION
- NEUROPATHIC ULCER
- INGROWN NAILS (ONYCHOCRYPTOSIS)

PES PLANUS (FLAT FOOT)

A 73-year-old female patient with type 2 diabetes diagnosed at the age of 55 years and treated with insulin since the age of 65 years, attended the diabetic foot clinic because of a small superficial painful ulcer over her medial malleolus. The patient complained of dysesthesias (she had a cold or warm sensation in her feet), and she had hypertension for which she had been treated with enalapril since the age of 55 years. The ulcer was noticed 4 weeks previously and had been caused by an external minor trauma.

On examination, bilateral pes planus with minor hyperkeratosis over the first metatarsal head was found (Figure 3.1). The ankle brachial index, peripheral pulses, vibration perception threshold, and monofilament (5.07) sensation were all normal. The ulcer was debrided on a weekly basis, and it healed in 4 weeks.

Pes planus (or flat foot) is characterized by diminished longitudinal and transverse concavities of the foot. Diminished plantar transverse concavity is associated with

an increase in frontal transverse convexity of the tarsometatarsal joint line (Lisfranc joint line) and divergence of the five metatarsal bones. The load transfer is displaced to the medial border of the mid-tarsal region. However, there is evidence that flat feet protect against loading of the metatarsal heads, although they are poor shock absorbers. Pes planus may cause bunionette formation and plantar heel spur pain, but other foot problems are uncommon. Foot orthotics and arch supports do not alter the osseous relationships and are ineffective in many patients. Surgical treatment is rarely indicated in adults.

Keywords: Pes planus; malleoli ulcer; infection

PES PLANUS DEFORMITY — BUNIONETTE

A 74-year-old male patient with type 2 diabetes diagnosed at the age of 61 years attended the outpatient diabetic foot clinic for chiropody treatment. On examination, he was found to have mild callus formation



Figure 3.1 Pes planus

at the plantar and the lateral area of the fifth metatarsal head (Figures 3.2 and 3.3). Bilateral pes planus (flat foot) deformity of his feet and a bony prominence at the lateral aspect of the fifth metatarsal head (a bunionette or tailor's bunion) were also found (see Figure 3.2). Blackening of the nail of the hallux was due to a subungual hematoma. Pedal pulses were palpable and the patient had severe peripheral neuropathy. The patient had the callus removed and was instructed in appropriate foot care. In addition, he was advised to wear suitable shoes with a wide toe box.

Pes planus or flat foot is the commonest foot deformity (prevalence is about 20%

in the adult population) and its prevalence increases with the age. The majority of flat feet are considered to be variations of normal. People with this deformity are able to walk as comfortably as people with normal arches (see also Figure 3.1).

Keywords: Pes planus; flat foot; bunionette

PES CAVUS

A 64-year-old female patient with type 2 diabetes diagnosed at the age of 62 years was referred to the outpatient diabetic foot



Figure 3.2 Pes planus with bunionette. Plantar aspect



Figure 3.3 Pes planus with bunionette. Dorsal aspect



Figure 3.4 Pes cavus

clinic for foot care. She had been treated with insulin for the last 4 years. The patient had a history of hypertension. No diabetic complications were mentioned.

On examination, peripheral pulses were bounding. She had severe peripheral neuropathy (no sensation of pain, light touch, temperature, vibration or 5.07 monofilaments) and dry skin. A high plantar arch due to pes cavus was noted, which was more apparent in the standing position. Mild hallux valgus, clawing of the toes, and callus formation over the inner aspect of the first metatarsal heads as well as at the tip of the second toe and the second metatarsal head bilaterally were observed (Figure 3.4). The patient had the callus removed, and the nails cut and she was educated in foot care. Suitable shoes and insoles were prescribed and she was advised to attend the foot clinic on a monthly basis for chiropody treatment.

Pes cavus is a deformity not necessarily related to diabetes. Indeed, the patient mentioned that her foot shape had been the same before the diagnosis of diabetes and her mother probably had the same deformity.

Normally the inner edge of the mid-foot is raised off the floor forming an arch, which extends between the first metatarsal and the calcaneus. When the arch of the foot is higher than normal (pes cavus) claw toes often develop. In cavus foot the forefoot, and especially the first ray, is drawn downwards and an abnormal distribution of plantar pressure upon standing and walking leads to callus formation under the metatarsal heads. Cavus feet tend to be stiffer than normal; some patients may be prone to ankle strains. Patients should be advised to wear appropriate shoes (extra depth and broad at the toe box) and



Figure 3.5 Bunionette with claw toes

orthotic, shock-absorbing insoles. Surgery for the correction of the abnormality is rarely recommended.

Keywords: Pes cavus

BUNIONETTE (TAILOR'S BUNION)

A 54-year-old female diabetic patient attended the outpatient diabetic foot clinic for regular chiropody treatment. She had severe diabetic neuropathy with reduced sensation of light touch, vibration, pain, temperature and 5.07 monofilaments. Peripheral pulses were normal. Muscle atrophy of the feet, claw toes, mild hallux valgus, varus deformity of the lesser toes, and an exostosis of the lateral part of the fifth metatarsal head (bunionette, [Figure 3.5](#)) were present. Another exostosis was noted at the tuberosity of the fifth metatarsal bone. Appropriate

shoes with a high and broad toe box were prescribed, and the patient was educated in correct foot care.

Bunionette, or tailor's bunion, is often associated with varus deformity of the lesser toes. Ulceration over a bunionette may occur in a patient who has no feeling of pain, and an infection of the ulcer may spread to the bursa and the underlying bone.

Keywords: Bunionette

CLAW TOES

A 56-year-old male patient with type 2 diabetes diagnosed at the age of 44 years attended the outpatient diabetes clinic. He had been treated with insulin since the age of 53 years, with excellent results (HBA_{1c}: 6.7%). He had background diabetic retinopathy.



Figure 3.6 Muscle atrophy with claw toes and hallux valgus

On examination, the patient had severe diabetic neuropathy with complete loss of sensation of pain, light touch and temperature; his vibration perception threshold was 40 V on both feet; Achilles tendon reflexes were absent. Peripheral pulses were normal and the ankle brachial index was 1.2 bilaterally. Temperature of the feet was normal; the skin was dry, with normal hair and nails, while mild vein distension was noted. Severe atrophy of the intrinsic foot muscles (lumbrical and interossei)—due to motor neuropathy—resulted in an imbalance of the foot muscles, and cocked-up toes (claw toes) (Figure 3.6). Such an appearance is so typical, that the diagnosis of peripheral neuropathy can be made by inspection of the feet alone.

A claw toe, the most common deformity in diabetic patients, consists of dorsiflexion of the metatarsophalangeal joint, while the proximal interphalangeal and distal interphalangeal joints are in plantar flexion (Figure 3.7). Shifting of the fat pads underneath the metatarsal heads to the front leaves the metatarsal heads exposed; high plantar pressures develop under metatarsal heads. This patient did not have problems with his feet. He was educated in appropriate foot care and instructed to wear suitable footwear with a toe box large enough to accommodate the deformity.



Figure 3.7 Claw toe

Keywords: Muscle atrophy; peripheral neuropathy; claw toes

CLAW AND CURLY TOE DEFORMITIES

A 68-year-old female patient with type 2 diabetes attended the outpatient diabetes clinic for her usual follow-up. On examination, she had severe diabetic neuropathy and palpable peripheral pulses. Claw toe deformity of her left second and third toes was noticed, as well as a curly fourth toe (Figure 3.8). Subungual hemorrhage and ingrown hallux nail, and hemorrhagic calluses of the second and third toes were also present. A hammer deformity was seen on the second toe of her right foot. Protective



Figure 3.8 Curly fourth toe with inward malrotation. Claw toes

footwear was prescribed and the patient was educated in foot care.

A curly toe consists of neutral position or plantar flexion of the metatarsophalangeal joint, and plantar flexion of the proximal interphalangeal and distal interphalangeal joints, by more than 5° each (Figures 3.9 and 3.10). Inward or outward rotation may be present. Curly toes may be either fixed or flexible.

Keywords: Claw toe; curly toe; hammer toe

VARUS DEFORMITY OF TOES

In varus deformity of toes the third, fourth and fifth toes drift medially. The nails of

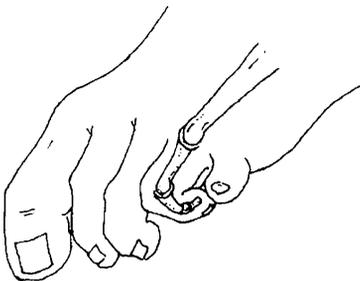


Figure 3.9 Curly fourth toe



Figure 3.10 Curly fourth toe. Note inward malrotation

the toes may cause superficial ulcers on the adjacent toes. This patient was a 60-year-old female with type 2 diabetes diagnosed at the age of 51 years. She had severe diabetic neuropathy; peripheral pulses were normal, and she had never had a foot ulcer. In addition to varus deformity, clawing of her toes was present (Figure 3.11). Varus deformity often co-exists with bunionette.

Keywords: Varus deformity of toes

HELOMA DURUM, BUNION, BURSITIS, CLAW TOE

A 67-year-old male patient with type 2 diabetes attended the outpatient diabetic foot



Figure 3.11 Varus and claw toes deformity

clinic because he had developed painless hyperkeratosis on the dorsum of his toes.

He had severe peripheral sensorimotor neuropathy; peripheral pulses were normal. Significant muscle atrophy was seen on the dorsum of his feet (Figure 3.12). Mild hallux valgus and claw toes deformity were also present. As a result of a bunion (see below) due to hallux valgus deformity, a red and swollen bursa developed at the medial aspect of both first metatarsal heads, caused by pressure and friction exerted on these areas by his shoes. Painless corns were also present on the dorsum of the toes. Such corns—called *heloma durum* or hard corns—are a result of pressure and friction on the deformed toes caused by wearing low toe box shoes. Suitable shoes (with a broad and high toe box) were prescribed in order to accommodate the deformity. The patient did well; *heloma durum* and bursitis did not relapse.

A bunion is a bony prominence that develops on the inner side of the foot, near the base of the first toe. An infected ulcer



Figure 3.12 *Heloma durum*, bunion, bursitis and claw toe

over a bunion or a *heloma durum* may lead to infection spreading into a joint or the bone.

Keywords: *Heloma durum*; bunion; bursitis; claw toe

HELOMA MOLLE

A 54-year-old male patient with type 2 diabetes diagnosed at the age of 48 years attended the outpatient diabetic foot clinic for callus removal. He had severe diabetic neuropathy (loss of sensation of pain, light touch, temperature, vibration and 5.07 monofilaments), and he complained of mild pain on his left little toe.

On examination, a painful corn was seen at the medial aspect of his left little toe (Figure 3.13).

Corns are circular hyperkeratotic areas which may be soft or hard. They have a polished or translucent center and may become painful due to persistent pressure and friction. Soft corns develop in the interdigital



Figure 3.13 Heloma molle

spaces; these are known as heloma molle, and they are caused by pressure and friction from the adjacent toe bones. This type of corn often has a soft consistency (in contrast to a heloma durum) due to moisture retention in the interdigital space. The commonest location of a heloma molle is the lateral side of the fourth toe, caused by pressure and friction on the adjacent head of the proximal phalanx of the fifth toe, but it may also occur in the other interdigital spaces. Osteoarthritic changes of the distal interphalangeal joints often cause heloma molle. Kissing heloma molles result when the ends of the phalanges are too wide. Tight shoes aggravate the problem. This condition is especially common in women

who wear high-heel shoes, which shift the body's weight to the front of the foot, squeezing the toes into a narrow, tapering toe box.

Heloma molle, like heloma durum may cause discomfort, and it may be complicated by infection. The patient is advised to wear wide shoes or shoes with a high toe box. Surgical removal of small portions of the bones or the exostoses that are involved in the pathogenesis of the heloma molle is the permanent treatment.

Keywords: Corns; heloma molle; heloma durum

HALLUX VALGUS WITH OVERRIDING TOE

A 69-year-old female patient with type 2 diabetes diagnosed at the age of 55 years and treated with antidiabetic tablets was referred to the outpatient diabetic foot clinic because of a recurrent ulcer over her first left metatarsal head. The patient had no macroangiopathic complications; peripheral neuropathy was found on examination.

Hallux valgus with fixed varus deformity and clawing of second toe in supraductus was noticed, together with callus formation



Figure 3.14 Hallux valgus with overriding toe

under her first metatarsal head and ulceration of its medial aspect (Figure 3.14).

Hallux valgus and the associated varus posture of the first metatarsal bone cause various deformities of the other toes, such as varus, clawing and valgus formation. The long and short extensor tendons of all the toes shrink like bowstrings, causing subluxation of the phalangeal bases. Contractures of tendons and joint capsules result in fixation of the deformity. Due to the deformity of the third and fourth toes the heads of the three central metatarsal bones become lowered, resulting in their exposure and callus formation. In more severe cases of hallux valgus, the line of load is displaced progressively towards the medial side of the foot, and the longitudinal arch becomes lower, leading to pes planovalgus.

Keywords: Overriding toe; hallux valgus

CONVEX TRIANGULAR FOOT (HALLUX VALGUS AND QUINTUS VARUS)

A 48-year-old female diabetic patient with type 2 diabetes diagnosed 6 months before

her first visit, and treated with sulfonylurea, was referred to the outpatient diabetic foot clinic because of an ulcer on her right foot.

The diabetes had been adequately controlled but the patient was already exhibiting signs of diabetic complications, such as background retinopathy and neuropathy. On examination, she had a right convex triangular foot, with an ulcer under the head of the fifth metatarsal head following callus formation at this site (Figure 3.15). She had symptomatic diabetic neuropathy, exemplified by a burning sensation in the feet, which was especially exacerbated at night; peripheral pulses were palpable and the ankle brachial index was 1.0 bilaterally. Small muscle atrophy of the feet was noted, as well as dry skin and loss of feeling of a 5.07 monofilament; vibration perception threshold was 30 V.

A plain X-ray showed a convex triangular foot deformity (Figure 3.16). This deformity is characterized by convergence of first and fifth toes, and claw deformities of the central three toes. The first and fifth metatarsals are short and diverge. Both longitudinal and transverse plantar concavities are accentuated, and the second and third metatarsals are fixed in excessive equinus



Figure 3.15 Neuropathic ulcer under fifth metatarsal head



Figure 3.16 Plain radiograph of a convex triangular foot

from this level. Cavus feet balance on the heel and the central part of the metatarsal paddle. This deformity may cause high pressures over the metatarsal paddle during walking.

Debridement was performed and appropriate footwear and insoles were prescribed (Figure 3.17). A suitable insole relieved pressure strain from the sole of the patient's foot by redistributing pressures. High plantar pressures can be seen on the graph produced by insole pressure sensors (Parotec system, Germany) (Figure 3.18), when the patient used her own shoes (Panel A), and after the prescribed insole and shoe were used (Panel B); pressures applied to the sole of the patient's foot during heel strike, mid-support and push-off phase of walking with

the patient's original shoe (left graph), and with the custom-made insole (right graph) are shown in Panel C.

After 6 weeks the ulcer healed completely (Figure 3.19).

Keywords: Convex triangular foot; hallux valgus; quintus varus

HALLUX VALGUS, OVERRIDING TOE, CLAW TOES, EDEMA

A 68-year-old female patient with type 2 diabetes diagnosed at the age of 45 years attended the outpatient diabetic foot clinic

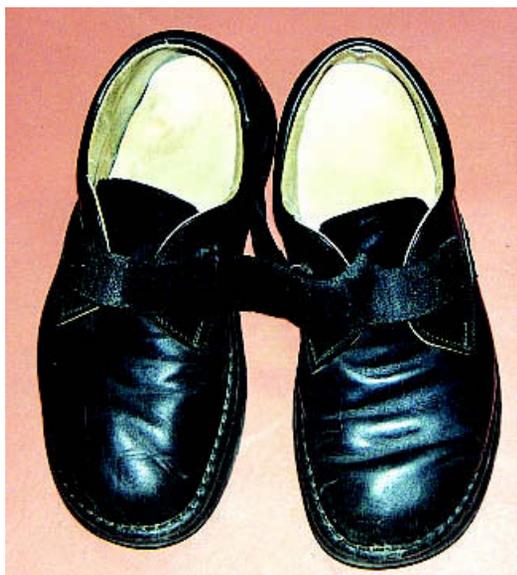


Figure 3.17 Extra-depth shoes and custom insoles

for routine chiropody treatment. She was being treated with insulin. The patient had hypertension, advanced background retinopathy which had been treated with laser in both eyes, and diabetic nephropathy (urine protein: 2.6 g/24 h). On examination, she had severe diabetic neuropathy and gross ankle edema due to nephropathy. Peripheral pulses were normal and the ankle brachial index was 1.1 on both feet. Mild hallux valgus, claw toes, overriding of the second to the third toe and lateral drip of the toes were observed (Figures 3.20 and 3.21). Callus formation at the inner aspect of the first and on the second metatarsal heads was noted. Fat pads on the first, second and third metatarsal heads were displaced distally to the base of the proximal phalanges due to clawing of the toes. A superficial painful infected ulcer at the dorsum of the second toe was also present, due to overriding and clawing of the toes. Debridement of the callus was carried out. The patient was put on clindamycin for 2 weeks. Treatment with frusemide 40 mg daily was also commenced to reduce edema. Extra depth

shoes and orthotic insoles were prescribed in order to reduce the pressure on the plantar area and the friction from the shoes on the deformed toes.

The correct shoes and orthotic insoles are often enough to reduce the risk for foot ulceration in the majority of the patients with foot deformities and loss of protective sensation. In addition, edema has a detrimental effect on the foot at risk, as it reduces local blood supply and has been associated with increased risk for ulceration. Therefore, reduction of ankle edema is recommended for patients at risk for ulceration.

Beyond diabetic nephropathy, other causes of ankle edema in diabetes include heart failure and diabetic neuropathy. Edema due to neuropathy is not rare. This form of edema results from sympathetic denervation, which leads to loss of the vasomotor reflex upon standing, arteriovenous shunting and increased capillary pressure. Neuropathic edema responds to the administration of the sympathomimetic agent ephedrine.

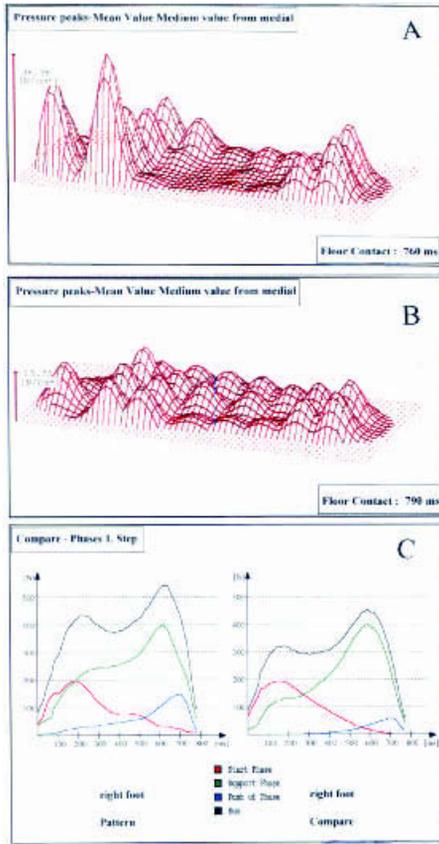


Figure 3.18 In-shoe plantar pressure measurements (A) when the patient used her own shoes; and (B) after wearing the prescribed insole and shoe. (C) Pressures on the sole of the patient's foot during walking in her own shoes (graph on left), and when wearing the custom-made insole (graph on right)

Keywords: Hallux valgus; toe overriding; claw toes; edema

ONYCHOMYCOSIS; HALLUX VALGUS AND HAMMER TOE DEFORMITY

A 68-year-old female patient with diabetes diagnosed at the age of 50 years and



Figure 3.19 The neuropathic ulcer shown in Figure 3.15 after it had healed following 6 weeks of treatment

treated with insulin, was referred to the outpatient diabetic foot clinic because of foot deformities and recurrent superficial toe ulcers.

The patient had findings of peripheral neuropathy. Peripheral pulses were palpable. No other diabetic complications were present.

Onychomycosis was noticed and confirmed by direct microscopic examination of nail specimens. The skin on her feet was dry; hallux valgus and hammer toe deformity of her second left toe were observed. Tiny superficial ulcers on the dorsum of her second and third toes due to shear pressure were present, as well as a small ulcer on the inner aspect of her great toe, and



Figure 3.20 Hallux valgus, overriding toe, claw toes and edema

a hemorrhagic callus on the tip of the left great toe (Figures 3.22 and 3.23).

Mild hallux valgus and hammer toe deformity on the right second and third toes was apparent, with a superficial ulcer on the dorsum of the second toe (Figure 3.24). Hammer toe is a complex deformity consisting of contraction (hyperflexion) of the proximal interphalangeal joint, while the metatarsophalangeal joint is either dorsiflexed or in the neutral position. The distal interphalangeal joint may be in the neutral position, hyperextended or in plantar flexion (Figure 3.25). Hammer toe may be flexible or rigid.

Overriding toe deformity often occurs in the second and the fifth toes. The cause of the overriding fifth toe is mainly

congenital, while a second overriding toe is acquired and multifactorial. Elongation and laxity of the plantar synovium bursa of the metatarsal joint result in dorsal subluxation of the affected joint. The second toe lacks plantar interossei muscles, therefore lumbrical muscles predominate, causing dorsiflexion of the toe. Subluxation of the metatarsophalangeal joint results in shrinkage of the dorsal synovium bursa and the dorsal interossei muscles. Further atrophy of the intrinsic muscles contributes to the development of the deformity which may be fixed or flexible.

Debridement of the calluses and instruction in foot care was provided to this patient, and shoes with a high toe box and shock absorbing insoles were prescribed.



Figure 3.21 Hallux valgus, overriding toe, claw toes and edema. Plantar aspect of the foot illustrated in [Figure 3.20](#)

Keywords: Onychomycosis; hallux valgus; hammer toe deformity

MALLET TOE

A mallet toe consists of plantar flexion of the distal interphalangeal, and neutral position of metatarsophalangeal and proximal interphalangeal joints ([Figure 3.26](#)).

Toe deformities (hammer, claw, curly, mallet toe and overriding of toes) are unknown in non-shoe wearing populations. Their incidence varies from 2 to 20%, and increases with age. Women are affected four to five times more often than men.

Most people have no underlying disease, although neuromuscular diseases and inflammatory arthropathies may be accompanied by such toe deformities.

Toe deformities are more common in people with diabetes, due to muscle atrophy and limited joint mobility. Deformities such as those described above, when present in a patient with loss of sensation due to diabetic neuropathy, pose a risk for the development of neuropathic ulcers, as prominences are susceptible to skin-on-shoe friction. Patients are instructed to check their feet every day. Shoes with a high toe box protect the deformed toes from ulceration.



Figure 3.22 Hallux valgus, toe overriding and onychomycosis



Figure 3.24 Mild hallux valgus and hammer toe deformity on the right second and third toes, with a superficial ulcer on the dorsum of the second toe. Right foot of the patient whose feet are shown in [Figures 3.22](#) and [3.23](#)



Figure 3.23 Hammer toe deformity of the second, third and fourth toes, hemorrhagic callus and onychomycosis. Anterolateral view of the foot shown in [Figure 3.22](#)



Figure 3.25 Hammer toe



Figure 3.26 Mallet toe

Keywords: Mallet toe; toe deformities

PROMINENT METATARSAL HEADS AND CLAW TOES

A 65-year-old male patient with longstanding type 2 diabetes attended the outpatient

diabetic foot clinic for callus removal and treatment of ulcers on the tip of his second and fifth right toes (Figure 3.27).

On examination, he had bounding pedal pulses, and severe peripheral neuropathy. Metatarsal heads were prominent, and claw toes were present.

Claw toe deformities may cause prominence of metatarsal heads with subsequent



Figure 3.27 Prominent metatarsal heads and claw toes

callus formation and ulceration. Ulcers may develop at the tips of the claw toes, since they are abnormally exposed to pressure during walking.

Protective footwear (high toe box and orthotic insoles) was provided to this patient.

Keywords: Claw toes; prominent metatarsal heads

POSTOPERATIVE HALLUX VALGUS AFTER SECOND TOE REMOVAL

A 55-year-old female patient with type 2 diabetes diagnosed at the age of 40 years, treated with insulin, and erratic glycemic control, visited the diabetic foot clinic because of recurrent callus formation. She

had background retinopathy, hypertension, and severe peripheral neuropathy, and a history of amputation of her left second toe 3 years previously due to osteomyelitis after a perforated ulcer.

After removal of her second toe, her left great toe gradually dislocated to a valgus posture, overriding the adjacent (third) toe (Figure 3.28). Gross callus formation developed at the medioplantar aspect of the first metatarsal head, which caused constant discomfort during walking and dancing (Figure 3.29). Callus was also noticed over the third metatarsal head.

At the outpatient clinic the callosity was removed and a full thickness ulcer revealed. More callus built up quickly as a result of the very active lifestyle of the patient and her refusal to wear appropriate footwear, she therefore had to attend the clinic every week.

A plain X-ray showed disarticulation of the left second toe, dislocation of the



Figure 3.28 Hallux valgus and toe overriding after second toe disarticulation



Figure 3.29 Gross callus formation on the first and third metatarsal heads after second toe disarticulation

metatarsophalangeal joint of the great toe, medial pronation of the first metatarsal head, and hallux valgus deformity with rotation, together with dislocation of the sesamoids, and arthritis; necrosis of the head of the third metatarsal bone was also evident (Figure 3.30).

She was referred to the orthopedic department where her second metatarsal was removed. The hallux valgus deformity was corrected by arthrodesis of the metatarsophalangeal joint.

After the operation there was no significant callus development within the next 3 months (Figure 3.31).

Keywords: Hallux valgus; prophylactic surgery; second ray amputation

FIRST RAY AMPUTATION

A 72-year-old male patient with type 2 diabetes diagnosed at the age of 56 years and



Figure 3.30 X-ray image of the foot illustrated in Figures 3.28 and 3.29. Disarticulation of the left second toe, dislocation of the metatarsophalangeal joint of the great toe, medial pronation of the first metatarsal head, and hallux valgus deformity with rotation, together with dislocation at the sesamoids and arthritis; necrosis of the head of the third metatarsal bone is also evident

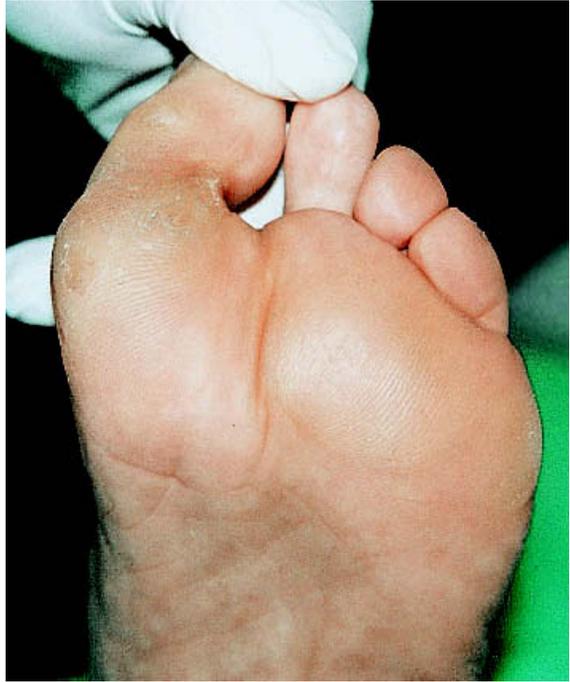


Figure 3.31 Photograph of the foot shown in Figures 3.28–3.30 3 months after arthrodesis of the first metatarsophalangeal joint and second ray amputation. Note the absence of significant callus formation

treated with sulfonylurea and metformin, attended the outpatient diabetic foot clinic because of a deep, infected neuropathic ulcer under the first metatarsal head. His diabetes control was acceptable (HbA_{1c} : 7.6%). He had a history of hypertension and dyslipidemia and was being treated with a combination of angiotensin converting enzyme inhibitor with diuretic and simvastatin. He had neuropathic pain in his feet. He described the pain as a burning sensation which worsened at night. On examination, an ulcer 3×3 cm in size and 1.5 cm in depth surrounded by callus formation was seen on the left first metatarsal head. Its base was sloughy. Second left claw toe deformity was also observed. Pedal pulses were palpable, the ankle brachial index was 1.0; the patient had findings of severe peripheral neuropathy (loss of sensation of light touch, pain, temperature, vibration, and 5.07 monofilaments; the

vibration perception threshold was 35 V on both feet).

A plain radiograph revealed osteomyelitis of the first metatarsal head extending to the base of the proximal phalanx of the great toe. Cultures of the base of the ulcer revealed the presence of *Staphylococcus aureus* and *Escherichia coli*. Based on the results of the swab culture he was given amoxicillin and clavulanic acid for 2 weeks. After this time a first ray amputation under local anesthesia was carried out. A culture of the bone was negative for pathogens but pathologic examination of the resected bone showed findings of chronic osteomyelitis (granulated fibrous tissue with a predominance of plasma cells and lymphocytes and involucrum formation at the periosteum). The postoperative period was free from complications and the wound healed well in 2 weeks (Figure 3.32). Antibiotics



Figure 3.32 First ray amputation due to osteomyelitis

were discontinued 7 days after the operation and he was put on imipramine for the neuropathic pain.

Removal of the great toe results in dysfunction of the foot during both stance and propulsion. This disability is related to the length of the removed metatarsal shaft. Most surgeons preserve the longest metatarsal shaft possible. The base of the proximal phalanx should be preserved, in order to keep the attachment of the short flexor of hallux intact, thus keeping sesamoids in place and maintaining the windlass mechanism. This mechanism protects the first metatarsal head from overloading during the propulsion phase of gait. In the case of an obligatory removal of hallux — due to osteomyelitis of the proximal phalanx — the surgeon should preserve all uninvolved portions of the

metatarsal, except the avascular sesamoids and their fibrocartilaginous plate. A hallux disarticulation at the metatarsophalangeal joint exposes the head of the third metatarsal to abnormally high pressure during stance, and may displace the second toe medially.

Keywords: First ray amputation; histology; chronic osteomyelitis

CALLUS UNDER BONE PROMINENCE

A 72-year-old male patient with type 2 diabetes attended the outpatient diabetes clinic for his usual follow-up. His diabetes control was fair with glibenclamide. He was free



Figure 3.33 Callus over prominence of metatarsal head

from retinopathy or nephropathy, but he had severe diabetic neuropathy. On examination a callus was present under the head of his right third metatarsal, which caused minor discomfort (Figure 3.33). Another bony prominence was evident on the outer aspect of his fifth metatarsal, without callus formation. Claw toes, onychomycosis and dry skin were also present. The callus was removed, and a tiny superficial ulcer revealed. The patient was prescribed extra depth shoes with orthotic insoles (preventive footwear). Hydrating cream was used to prevent skin cracking.

Keywords: Callus; claw toes; dry skin

CALLUS OVER PROMINENT METATARSAL HEADS

A 70-year-old female patient who had type 2 diabetes since the age of 50 years and was being treated with insulin, attended the foot clinic for chiropody treatment. She had a history of ischemic heart disease (myocardial infarction and stroke), peripheral vascular disease treated with low dose

of aspirin; and proliferative retinopathy. She complained of numbness in both feet and a deep aching pain in her calves and painful heel cracks.

On examination, peripheral pulses were absent and her ankle brachial index was 0.8 on the left and 0.7 on the right. The vibration perception threshold was 30 V in both feet. Achilles tendon reflexes were absent, and pain, temperature, light touch and vibration sensation were severely diminished. Pes cavus and hallux valgus were present on both feet (most prominent on the left), together with an obvious prominence of her metatarsal heads and callus formation. The fat pads of her metatarsal heads were translocated towards the toes. The skin on her feet was dry (Figure 3.34). The calluses were debrided on a regular basis, and appropriate footwear was prescribed. Heel cracks (see Figure 4.6) persisted despite debridement.

Calluses develop in areas of high pressure in the feet as a physiological reaction of the skin in response to loading. A callus adds further pressure to the underlying tissues functioning as a foreign body under the foot. Prospective studies have shown that regular removal of calluses reduces the risk of foot ulceration.

Keywords: Prominent metatarsal heads; callus

HEMORRHAGIC CALLUS

A 64-year-old male patient with type 2 diabetes diagnosed at the age of 47 years attended the outpatient diabetic foot clinic because of an ulcer under his right foot.

On examination, a painless ulcer surrounded by a hemorrhagic callus was seen under the third metatarsal head (Figure 3.35). Claw toe deformity, a curly



Figure 3.35 A neuropathic ulcer under a hemorrhagic callus

Figure 3.34 Callus over prominence on metatarsal heads. Pes cavus and hallux valgus



fourth toe, and a heloma molle in the fourth interdigital space were also observed. The patient had bounding peripheral pulses and severe peripheral neuropathy. After sharp debridement of his callus, an ulcer of dimensions 2.0×1.5 cm and depth 1 cm was revealed. Plantar fascia was exposed. A plain radiograph excluded osteomyelitis. The patient was instructed in foot care. Offloading of the ulcer area was achieved by the use of an 'almost half' shoe (Figure 3.36) and a total-contact orthotic insole, with a window under the ulcer area. These shoes cause instability, so the patient was instructed to use a crutch. The ulcer healed completely in 8 weeks.

The cause of the ulcer in this patient was high plantar pressure under his prominent metatarsal heads (Figure 3.37). After the ulcer had healed, protective footwear (extra depth shoes and custom-made insoles) was prescribed in order to reduce the peak pressure on the third metatarsal head. No relapse of the ulcer occurred in the subsequent months.

Keywords: Hemorrhagic callus; half shoes; protective footwear



Figure 3.36 Therapeutic half shoe for the treatment of forefoot ulcers

ULCER UNDER A CALLUS AREA

A 70-year-old male patient with longstanding type 2 diabetes attended the outpatient diabetic foot clinic for callus removal on his right foot. On examination, a neuropathic ulcer surrounded by callus was noticed under his fourth metatarsal head (Figure 3.38). He had normal peripheral pulses and severe peripheral neuropathy. Claw toes, varus deformity of the foot and prominent metatarsal heads on his right foot

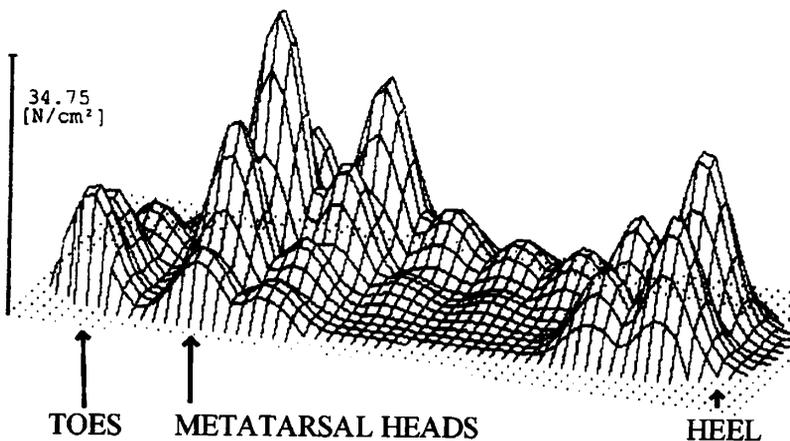


Figure 3.37 Peak plantar pressures recorded with a pedobarograph

were observed. Discoloration of the skin on the lower tibia due to venous insufficiency was also evident. The callus was debrided. Shoes and insoles similar to those shown in [Figure 3.36](#) were prescribed until the ulcer healed. The cause of the ulcer in this patient was the callus resulting from high plantar pressures. High peak pressures are present in almost all cases where there are prominent metatarsal heads due to claw toe deformity. Prevention of callus formation is necessary to avoid recurrence of the ulcer. Protective footwear was prescribed after the ulcer had healed.

Keywords: High plantar pressure; callus, prominent metatarsal heads; varus deformity



Figure 3.38 A neuropathic ulcer under a callus

ULCER UNDER HALLUX

A 70-year-old male patient with longstanding type 2 diabetes treated with insulin and sulfonylurea, attended the outpatient diabetic foot clinic because of a hemorrhagic callus under the phalangophalangeal joint of the right hallux ([Figure 3.39](#)). He had ischemic heart disease, hypertension, peripheral vascular disease, background retinopathy and microalbuminuria. The patient had severe diabetic neuropathy; the ankle brachial index was 0.7. After his callus was debrided a clean neuroischemic ulcer was revealed. A plain radiograph excluded osteomyelitis. Therapeutic footwear was prescribed and the ulcer healed in 6 weeks.

The forefoot is the usual site for ulceration. In one series, ulcers of the forefoot accounted for 93% of all foot ulcers. Almost 20% of the ulcers developed under the hallux, 22% over the metatarsal heads, 26% on the tips of the toes and 16% on the dorsum of the toes. Ulcer under the hallux is associated with rigid hallux and high peak pressures on this area.

Keywords: Hemorrhagic callus; prevalence of foot ulceration

HEEL CRACKS

Painful heel cracks due to dry skin were noted in the patient whose feet are shown in [Figure 3.34](#) ([Figure 3.40](#)).

Dry skin in diabetic patients is caused by sympathetic cholinergic denervation of the sweat glands in their feet. Patients with dry foot skin often develop reactive hyperhidrosis of the upper body. Heel cracks



Figure 3.39 Hemorrhagic callus under the hallux



Figure 3.40 Heel cracks

may become infected and may lead to deep ulcers with calcaneous involvement if left untreated. The crack resists healing, despite the correct foot care. Heel cracks are aggravated by microvascular disease and neuropathy, and resist healing, despite adequate foot care. Local application of hydrating creams—avoiding the areas between the toes—is the treatment which is usually recommended.

Keywords: Dry skin; heel cracks

BILATERAL CHOPART DISARTICULATION

A 73-year-old male patient with type 2 diabetes diagnosed at the age of 61 years attended the outpatient diabetes foot clinic for a chronic ulcer under his left partially amputated foot. He had had bilateral mid-tarsal (Chopart) disarticulations (on the right foot at the age of 66 years and on the left foot at the age of 68 years) because

of infected foot ulcers under the metatarsal heads complicated by osteomyelitis.

On examination, his feet pulses were palpable, but the patient had severe peripheral neuropathy. A full thickness neuropathic ulcer, which developed 2 months after the amputation, was evident on the plantar area of the left foot (Figure 3.41). The patient had never used any ankle prosthesis or orthosis, but instead used crutches and shoes with a firm outsole and a soft molded insert. The ulcer healed for a period of only 2 months, when the patient was hospitalized because of a hip fracture.

Chopart disarticulation is performed through the talonavicular and calcaneocuboid joints, preserving the hindfoot only (talus and calcaneus). As no muscles attach to the talus, all active dorsiflexion of the remaining short foot is lost. However, dorsiflexion can be restored, by reattaching the anterior tibial tendon to the neck of the talus. Chopart disarticulation preserves the normal length of the leg and the patient can undertake limited walking without a prosthesis. Reasonable walking is possible by the use of an intimately fitting fixed-ankle prosthesis or orthosis placed into a shoe with a rigid rocker bottom.

In the present case, walking without crutches was not possible even if an appropriate prosthesis was used because of the bilateral Chopart disarticulation. However, the use of a prosthesis and offloading the pressure on the ulcerated area with suitable insoles helped to heal the ulcer. In addition, the patient's severe instability, which was the cause of the hip fracture, was reduced.

Any type of amputation alters the biomechanics of the foot and is considered to be a risk factor both for a recurrence of foot ulceration and for a new amputation. Several studies have shown that previous amputations account for 30–50% of new amputations on the same or the contralateral foot within the following 5 years.

Keywords: Neuropathic ulcer; mid-tarsal disarticulation; Chopart disarticulation

NEUROPATHIC ULCER

An ostensibly small neuropathic ulcer surrounded by callus formation was present under the fourth metatarsal head



Figure 3.41 Full thickness neuropathic ulcer in a patient with Chopart disarticulation



Figure 3.42 A neuropathic ulcer under callus formation in a patient with fourth toe disarticulation

(Figure 3.42) of a patient with severe diabetic neuropathy. A history of fourth toe disarticulation at the metatarsophalangeal joint was reported to have occurred 2 years previously because of osteomyelitis in the proximal phalanx. Claw second and third toe, quintus varus (due to fourth toe disarticulation), dry skin and heel cracks were also present. The real size of the ulcer was $1.5 \times 1.5 \times 1.0$ cm post-debridement. The little toe diverged medially and the

third toe laterally. Therapeutic footwear was prescribed and the ulcer healed in 2 months.

A fourth ray amputation may lead to better functional and cosmetic results. Sole incisions pose a risk for ulceration; therefore incisions are carried out on the dorsum or the side of the foot. Scar tissue which has healed over an ulcer may predispose to new ulceration in a similar manner to callus formation.



Figure 3.43 Onychocryptosis (ingrown nail) of both halluxes. Note brown nail discoloration probably caused by chronic infection with *Candida albicans*. Second, third, and fourth left claw toe deformity

Keywords: Fourth toe disarticulation; neuropathic ulcer

INGROWN NAILS (ONYCHOCRYPTOSIS)

An ingrown toenail is a common condition usually affecting the hallux. A section of a nail curves into the adjacent flesh and becomes embedded in the soft tissue (Figure 3.43). Peeling the nail at the edge or trimming it down at the corners is the most common cause. In addition to congenital or traumatic reasons, ingrown nails may be caused by tight shoes or socks which press

on the sides of the nail making it curve into the skin.

An ingrown nail predisposes to local infection (paronychia) as it provides an entry point for pathogens; therefore it should be treated as soon as it is recognized. Nails should be trimmed in a straight line.

Infection with *Candida albicans* is another cause of chronic paronychia, especially when patients' feet are exposed to moisture for long periods. The nail is usually affected and becomes ridged, deformed and brown.

Keywords: Onychocryptosis; ingrown nail

Chapter IV

SOME UNCOMMON CONDITIONS

- ONYCHOGRYPOSIS
- PALMOPLANTAR KERATODERMA
- CALCIUM PYROPHOSPHATE DIHYDRATE (CPPD) DEPOSITION DISEASE
- HYPERKERATOTIC ECZEMA
- NECROBIOSIS LIPOIDICA
- SQUAMOUS CELL CARCINOMA
- DERMATOFIBROSARCOMA PROTUBERANS

ONYCHOGRYPOSIS

A 75-year-old male patient with type 2 diabetes diagnosed at the age of 64 years was referred to the foot clinic for foot care. He was a psychiatric patient treated on an out-patient basis. The patient had findings of peripheral neuropathy with loss of sensation of pain, light touch, vibration and temperature. Peripheral pulses were palpable. Claw toes and extreme onychogryposis was noted (Figure 4.1). His nails were cut using a special nail trimmer. Instruction in foot care was given; extra depth shoes were provided in order to accommodate the deformity. He visited the clinic on a monthly basis and had his nails cut without any other foot problems.

Onychogryposis is caused by chronic repetitive trauma particularly to the nails on the great toe. The nails may be grossly thickened, hard and very elongated (Figure 4.2 shows this condition in another patient). They may be elevated from the nail bed, curved inwards or turned sideways.

The deformed nail can press against another toe causing ulcerations. When the patient does not wear shoes, the deformed toenail often grows vertically. When socks or shoes are being worn, the deformed toenails tend to develop in such a way as to accommodate the clothing.

Keywords: Onychogryposis

PALMOPLANTAR KERATODERMA

A 64-year-old male patient with type 2 diabetes diagnosed at the age of 55 years attended the foot clinic for foot care and instruction in the management of his condition, palmoplantar keratoderma. On examination diffuse thickening of the palmar and plantar skin, together with hyperkeratosis was noted (Figure 4.3). Nail deformities were also observed. He had findings of peripheral neuropathy, while the peripheral arteries were palpable.



Figure 4.1 Onychogryposis



Figure 4.2 Onychogryphosis



Figure 4.3 Palmoplantar keratoderma

The patient was instructed in appropriate foot care. Local debridement with keratolytics was prescribed. Protection from friction with soft insoles may be helpful in this condition.

Palmoplantar keratoderma is an autosomal-dominant trait characterized by diffuse,

thickened hyperkeratosis of the palms of the hands and soles of the feet. The hyperkeratosis may be so thick that the skin may crack, especially in dry, cold weather. Infection with *Tinea pedis* frequently occurs as the fissures provide a portal of entry for the fungus. The nails on

the hands and toes may be dystrophic and become infected with fungus.

Keywords: Palmoplantar keratoderma

CALCIUM PYROPHOSPHATE DIHYDRATE (CPPD) DEPOSITION DISEASE

A 74-year-old female with type 2 diabetes diagnosed at the age of 68 years and treated with sulfonylurea with acceptable diabetes control, was referred to the outpatient diabetic foot clinic for possible osteomyelitis of her fifth left toe. She had intense pain at this site when resting and walking. The pain started after the patient had worn a tight pair of shoes for a few hours.

On examination, redness, edema, and callus formation were noted at the outer aspect of the left fifth toe (Figure 4.4). She had findings of diabetic neuropathy (no

sensation of vibration, no Achilles tendon reflexes, but she could feel pinpricks; vibration perception threshold was 45 V on both feet). Peripheral pulses were palpable.

Debridement of the callus revealed a cheesy material emanating from the base of a superficial ulcer. A culture of this material did not reveal any microorganisms. A plain radiograph showed radiodense deposits at the articular bursae of the distal interphalangeal joint; no osteomyelitis was apparent (Figure 4.5). Examination of this material with compensated polarized light microscopy showed rhomboid-shaped and weakly positive birefringent crystals, which is typical of CPPD deposition disease.

The patient was advised to rest. She visited the foot clinic on a weekly basis for callus debridement. The ulcer healed completely in 3 weeks.

CPPD deposition disease (or pseudogout) of the foot joints may pose a problem with diagnosis when the location is atypical. The knee is the most frequent joint affected by pseudo-gout, followed by the



Figure 4.4 Painful inflammatory lesion of the fifth toe, due to calcium pyrophosphate dihydrate deposition disease



Figure 4.5 Radio-dense deposits at the articular bursae of the distal interphalangeal joint of the fifth toe due to calcium pyrophosphate dihydrate deposition disease

wrist, shoulder, ankle, elbow and hands, although every joint can be affected. Treatment includes rest, aspiration of the joint fluids, and systemic use of non-steroidal anti-inflammatory medication.

Keywords: Pseudo-gout; calcium pyrophosphate dihydrate deposition disease; CPPD deposition disease

HYPERKERATOTIC ECZEMA

A 65-year-old male patient with longstanding diabetes visited the outpatient diabetic foot clinic for a chronic pruritic lesion of his left foot.

On examination, he had severe diabetic neuropathy and peripheral pulses were palpable. A hyperkeratotic lesion with dense yellowish scales over a red skin patch was observed on the plantaro-lateral aspect of his left foot (Figure 4.6). The scales were firmly adherent on the epidermis, and not

easily debrided. Dry skin on the heel was also present.

The patient was referred to the dermatology department for treatment.

This situation occurs on the palms of the hands and soles of the feet, almost exclusively in men. It may result from irritation or allergy, although the cause is usually unknown. Topical moisturizers containing lactic acid or urea are applied after soaking the affected area for 20 min. Topical coal tar preparations may be applied daily under occlusion if severe lichenification is present. Per os antihistaminic medication, or low-dose corticosteroids may be of some help for short periods.

Keywords: Hyperkeratotic eczema

NECROBIOSIS LIPOIDICA

A 50-year-old lady with type 1 diabetes diagnosed at the age of 38 years, visited the outpatient clinic for control of her



Figure 4.6 Hyperkeratotic eczema. Hyperkeratotic lesion with dense yellowish scales over a red skin patch. The scales are firmly adhered to the epidermis and not easily debrided with a blade. Dry skin on the heel

diabetes (HbA_{1c} : 7.0–7.9%) on a regular basis. She was free of micro- or macro-angiopathy. Two years after her diabetes was diagnosed, the patient noticed a few small, red, irregular, violaceous papules on the dorsum of her feet. These papules slowly enlarged, coalesced and became scaly, irregular plaques, with minimal central atrophy, and an advancing red border (Figure 4.7). New lesions appeared on her left ankle and right leg. Apart from causing cosmetic problems, the lesions were asymptomatic.

A biopsy of these lesions showed necrobiosis lipoidica (formerly ‘necrobiosis lipoidica diabetorum’), an unusual disorder of unknown mechanism, strongly associated with diabetes mellitus but also found in subjects with normal or abnormal glucose tolerance.

Typically, such lesions occur on the anterior shin of both lower legs, but they also may be located on the arms, hands or head. They may precede the diagnosis of diabetes, and sometimes they



Figure 4.7 Necrobiosis lipoidica. Scaly, irregular plaques, with minimal central atrophy and an advancing red border

are pruritic, dysesthetic or painful. They ulcerate—usually after a trauma (shown in Figures 4.8 and 4.9 in other patients)—in approximately 35% of the cases, but do not usually lead to infection.

Histological examination of the lesions shows necrobiosis, which provides the foci for ‘hyalinized’ collagen bundles (Figure 4.10), fibrosis, histiocyte infiltration (Figure 4.11) and granulomata (Figure 4.12). Capillary walls become thickened (Figure 4.10).

Topical application of corticosteroids may have fair results against progression of



Figure 4.8 Ulcerated lesions associated with necrobiosis lipoidica. Note a not ulcerated lesion over the epiphysis of the fibula



Figure 4.9 Infected ulcerated lesions associated with necrobiosis lipoidica

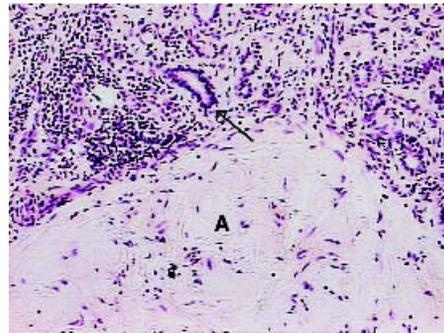


Figure 4.10 Histological findings in samples taken from the patient with necrobiosis lipoidica shown in Figure 4.7. A, foci of ‘hyalinized’ collagen bundles (necrobiosis), fibrosis, and histiocyte infiltration. Thickened capillary walls are evident (arrow). H&E stain, $\times 100$

the lesions. Various other agents have been tried, such as aspirin or pentoxifyllin, with mixed results.

Keywords: Necrobiosis lipoidica

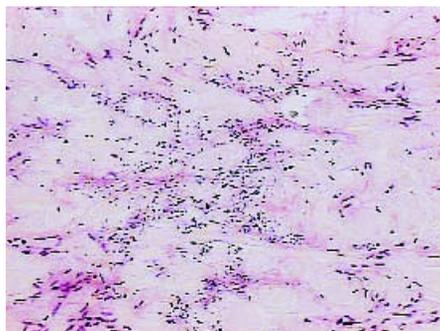


Figure 4.11 Histological findings in samples taken from the patient with necrobiosis lipoidica shown in Figure 4.7. Foci of 'hyalinized' collagen bundles (necrobiosis), fibrosis and histiocyte infiltration can be seen. H&E stain, $\times 100$



Figure 4.12 Histological findings in samples taken from the patient with necrobiosis lipoidica shown in Figure 4.7. Note the presence of a granuloma (arrow). H&E stain, $\times 100$

SQUAMOUS CELL CARCINOMA

Squamous cell carcinoma (SCC) developed on a neglected burn scar in a 48-year-old diabetic male patient. SCC is the second most common skin cancer after basal cell carcinoma. It arises from the dermis and it is most common in areas exposed to the sun. It is an aggressive and invasive

cancer; it may penetrate underlying tissues, and it metastasizes in distant tissues, lymph nodes, and organs. Presentations vary and for this reason the neoplasm is difficult to diagnose. Pink, red or tan plaques, ulcers (Figure 4.13) or erosions and scaling may be apparent. Secondary SCC arises in areas of old scars, especially burn scars, chronic non-healing wounds and radiation lesions (Marjolin's ulcers). Plastic surgeons used a free latissimus dorsi musculocutaneous flap to repair the defect, after extensive



Figure 4.13 Squamous cell carcinoma presented as an ulcer on the lateral aspect of the foot of a diabetic patient. The design of the excision and the recipient vessels are indicated. (Courtesy of O. Papadopoulos)



Figure 4.14 Free latissimus dorsi musculocutaneous flap used to repair the defect, after extensive excision of a squamous cell carcinoma. Patient whose foot is shown in [Figure 4.13](#). (Courtesy of O. Papadopoulos)

removal of the cancer, within a healthy border ([Figure 4.14](#)).

Keywords: Squamous cell carcinoma

DERMATOFIBROSARCOMA PROTUBERANS

Recurrent dermatofibrosarcoma protuberans (DFSP) was diagnosed in a 71-year-old

male diabetic patient. DFSP is an uncommon aggressive soft tissue sarcoma of low malignant potential, arising in the dermis of young to middle-aged adults and it is slightly more frequent in men than women (57 versus 43%). It is most commonly located on the trunk and proximal extremities. Initially it presents as an asymptomatic bluish, red or flesh-colored nodule with a diameter of a few millimeters



Figure 4.16 A free latissimus dorsi musculocutaneous flap was used to repair the defect, after extensive excision of a dermatofibrosarcoma protuberans of heel. Patient of [Figure 4.15](#). (Courtesy of O. Papadopoulos)



Figure 4.15 Ulceration of recurrent dermatofibrosarcoma protuberans of the heel of a diabetic patient. The design of the excision and the recipient vessels are indicated. (Courtesy of O. Papadopoulos)

to >20 cm located on top of plaque-like lesions, or as superficial ulceration of some of these nodules. It infiltrates the surrounding tissues and, if untreated, it may ulcerate (Figure 4.15). It may recur after surgical excision and lead to metastases. Moh's micrographic surgery, using wide margin resection, is the mainstay of treatment.

Plastic surgeons used a free latissimus dorsi musculocutaneous flap to repair the defect, after wide removal of the cancer (Figure 4.16).

Keywords: Dermatofibrosarcoma protuberans

Chapter V

NEUROPATHIC ULCERS AT VARIOUS SITES

- NEUROPATHIC ULCER OVER PROMINENT FIRST METATARSAL HEAD
- NEUROPATHIC ULCER OVER PROMINENT METATARSAL HEADS
- NEUROPATHIC ULCER OVER A COLLAPSED MIDFOOT
- NEUROPATHIC ULCER UNDER FOURTH METATARSAL HEAD
- NEUROPATHIC ULCERS UNDER PROMINENT METATARSAL HEADS
- ULCERS OVER A CHARCOT FOOT
- A NEUROPATHIC ULCER UNDER THE HEEL
- BURNS ON TOES AND FOREFOOT
- CHRONIC NEUROPATHIC ULCER COMPLICATED BY OSTEOMYELITIS
- ON THE TOES
- ON THE MIDFOOT
- ON THE HEEL

NEUROPATHIC ULCER OVER PROMINENT FIRST METATARSAL HEAD

A 54-year-old male patient with type 2 diabetes diagnosed at the age of 45 years was referred to the outpatient diabetic foot clinic because he had developed an ulcer on the plantar area of his left foot. He was treated with antidiabetic tablets and diabetes control was good (HbA_{1c} : 7.1%). On examination he had a full thickness ulcer on the head of the first metatarsal in an area where there was gross callus formation (Figure 5.1). No signs of infection were observed. He had findings of diabetic neuropathy (no sensation of pain, light touch, temperature or vibration). Vibration perception threshold was 45 V on the right and >50 V on the left foot. His peripheral

pulses were palpable and the ankle brachial pressure index was 1.2 bilaterally.

The patient did not have a previous history of problems with his feet. He denied any pain or trauma. He was aware of the presence of the ulcer, after he had seen discharge on his socks and the insole of his shoes. Debridement of the ulcer was carried out and the patient was advised to rest his feet; therapeutic footwear was also prescribed (Figure 5.2). This patient attended the diabetic foot clinic on a weekly basis and he changed the dressings every day. The ulcer healed completely in 10 weeks.

This is a typical neuropathic ulcer. Such ulcers are painless—unless they become infected—and develop in patients with neuropathy under areas of high-pressure loading. A callus forms at points of high repetitive pressure on the sole of the foot



Figure 5.1 Neuropathic ulcer on the first metatarsal head



Figure 5.2 Therapeutic footwear prescribed for the patient whose foot is shown in [Figure 5.1](#). Among the most commonly used therapeutic footwear is the rocker style shoe. Notice that the apex (ridge) of the rocker sole is located behind the metatarsal heads

and is a powerful predictor of ulceration. Such areas are the metatarsal heads and the plantar aspect of the great toe. Callus formation on the heel is not very common. In addition, calluses can develop over areas of bony prominences at other sites in the case of foot deformities (claw and hammer toes, toe overriding, neuro-osteoarthropathy). Even though the etiology of callus formation has not been determined, the fact that a callus acts as a foreign body in the shoe and contributes to high plantar pressure is well known. It is therefore recommended that callus formation should be prevented and when a callus is present, it should be removed regularly. Appropriate footwear is thought to prevent callus formation and the efficacy of this measure will be reflected by the proportion of patients wearing the correct footwear who develop ulcers. Hemorrhage into a callus is known as a 'pre-ulcer' and it should be treated as an ulcer.

Keywords: Neuropathic foot ulcer

NEUROPATHIC ULCER OVER PROMINENT METATARSAL HEADS

A 53-year-old female patient who had had type 2 diabetes since the age of 41 years and was being treated with insulin, was referred to the outpatient foot clinic because of a chronic foot ulcer. She had background retinopathy, cataract, hypertension and ischemic heart disease. The patient complained of numbness and a sensation of pins and needles in her feet, which worsened during the night.

On examination she was found to have a full thickness ulcer under her second and third prominent metatarsal heads and claw toes ([Figure 5.3](#)). The patient had severe peripheral neuropathy (no sensation of light

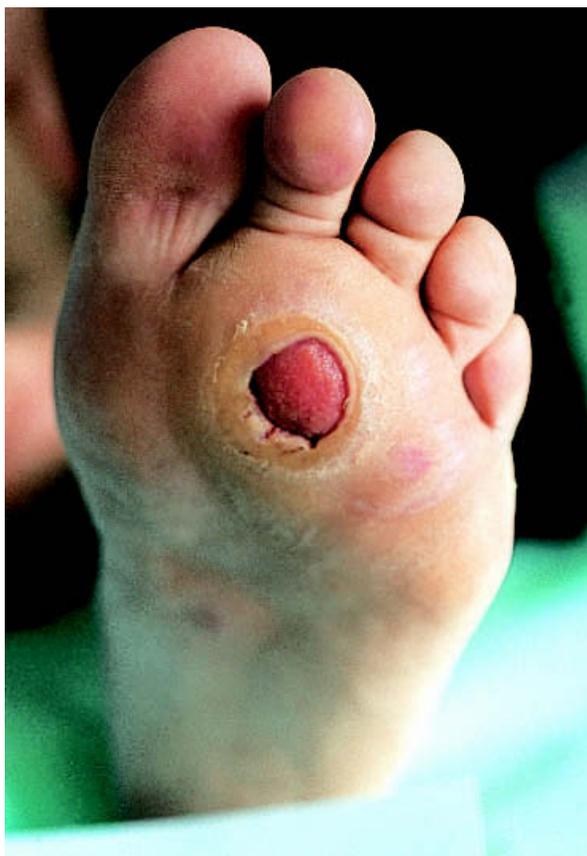


Figure 5.3 Neuropathic ulcer over prominent metatarsal heads

touch, pin prick, temperature, 5.07 monofilament, absence of Achilles tendon reflexes; and a vibration perception threshold over 50 V). Peripheral pulses were palpable and the ankle brachial pressure index was 1.1 bilaterally.

The patient reported having a callus—probably due to high peak plantar pressures at the site of the callus—for the past 2 years, which she treated with pumice stone. Six months before her first visit, she noticed that the callus was harder and its base had become purple; when she decided to remove it using a blade, an ulcer developed, which she then treated with local antiseptics.

Debridement of the ulcer was carried out on a weekly basis.

Healthy granulating tissue was present at the base of this clean ulcer, together with mild callus formation at the border. The patient was advised to take prolonged bed rest and the ulcer healed completely in 6 weeks. Appropriate preventive footwear and orthotic insoles were prescribed in order to prevent the formation of a new ulcer.

This patient erroneously thought that pain in her feet was proof of a healthy peripheral nerve system. The combination of painful neuropathic symptoms and at the same time, complete absence of sensation

(a 'painful–painless foot') is a quite common feature of neuropathic diabetes.

Keywords: Neuropathic ulcer; granulating tissue

NEUROPATHIC ULCER OVER A COLLAPSED MIDFOOT

A typical neuropathic ulcer under a bony prominence in a patient with midfoot collapse due to neuro-osteoarthropathy is shown in [Figure 5.4](#). Callus formation is present at the margins of the ulcer, while



Figure 5.4 Neuropathic ulcer over a bony prominence in a patient with neuro-osteoarthropathy

its base is clean, covered by healthy granulating tissue.

Therapeutic footwear was prescribed (extra depth shoes with an orthotic insole and a window under the ulcerated area) and the patient was advised to minimize his activities. The ulcer healed in 3 months.

Ulcers in patients with midfoot collapse recur very often. Prevention of new ulcers over the same bony prominence is achieved by prophylactic surgery (osteotomy of the prominent bone). Preservation of plantar ligaments is essential, since their extensive resection may cause progression of the rocker bottom deformity.

Keywords: Neuropathic ulcer; bony prominence; prophylactic osteotomy

NEUROPATHIC ULCER UNDER FOURTH METATARSAL HEAD

A 74-year-old female patient with type 2 diabetes diagnosed at the age of 62 years, was referred to the outpatient diabetic foot clinic because of callus formation on her right sole. She was being treated with insulin and had a history of hypertension and ischemic heart disease.

On examination she was found to have severe peripheral neuropathy and normal peripheral pulses. In addition, significant muscle atrophy of her feet, claw toes and a hemorrhagic callus on the fourth metatarsal head of her right foot were found ([Figure 5.5](#)). An impressive finding was the palpation of her metatarsal heads just below the skin as the fat pads had been displaced anteriorly. After callus removal a superficial ulcer was revealed ([Figure 5.6](#)). An anteroposterior radiograph showed diffuse



Figure 5.5 Hemorrhagic callus under the fourth metatarsal head. Claw toes and prominent metatarsal heads are also present



Figure 5.6 A neuropathic ulcer in the same patient whose foot is shown in [Figure 5.5](#)

demineralization of the foot and significant widening with periosteal reaction at the metatarsal heads ([Figures 5.7 and 5.8](#)). The patient was advised to rest. Extra depth



Figure 5.7 Diffuse osteopenia and significant widening with periosteal reaction on the metatarsal heads can be seen in this X-ray of the foot shown in [Figure 5.5](#)

shoes and orthotic insoles were prescribed in order to accommodate her deformed toes and relieve the load under the metatarsal heads. Post-debridement in-shoe pressures when she used her own shoes showed a significant load under her metatarsal heads ([Figure 5.9 Panel A](#)). The maximum pressure in this area was 282 kPa; however, after insertion of an orthotic insole the maximum in-shoe pressure was reduced to 155 kPa ([Figure 5.9 Panel B](#)). The ulcer healed in 8 weeks.

Reduced thickness of the fat pad is associated with high plantar pressures. Although some authors have suggested that threshold pressures of 500–1000 kPa may lead to the development of foot ulceration when walking barefoot, it seems that each patient has an individual threshold. In the present case the maximum pressure was obviously below this threshold. However, high plantar



Figure 5.8 Significant widening with periosteal reaction of the first three metatarsal heads (same patient whose foot is shown in [Figures 5.5–5.7](#))

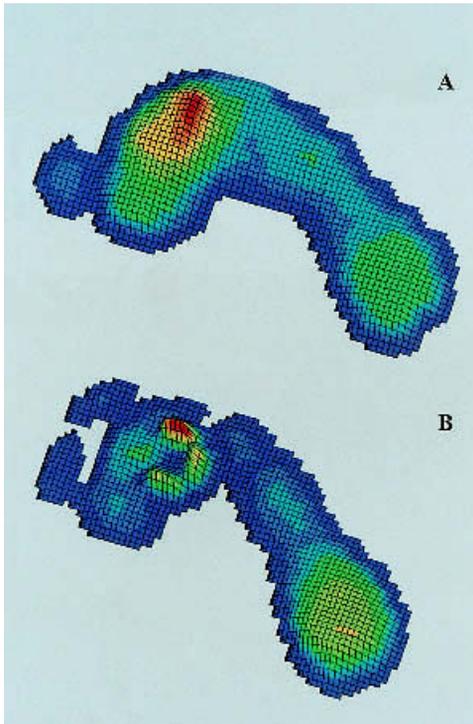


Figure 5.9 Plantar pressures before (A) and after (B) orthotic insoles in the patient whose foot is shown in [Figures 5.5–5.7](#)

pressures alone do not cause foot ulceration; a combination of different risk factors (mentioned in Chapter 1) is necessary for the development of ulceration.

Demineralization of the foot bones is not common, but when this occurs it signifies an adequate circulation, which is a prerequisite for bone resorption. Localized, mature periosteal reaction and demineralization involving metatarsal heads is common in diabetic patients with neuropathy. Its etiology is poorly understood. Focal osteolysis of phalanges, metatarsal heads, and other single foot bones, as well as stress fractures of the metatarsal heads can also be seen in neuropathic patients. Bone resorption at the phalanges may be so extensive that a part or even a whole phalanx may be resorbed. Metatarsal resorption usually starts from the metaphysis and extends to the epiphysis sparing the diaphysis. Bones which have become demineralized may have a pencil-like appearance.

Keywords: Neuropathic ulcer; plantar pressures, periosteal reaction

NEUROPATHIC ULCERS UNDER PROMINENT METATARSAL HEADS

This 32-year-old type 1 female diabetic patient, diagnosed at the age of 16 years, attended the outpatient diabetic foot clinic for chronic neuropathic ulcers of her feet. She was treated with intensive insulin treatment. The patient had a renal transplant at the age of 30 years, because of end-stage renal failure due to diabetes, and she had laser treatment on both eyes at the age of 28 years. Soon after her transplantation she noticed a bulla under her last three left metatarsal heads which readily ruptured and a superficial ulcer developed. She also reported an ulcer of 2 years' duration under the third metatarsal head of her right foot. She had never been instructed in foot care and had never worn



Figure 5.10 Neuropathic ulcers under prominent metatarsal heads and on the midsole. Claw toes and dry skin are also apparent

the correct footwear. She had two small children and had not been taking good care of her feet. The patient was being treated with erythropoietin injections, cyclosporin, methylprednisolone, mycophenolate mofetil and furosemide.

On examination she was found to have bounding pedal pulses, and severe diabetic neuropathy. The vibration perception threshold was above 50 V in both feet bilaterally.

A non-infected neuropathic ulcer was noted under her left third, fourth and fifth metatarsal heads. Its dimensions were $3.5 \times 4 \times 0.4$ cm, and it was surrounded by callus. A smaller neuropathic ulcer was also observed under her midsole (Figure 5.10). Claw toe deformity of her lesser toes, dry skin and desquamation of the tip of her third toe were also present. Under her



Figure 5.11 Neuropathic ulcer surrounded by callus. Claw toe. Right foot of patient whose left foot is shown in Figure 5.10

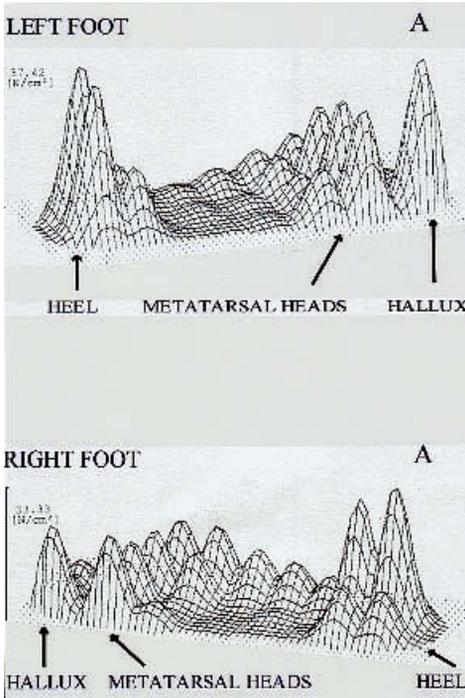


Figure 5.12 Original in-shoe peak plantar pressures on the left (upper panel) and right foot (lower panel) of the patient whose feet are illustrated in [Figures 5.10](#) and [5.11](#)



Figure 5.13 Healing neuropathic ulcers in the patient whose feet are shown in [Figures 5.10–5.11](#). Note bunionette deformity at the right foot

right third metatarsal head a neuropathic ulcer was noted in an area of gross callus formation, in addition to claw toe deformity ([Figure 5.11](#)). A callus was present under her right fifth metatarsal head over a bunionette deformity. Mild callus formation was observed on the heels of both feet. Onychomycosis affecting all toes was also present (discussed in Chapter 8, see [Figure 8.7](#)).

A plain radiograph did not reveal osteomyelitis. Sharp debridement was performed and therapeutic half shoes were prescribed. In-shoe peak pressure measurement showed high pressures under both heels, metatarsal heads, and halluxes when the patient wore

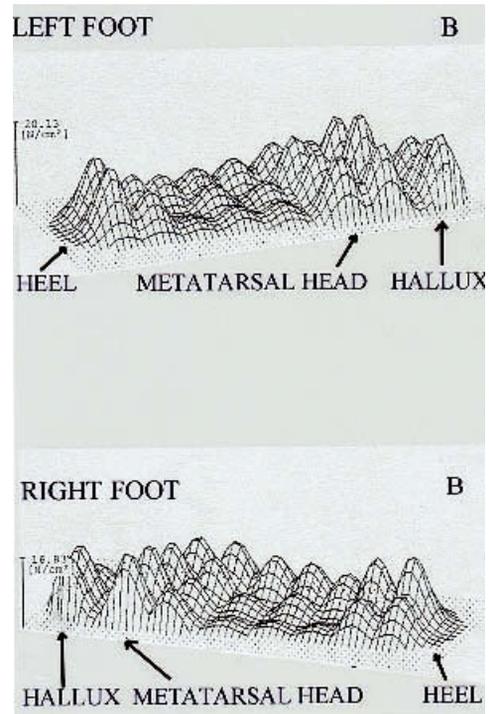


Figure 5.14 Effect of orthotic insoles and correct footwear on in-shoe peak plantar pressures on the left (upper panel) and right foot (lower panel) in the patient whose feet are illustrated in [Figures 5.10–5.11](#)

her own shoes (Figure 5.12). She had standard treatment on a weekly basis and the ulcers began to heal slowly. Six months after her first visit, an ulcer developed under her left third metatarsal head and a callus under her right fifth metatarsal head (Figure 5.13). New shoes were prescribed with orthotic insoles: the in-shoe peak pressures were reduced from 33.3 to 16.83 N/cm² under her right, and from 37.42 to 20.13 N/cm² under her left foot (Figure 5.14).

The patient continued visiting the outpatient foot clinic almost every week, and 6 months after her first visit her ulcers had healed.

Keywords: Neuropathic ulcer; peak plantar pressures

ULCERS OVER A CHARCOT FOOT

The following two figures (before and after debridement) show the left foot of a male patient of 62 years of age with type 2 diabetes diagnosed at the age of 48 years and treated with insulin. A smoker since the age of 18 years, the patient had had an ulcer on the plantar aspect of his left hallux which was complicated by osteomyelitis and led to amputation 3 years previously. One year before his first visit to the foot clinic the patient developed an ulcer on the lateral aspect of his left foot which resulted in osteomyelitis and surgical debridement of the metatarsal bone. After a femoral-popliteal bypass graft in his left foot, the patient developed neuro-osteoarthropathy. He presented to the outpatient clinic with two painless ulcers under his first and third metatarsal heads surrounded by hemorrhagic calluses.

Hyperkeratosis under his fifth metatarsal head and a scar at the site of the surgical debridement were noted (Figure 5.15).

The graft was functioning well and the patient had no claudication. Debridement of the ulcer under his fourth metatarsal exposed the bone (Figure 5.16). Cultures were obtained from the sloughy base of the ulcer—a positive sign of infection—and the patient was treated with an empirical combination of cotrimoxazole and clindamycin. The patient did not attend follow-up, therefore no X-ray or any further studies are available.

Charcot foot typically does not develop in patients with peripheral vascular disease since increased blood supply to the bone is needed for the osseous tissue to be overmetabolized. Autonomic sympathetic neuropathy leads to bone arteriovenous



Figure 5.15 Hallux disarticulation at the metatarsophalangeal joint, callus under first and fifth metatarsal heads, and deep infected neuropathic ulcer under the third metatarsal head. Claw toes



Figure 5.16 Foot shown in [Figure 5.15](#) after sharp debridement. Note bone exposure at the base of the ulcer under the third metatarsal head

shunting, hypervascularity and demineralization. Some cases are reported to occur after bypass surgery of the arteries.

Exposure of the bone denotes osteomyelitis and it should be treated accordingly.

Keywords: Neuropathic ulcers; Charcot foot; osteomyelitis; amputation

A NEUROPATHIC ULCER UNDER THE HEEL

A 51-year-old female patient with type 2 diabetes since the age of 38 years and

treated with insulin, was referred to the outpatient diabetic foot clinic because of a chronic non-healing ulcer under her right heel. She had good diabetes control (HBA_{1c}: 7.2%). Four months before her first visit she noticed a painless blister on the right heel caused by a small stone in her shoe; the blister ruptured and since the patient did not feel any pain she did not give her foot any attention. Some discharge was present on her socks, but it was the patient's daughter who saw a superficial ulcer on the right heel. The patient visited a primary care clinic and was advised to clean the ulcer with povidone iodide and apply clean dressings every day. A 2-week course of

antibiotics was prescribed. She continued her daily activities and after 4 months the ulcer was still active.

On examination the patient was found to have severe diabetic neuropathy with loss of sensation of pain, temperature, light touch and vibration. The vibration perception threshold was 36 V on both feet. Peripheral pulses were normal and the ankle brachial index was 1.2 and 1.1 in the right and left foot respectively. A full thickness ulcer with a sloughy base was noted on the right heel (Figure 5.17). No other signs of infection were present. An X-ray did not show involvement of the calcaneus. Cultures from the base of the ulcer revealed *Staphylococcus aureus*. She was treated with amoxicillin–clavulanic acid for 2 weeks and the ulcer was debrided on a weekly basis; dressings were changed daily. Meanwhile she was advised to rest and heel-free shoes to offload pressure

from the ulcerated area were prescribed (Figure 5.18). After 6 months the ulcer had healed completely (Figure 5.19).

Bedridden patients develop heel ulcers or gangrene quite frequently (20–30%),



Figure 5.18 Commercially available heel-free shoes for the treatment of hindfoot ulcers



Figure 5.17 Deep heel neuropathic ulcer with infected sloughy bed caused by trauma



Figure 5.19 Hindfoot shown in Figure 5.17 after the ulcer has completely healed



Figure 5.20 Neuropathic heel ulcer caused by shoe seam

usually on the posterolateral aspect. Excessive walking in new shoes can cause blister formation on the posterior aspect of the heel in patients with neuropathy. Shoe seams may also cause ulcers on the heel (Figure 5.20). Therefore shoes and socks without seams are prescribed to patients with loss of protective sensation. Heel ulceration is difficult in management since debridement in this area precludes functional weight bearing. Major amputations are often necessary when heel ulcers are infected.

Keywords: Neuropathic ulcer; heel

BURNS ON TOES AND FOREFOOT

A 55-year-old male patient with type 2 diabetes since the age of 43 years attended the outpatient diabetic foot clinic due to ulcers on his feet. His diabetes was poorly controlled with sulfonylureas and he had a history of a disarticulated left great toe

at the metatarsophalangeal joint due to osteomyelitis.

On examination the patient was febrile; peripheral pulses were palpable, the ankle brachial index was 1.2; the vibration perception threshold was over 50 V in both feet; temperature, light touch and pinprick sensation were absent as were the Achilles tendon reflexes. Blood pressure was normal; no other diabetic complications were found. HbA_{1c} was 11.0%. There was a perforating dirty ulcer on the outer aspect of his right foot. A large amount of callus had built up around the plantar orifice (Figures 5.21 and 5.22). The patient reported edema of the forefoot which had recently subsided as was evident from the scaling of the skin. Callus formation was also observed over the second, third and fifth metatarsal heads of the left foot. The patient was empirically treated with ciprofloxacin.

Debridement of the callus was carried out. Cultures revealed *Staphylococcus aureus* and *Escherichia coli*. Osteomyelitis of the fifth metatarsal head was evident on a plain radiograph (Figure 5.23). The patient



Figure 5.21 Perforating, infected neuropathic ulcer under the fifth metatarsal head. Scaling is due to edema that has subsided



Figure 5.22 Right foot: neuropathic ulcer shown in [Figure 5.21](#). Left foot: hallux disarticulation, medial displacement of second toe with claw deformity; callus formation under second, third and fifth metatarsal heads



Figure 5.23 Plain radiograph of the right foot of the patient whose foot is shown in [Figure 5.21](#). Osteomyelitis of the fifth metatarsal head and the proximal phalanx of the fifth toe, subluxation of the metatarsophalangeal joint, calcification of the digital artery between the first two metatarsals and osteoarthritis of the first distal phalangophalangeal joint of the hallux are all apparent

continued ciprofloxacin treatment; cotrimoxazole was added for almost 6 months and the ulcer gradually healed ([Figure 5.24](#)) with the help of therapeutic shoes.

Instruction in appropriate foot care was provided. The patient visited the outpatient clinic erratically; callus formation on the site of the healed ulcer was removed every 3 months; he refused strict glycemic control as he was afraid that episodes of hypoglycemia would jeopardize his position at work. He used intermediate-acting insulin at bedtime and sulfonylureas during the day. His HbA_{1c} remained at 9.0% during the following year. Preventive footwear was not accepted.

The patient attended the clinic 2 years later because of multiple burns over the

tips of his toes and superficial ulcers over the fifth metatarsal heads of both feet ([Figure 5.25](#)). He had put his feet in front of the fire in order to dry out his wet socks. No pain was felt. Although the patient was aware of the burns he continued his activities for a week before this visit.

Full thickness burns were present over the tips of all toes. Blisters over the right fifth metatarsal head and the left fourth and fifth toes were removed and ulcers had developed since the patient was still working regularly, despite medical advice to the contrary ([Figure 5.26](#)). Calluses formed around the new plantar ulcers. Amoxicillin–clavulanic acid treatment was initiated and the patient attended the diabetic foot clinic on a weekly basis.



Figure 5.24 The ulcer shown in [Figure 5.21](#) after it has almost completely healed



Figure 5.25 Thermal injury sustained by the patient whose feet are illustrated in [Figure 5.22](#)



Figure 5.26 Neuropathic ulcers under the fifth metatarsal heads and progression of thermal injury in the patient whose feet are shown in Figures 5.21–5.25. The patient did not comply with doctors’ instructions

All ulcers healed within 2 months except the one on the right great toe, which was complicated by osteomyelitis and acute soft

tissue infection. Five months after the burn his right hallux had to be disarticulated.

The patient still refused preventive shoes and 4 months after this second amputation new ulcers developed under the fifth metatarsal heads bilaterally (Figure 5.27).

Keywords: Thermal injury; osteomyelitis

CHRONIC NEUROPATHIC ULCER COMPLICATED BY OSTEOMYELITIS

A 55-year-old male patient with type 2 diabetes diagnosed at the age of 50 years was referred to the outpatient diabetic foot clinic because of a chronic neuropathic ulcer on his right foot. He had a history of hypertension, background retinopathy in both eyes and diabetic nephropathy (urine protein 1.5 g/24 h). He had been treated with sulfonylurea but had discontinued



Figure 5.27 Right hallux disarticulation at the metatarsophalangeal joint and recurrence of ulcers under the fifth metatarsal heads (patient whose feet are shown in Figures 5.21–5.26)

the treatment 1 year before his first visit, when overt nephropathy developed. He had excellent diabetes control (HBA_{1c} : 6.4%).

On examination his feet pulses were bounding (ankle pressure index 1.2 bilaterally); he had severe peripheral neuropathy: no sensation of pain, light touch, vibration or temperature; the vibration perception threshold was 48 V on the left and above 50 V on the right foot. A full thickness clear neuropathic ulcer surrounded by callus was observed under the right first metatarsal head, with dimensions of $3 \times 3 \times 0.5$ cm (Figure 5.28). Mild claw deformities of the toes and displacement of the metatarsal fat pads to the base of the proximal phalanges due to muscle atrophy were also noted.

Sharp debridement was carried out and special extra depth shoes with an orthotic insole were prescribed. Care was taken to offload pressure from the ulcerated area.



Figure 5.28 Neuropathic ulcer under prominent first metatarsal head. Healthy granulating tissue can be seen at the base of the ulcer

The patient was advised to limit his daily activities and he attended the diabetes foot clinic on a weekly basis. The size of the ulcer was reduced by half within 4 weeks. Two weeks later, after a professional trip, the patient visited the clinic again. His ulcer was infected and a large amount of callus had formed around it. His right hallux had a ‘sausage-like’ appearance and signs of infection were observed (redness and edema). A culture from the base of the ulcer revealed the presence of *Staphylococcus aureus* and *Enterobacter cloacae* post-debridement. A radiograph at that time showed mild erosion of the first metatarsal head.

The patient was given treatment with cotrimoxazole and clindamycin. The radiograph was repeated 2 weeks later and extensive erosion of the first metatarsal head was revealed (Figure 5.29). Acute osteomyelitis was diagnosed. The patient continued with the antibiotics for 12 weeks and had regular chiropody treatment on a weekly basis. The ulcer healed completely in 20 weeks (Figure 5.30).

Treatment of acute osteomyelitis should be based on bone cultures when possible, and should be continued for 6–12 weeks. The commonest pathogen of



Figure 5.29 Erosion of first metatarsal head with periosteal reaction due to osteomyelitis (patient whose ulcer is shown in Figure 5.28)

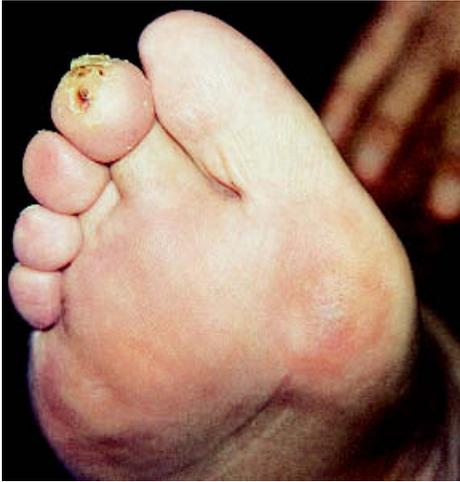


Figure 5.30 Healed ulcer of the patient whose feet are shown in **Figures 5.28–5.29**. Note the scar over the ulcerated area and callus formation at the tip of the second toe due to claw deformity

acute osteomyelitis in patients with foot ulcers is *Staphylococcus aureus* (60–90%). Other pathogens include *Staphylococcus epidermidis*, *Escherichia coli*, *Pseudomonas aeruginosa*, and other *Enterobacter* spp. More than one pathogen is often isolated. In order to achieve therapeutic levels of antibiotics in the bone it is preferable to administer antibiotics intravenously

for the first 2 weeks. However, oral antibiotics with good bioavailability (fluoroquinolones, clindamycin) may be adequate for therapy. Treatment regimens for staphylococcal osteomyelitis are as follows:

- Clindamycin 600 mg × 3 orally or 600 mg × 3 i.v.
- Fucidic acid 500 mg × 3 orally or 500 mg × 3 in a 500-ml solution delivered slowly i.v. (over 4–6 h)
- Cotrimoxazole 960 mg × 2 orally or i.v.
- Ciprofloxacin 750 mg × 2 orally, or 400 mg × 3 i.v.
- Rifampicin 900 mg × 1 orally or i.v.
- Teicoplanin 600 mg × 1 orally or i.m. or i.v.
- Vancomycin 500 mg × 4 i.v. or 1 g × 2 i.v.

Fluoroquinolones, teicoplanin and vancomycin should be prescribed for methicillin-resistant staphylococcus only. Fluoroquinolones in particular, should always be combined with another anti-staphylococcal drug in the first month of treatment, since it is likely that a resistant strain will prevail in the infection.

Keywords: Acute osteomyelitis; treatment

Chapter VI

NEURO-ISCHEMIC ULCERS AT VARIOUS SITES

- UNDER HALLUX
- NEURO-ISCHEMIC ULCER WITH OSTEOMYELITIS UNDER THE HALLUX
- ON THE DORSUM OF THE FOOT
- INTERDIGITAL
- AT MEDIAL SIDE OF THE FOOT
- ON FIRST METATARSAL
- ON MIDSOLE AND HEEL
- ON FOREFOOT
- NEURO-ISCHEMIC ULCER ON THE HALLUX WITH OSTEOMYELITIS
- ON THE DORSUM OF CLAW TOES
- OVER THE FIFTH METATARSAL HEAD

NEURO-ISCHEMIC ULCER UNDER HALLUX

A 68-year-old obese male patient with type 2 diabetes diagnosed at the age of 46 years visited the outpatient diabetic foot clinic because of two chronic ulcers on his right hallux. He was treated with a combination of sulfonylurea during the day and a mixture of 20% rapid acting–80% intermediate acting insulin before dinner; he also had dislipidemia which was being treated with simvastatin.

On examination, he had severe diabetic neuropathy (no sensation of light touch, pin prick, temperature, 5.07 monofilament, absence of Achilles tendon reflexes and a vibration perception threshold over 50 V).



Figure 6.1 Infected full thickness neuro-ischemic ulcers under the right hallux with edema and superficial ulceration at the tip and a subungual hematoma caused by inappropriate footwear

Peripheral pulses were palpable, but the ankle brachial index was 0.7 bilaterally.

Two painful full thickness neuro-ischemic ulcers with sloughy bed were seen under his right hallux; edema and superficial ulceration at the tip of his hallux, and subungual hematomata of the first two toes were also present (Figure 6.1). There was no X-ray evidence of osteomyelitis. Sharp debridement was carried out and he was treated empirically with amoxicillin–clavulanic acid. No pathogen was isolated on swab cultures, probably due to the use of local antiseptics. Offloading of pressure was successful with the help of appropriate therapeutic footwear (see Figure 3.36) and the ulcer began to heal smoothly (Figure 6.2); 3 months after the initial visit it had healed completely (Figure 6.3).

Keywords: Neuro-ischemic ulcer

NEURO-ISCHEMIC ULCER WITH OSTEOMYELITIS UNDER THE HALLUX

A 67-year-old man who had type 2 diabetes since the age of 44 years and was being treated with insulin, visited the diabetic foot clinic because of an ulcer on his left hallux. He had acceptable diabetes control. He had proliferative retinopathy which had been treated with laser, and intermittent claudication at 400 m. He had smoked for 27 years.

Twenty days before his visit, he had worn a new pair of shoes and driven his car for a long distance. The following day a blister developed on his left great toe. Within a day the area became edematous and black.

On examination the patient was found to have a deep, foul-smelling ulcer with



Figure 6.2 Healing of the ulcers shown in [Figure 6.1](#)



Figure 6.3 Hallux ulcers shown in [Figures 6.1](#) and [6.2](#) after they have fully healed

gangrenous areas and a purulent discharge over the medial and dorsal aspect of his left great toe ([Figure 6.4](#)).

Peripheral pulses were weak and the ankle brachial index was 0.8. The patient could not feel pain, temperature, light touch, or a 5.07 monofilament. The vibration perception threshold was 40 and 45 V at the tips of the left and right great toe respectively.

Sharp debridement was carried out revealing the underlying bone. An X-ray showed osteomyelitis of the distal phalanx ([Figure 6.5](#)). The patient was treated empirically with ciprofloxacin and clindamycin as a swab culture from the base of the ulcer had revealed *Staphylococcus aureus*. Despite local foot care and the systemic antibiotics, the ulcer was still active and osteomyelitis spread locally affecting



Figure 6.4 Deep tissue infected neuro-ischemic ulcer with necrotic areas on the left hallux



Figure 6.5 Plain radiograph of the foot illustrated in [Figure 6.4](#), showing osteomyelitis involving proximal interphalangeal joint and adjacent phalanges of the left hallux

the proximal phalanx. Three months after his first visit the patient's left great toe was amputated ([Figure 6.6](#)). Antimicrobial treatment was continued for 2 weeks after the amputation.

The consequences of hallux disarticulation at the metatarsophalangeal joints have been discussed previously (see [Figure 3.32](#)).

Keywords: Neuro-ischemic ulcer; osteomyelitis; amputation

NEURO-ISCHEMIC ULCER ON THE DORSUM OF THE FOOT

A large (3.5 × 2.0 cm) painless neuro-ischemic ulcer developed on the right foot of a 68-year-old male patient with type 2 diabetes which had been diagnosed at the age of 61 years. Peripheral pulses were



Figure 6.6 Disarticulation at the metatarsophalangeal joint of left hallux of the patient whose foot is shown in [Figures 6.4](#) and [6.5](#)

weak and the ankle brachial index was 0.6. At the base of the ulcer the fascia of the dorsum of the forefoot was exposed ([Figure 6.7](#)). There were no signs of infection. A subungual hematoma of the hallux and an ulcer which was healing on the second toe were noted in addition to significant ankle edema. The ulcer was a result of friction between the foot and the forepart of the patient's narrow shoe upper (vamp) following the rupture of a large blister.

Therapeutic footwear was prescribed ([Figure 6.8](#)) and the ulcer healed within 2 months.

Appropriate footwear was prescribed subsequently in an attempt to avoid recurrence of the ulcer (preventive footwear). Such footwear is made of soft, self-moldable material without any seams, and has extra depth in order to accommodate an



Figure 6.7 A large neuro-ischemic ulcer with exposed fascia on the dorsum of the forefoot. There is a subungual hematoma of the hallux and a healing ulcer on the second toe in addition to significant ankle edema



Figure 6.8 Therapeutic footwear for ulcers on the dorsum of the forefoot

appropriate insole and the forefoot deformities (Figure 6.9). Skin injuries due to shoe friction are thus avoided; this is essential for patients whose skin is thin and fragile due to arterial disease.

Keywords: Foot; dorsum; neuro-ischemic ulcer

INTERDIGITAL NEURO-ISCHEMIC ULCER

A neuro-ischemic ulcer on the lateral aspect

of the fourth toe (Figure 6.10) was caused by pressure from the patient's little toe. Shoes with narrow toe boxes are often the cause of such ulcers. Mild callus formation—due to pressure of the adjacent toes—was seen around the ulcer. The patient suffered mild discomfort. A silicone-ring (Figure 6.11) was used to keep the third and fourth toes apart until the ulcer healed. The patient was instructed in foot care and the correct footwear was also prescribed.

Keywords: Interdigital neuro-ischemic ulcer

Figure 6.9 Preventive footwear and shock-absorbing insole for patients at risk for ulceration. The shoe upper is made of soft self-moldable material without seams. A high toe box facilitates insertion of the insole



Figure 6.10 Interdigital neuro-ischemic ulceration caused by tight shoes



Figure 6.11 Silicone ring used to keep adjacent toes apart

**NEURO-ISCHEMIC ULCERS
ON THE MEDIAL SIDE
OF THE FOOT**

A 51-year-old obese male patient who had type 2 diabetes since the age of 34 years and was currently being treated with glimepiride, visited the outpatient diabetic foot clinic. During the past 12 months his diabetes control varied (HbA_{1c}: 7.5–9.0%). The patient had hypertension which was being treated with quinapril and furosemide; he was a smoker until the age of 49 years and was currently being treated with inhalations of ipratropium bromide and oral theophylline for the management of chronic obstructive pulmonary disease. Painful leg and foot neuropathy was treated with carbamazepine, with fair results. A vascular surgeon had

prescribed low dose aspirin and buflomedil. Background retinopathy (hemorrhages and soft exudates) and nephropathy were also diagnosed.

Moderate pes planus was noted (Figure 6.12). Reflexes of both knees and Achilles tendons were absent, and there was decreased deep and superficial sensation (light touch, cold and warm sensation, monofilament, pin-prick sensation and vibration perception threshold). The patient also suffered from venous insufficiency, mild ankle edema and skin atrophy. Hematocrit, 35.5%; creatinine, 1.3 mg/dl (114.9 μmol/L); urine protein, 1100 mg/24 h; normal plasma lipid profile; BMI, 32 kg/m². A 4-cm aneurysm of the abdominal aorta was found by ultrasound scan. The ankle brachial index was 1.4 due to calcification of the posterior tibial and the dorsal pedal arteries. A triplex of the leg arteries



Figure 6.12 Neuro-ischemic ulcers on the hindfoot

revealed significant diffuse stenoses mainly of the arteries in the left leg.

The patient had two painful superficial ulcers on the medial aspect of his right foot due to trauma from his footwear, which he first noticed 3 months earlier. He used topical povidone iodide with no improvement.

The ulcers were clean without signs of infection. A mild callus had formed as a result of shoe friction. At the clinic the ulcers were debrided on a weekly basis and dressed with standard gauze with 15% saline. They healed completely in 1 month. Povidone iodide was discontinued as it impairs wound healing. Instruction in appropriate foot care and foot hygiene was provided, and suitable footwear was prescribed.

Neuro-ischemic ulcers comprise almost 40% of all diabetic foot ulcers. Ischemic ulcers develop at sites which are not stressed by high pressure, such as the lateral, medial or dorsal aspect of the foot and are usually painful. Intervention with

vascular surgery (bypass grafting or percutaneous transluminal angioplasty) is usually needed in order to restore the blood supply to the periphery.

Keywords: Peripheral vascular disease; neuro-ischemic foot ulcers; pes planus

NEURO-ISCHEMIC ULCER ON THE FIRST METATARSAL WITH OSTEOMYELITIS

An ostensibly small, painless neuro-ischemic ulcer on the medial-plantar area of the first metatarsal head with callus formation and purulent discharge was the reason for this patient's visit (Figure 6.13). Claw deformity of lesser toes was present. After debridement, a $1.5 \times 1.0 \times 1.0$ cm ulcer was revealed. A plain radiograph showed osteomyelitis of the first metatarsal head. *Staphylococcus aureus* was isolated from



Figure 6.13 An ostensibly small neuro-ischemic ulcer complicated by osteomyelitis. Claw deformity of lesser toes is also apparent

the discharge and the patient was treated with clindamycin for 6 months, with a good outcome.

Keywords: Neuro-ischemic ulcer; osteomyelitis

**NEURO-ISCHEMIC ULCERS
ON THE MIDSOLE
AND HEEL**

After surgical debridement this diabetic patient suffered from two painful neuro-ischemic ulcers on the right midsole and the medial aspect of the heel (Figures 6.14 and 6.15). Cellulitis around the plantar ulcer was observed. Pedal pulses were weak and the ankle brachial index was 0.7. The ulcers resulted from ruptured blisters which had developed after prolonged walking in new shoes. Initially the ulcers were painless due to peripheral neuropathy,

and the patient continued his activities. An angiogram showed mild atheromatous disease at the iliac and common femoral artery, severe stenosis in the middle of the right superficial femoral artery and a lesser degree of stenosis in the popliteal arteries. Balloon angioplasty of the right superficial femoral artery was carried out and an intravascular stent was inserted. Use of a wheelchair to offload pressure, adequate use of various antibiotics and a revascularization procedure resulted in complete healing of the ulcers.

Keywords: Neuro-ischemic ulcers

**NEURO-ISCHEMIC ULCER
ON THE FOREFOOT WITH
OSTEOMYELITIS**

A 64-year-old male patient with type 2 diabetes that had been diagnosed at the age



Figure 6.14 The deep neuro-ischemic ulcer with surrounding cellulitis on the right sole resulted from ruptured blisters which developed after prolonged walking in new shoes



Figure 6.15 Heel ulcer in the patient whose foot is shown in [Figure 6.14](#). The yellowish appearance of the bed of the ulcer is indicative of ischemia

of 55 years, was referred to the outpatient diabetic foot clinic because of an infected chronic ulcer on his left foot. The patient had a history of heart failure, ischemic heart disease and stage II peripheral vascular disease (intermittent claudication) according to the Fontaine classification (see Chapter 1). He also reported burning pain and numbness in his feet which worsened during the night. Three months earlier, after a long walk, the patient noticed the appearance of a small ulcer under his left first metatarsal head. He did not ask for medical help at that time since he felt no pain. A yellowish discharge was present on his socks and the insole of the left shoe.

On examination, an infected, foul-smelling ulcer was observed under his second metatarsal head, extending into the second web space (Figure 6.16). Another ulcer surrounded by callus was also noted under the first metatarsal head. Peripheral pulses were weak on both feet. He had findings of severe diabetic neuropathy. After debridement a purulent discharge emanated from the deeper tissues of the dorsum of the foot. A plain radiograph did not reveal osteomyelitis. A culture of the pus revealed *Staphylococcus aureus*. The patient was afebrile, but he was admitted to the hospital and treated with i.v. administration of amoxicillin–clavulanic acid. Two weeks after his admission osteomyelitis at the proximal phalanx of the second toe was diagnosed. The patient sustained a second toe disarticulation at the metatarsophalangeal joint. The wound healed well, and the infection subsided completely.

Several relapses of foot ulceration occurred in the following years. The patient attended the foot clinic erratically and did not wear appropriate footwear. Two years after his amputation a new neuro-ischemic ulcer developed on the midsole (Figure 6.17) caused by a worn-out insole.



Figure 6.16 An infected neuro-ischemic ulcer soaked in profound discharge, on the plantar area between the first and the second left metatarsal heads extending into the second web space. A second ulcer surrounded by callus is also seen under the first metatarsal head

A new neuro-ischemic ulcer under his first metatarsal head was also present. There was callus formation below his disarticulated second toe.

Refusal to wear suitable footwear is a major problem in patients at risk for foot ulcers. Although there is evidence to suggest that the correct footwear reduces the incidence of foot ulcers, and many health-care systems cover 70–100% of the cost of preventive footwear (shoes and insoles), only 20% of patients wear appropriate footwear on a regular basis. Effective education may increase this rate. In addition, the recurrence of ulcers after initial healing is also common. A recurrent ulcer is



Figure 6.17 The same patient whose foot is illustrated in [Figure 6.16](#), two years after second toe disarticulation. A neuro-ischemic ulcer caused by a worn-out insole is seen on mid-sole. A recurrent neuro-ischemic ulcer is present under the first metatarsal head. A callus has formed below the disarticulated second toe

defined as any tissue breakdown at the same site as the initial ulcer occurring during the 30 days following the initial healing. Any new ulcer that occurs at the same site within 30 days of healing is considered to be part of the original episode. An ulcer at a different site is considered to be a new episode independent of the time of its development. New ulcers develop at the same or different sites in a foot with prior foot ulceration in about 50% over 2–5 years. Thus the healing of an ulcer is just the first step in the management of the patient at risk. Appropriate education, prescription of the correct

footwear and reduction—if possible—of the risk factors for foot ulceration (correction of foot deformities, regular callus removal, improvement in vascular supply to the feet), may reduce the risk for recurrence of foot problems in patients with diabetes.

Keywords: Neuro-ischemic ulcer; recurrent ulcers; compliance with suitable footwear

NEURO-ISCHEMIC ULCER ON THE HALLUX WITH OSTEOMYELITIS

A 76-year-old female patient with type 2 diabetes diagnosed at the age of 62 years, attended the outpatient diabetic foot clinic for a chronic ulcer on the right hallux. She had a history of ischemic heart disease and peripheral vascular disease.

On examination she had findings of peripheral neuropathy. Pedal pulses were weak on both feet. The patient had a painful neuro-ischemic ulcer with dimensions $1.0 \times 1.0 \times 0.4$ cm and a sloughy base on the medial aspect of the right hallux caused by a tight shoe ([Figure 6.18](#)). A plain radiograph revealed osteomyelitis involving the condyle of the proximal phalanx of the hallux ([Figure 6.19](#)). The ankle brachial index was 0.6. Duplex ultrasonography of the arteries of the legs revealed multilevel bilateral atherosclerotic disease in her superficial femoral arteries and severe stenosis in the arteries of her left tibia. The pedal arteries were not involved. The patient underwent a femoropopliteal and a popliteal-peripheral bypass. Since sharp debridement of the ulcer was too painful, a dextranomer was applied for mechanical debridement on a daily basis. A swab culture and a culture of



Figure 6.18 Neuro-ischemic ulcer with a sloughy base on the medial aspect of the right hallux



Figure 6.19 Osteomyelitis of the condyle in the proximal phalanx of the hallux of the foot shown in [Figure 6.18](#)

the sequestrum seen in a plain radiograph revealed *Pseudomonas aeruginosa* and the patient was treated with ciprofloxacin. With local wound care and antibiotic treatment the ulcer healed completely in 12 weeks (Figure 6.20). She continued with antibiotic treatment for a total of 6 months.

Inadequate blood supply prevents healing of foot ulcers especially when they are complicated by osteomyelitis.

Debridement of an ulcer is the cornerstone of the management of active, acute or chronic wounds. The aim of debridement is to remove fibrin (white, yellow or green tissue seen on the bed of an ulcer) and necrotic tissue (black tissue) and to produce a clean, well vascularized wound bed. Types of debridement are as follows:

- Sharp surgical (using scalpels), the gold standard for wound preparation, removes both necrotic tissue and microorganisms
- Mechanical (using wet-to-dry dressings, hydrotherapy, wound irrigation and dextranomers)
- Enzymatic (using chemical enzymes such as collagenase, papain or trypsin in a cream or ointment base)
- Autolytic debridement (using *in vivo* enzymes which self-digest devitalized tissue such as hydrocolloids, hydrogels, and transparent films)

Callus formation at the borders of neuropathic ulcers should be removed. The majority of patients with severe diabetic neuropathy feel no pain, therefore extensive sharp debridement or even operations on the feet can be performed without anesthesia.

The use of enzymatic debridement is increasing. Chronic wounds are enzymatically debrided in elderly patients when regular, sharp debridement is not possible, e.g. if the necrotic zone is thin; in ulcers with sinuses; and as an additional procedure to sharp debridement. Combination of collagenase with hydrogels or alginates seems to have synergistic effects.

Autolytic debridement uses the body's own enzyme and moisture to re-hydrate, soften and finally liquefy hard eschar and slough. It is selective, as only the necrotic tissue is liquefied, and painless to the patient. Its main indication is non-infected ulcers with mild to moderate exudates. Autolytic debridement can be achieved with the use of occlusive or semi-occlusive dressings which maintain the wound fluid in contact with the necrotic tissue. (For a more detailed description of the different types of dressings and their indications see Chapter 2.)

The use of sterile maggots (biosurgery, larval therapy, maggot debridement



Figure 6.20 The final stages of ulcer healing in the foot shown in Figures 6.18 and 6.19. Note the chronic onychomycosis of the hallux with brown discoloration and thickening of the nail



Figure 6.21 Neuro-ischemic ulcers on the dorsum of claw toes



Figure 6.22 Commercially-available preventive footwear with high toe box and minimal seaming for forefoot deformities

therapy) is a practical and highly cost-effective alternative to conventional dressings or surgical intervention in the treatment of sloughy or necrotic wounds. It is also a valuable tool in cases where wounds have been infected with antibiotic-resistant pathogens.

All chronic wounds are contaminated with bacteria. Studies have shown that a burden of 1.0×10^6 colony-forming units per gram of tissue can cause significant tissue damage and impair healing. The use of cadexomer iodide decreases microbial load, and is particularly useful in the treatment of wounds colonized by methicillin-resistant *Staphylococcus aureus*, *Pseudomonas aeruginosa* or *Candida albicans*.

Other local antimicrobials are also effective against a wide range of common microorganisms.

Keywords: Neuro-ischemic ulcer; osteomyelitis; types of debridement

NEURO-ISCHEMIC ULCERS ON THE DORSUM OF CLAW TOES

Severe claw toe deformity, combined with peripheral diabetic neuropathy and vascular disease, predisposes to ulceration of the dorsum of the toes after repetitive trauma due to irritation of the thin skin by inappropriate shoes (Figure 6.21). The use of extra depth shoes such as those shown in Figure 6.22, in addition to basic foot care, should be sufficient to ensure ulcer healing and prevention of recurrence, provided the ulcers are not infected. Non-invasive vascular testing of this patient revealed multilevel stenosis of the arteries in both legs. The patient was referred to the vascular surgery department.

Keywords: Neuro-ischemic ulcers on the dorsum of toes; preventive footwear; claw toes

NEURO-ISCHEMIC ULCER WITH OSTEOMYELITIS OVER THE FIFTH METATARSAL HEAD

A 49-year-old male patient with a 4-year history of type 2 diabetes being treated with gliclazide, and an 8-year history of multiple sclerosis, was admitted because of mild fever and ulcers on his right foot. He had sustained an amputation of the last two phalanges of his right fifth toe 2 years before admission.



Figure 6.23 Neuro-ischemic ulcers on the right foot over the fifth and first metatarsal heads. The last two phalanges of the fifth toe have been amputated and there is a superficial ulcer on the dorsum of the second toe. Onychodystrophy is due to peripheral vascular disease

On examination he had a temperature of 37.9°C, a pulse rate of 82 pulses per minute and his blood pressure was 140/80 mmHg. An infected ulcer was present on the upper aspect of his foot over the base of his amputated toe, and a second one over the plantar aspect of the fifth metatarsal head (Figure 6.23). He had hypoesthesia in both feet, and absence of pulses in his right leg and foot. There were pulses in his left foot and both femoral arteries. Achilles tendon reflexes were reduced and he had a Babinski sign on the right foot. His white blood cell count was 12,200/mm³ with 74.7% neutrophils. His erythrocyte sedimentation rate was 38 mm/h. Blood glucose was 188 mg/dl (10.4 mmol/l) and his HbA_{1c} was 7.5%. Protein was present in a urine



Figure 6.24 X-ray of the foot shown in Figure 6.23. There is osteomyelitis in the fifth metatarsal head and the distal phalanges of the fifth toe have been amputated

sample. An X-ray revealed osteomyelitis of the head of the fifth metatarsal, right under the ulcerated area (Figure 6.24). The patient was treated empirically with clotrimoxazole and clindamycin. *Streptotomonas maltophilia* was isolated from a swab culture

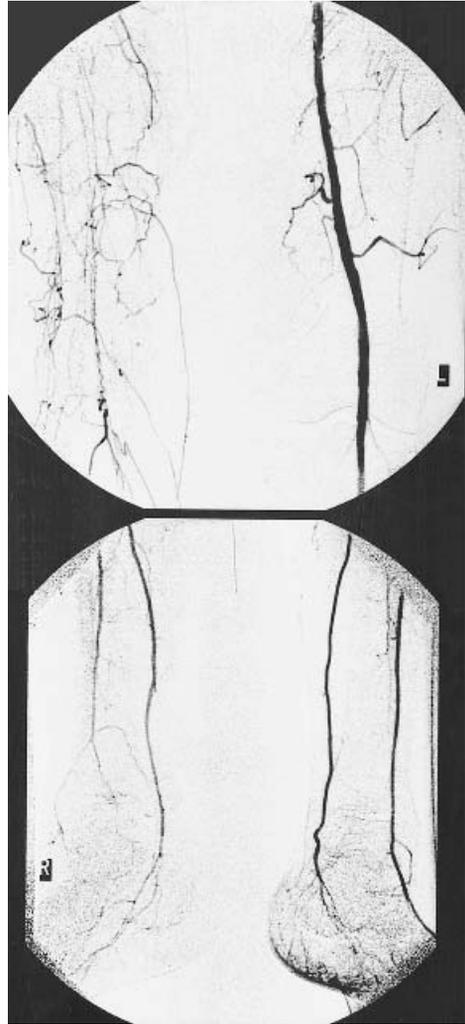


Figure 6.25 Arteriography of the patient whose foot is shown in Figure 6.23. There is severe obstruction of the distal part of the right femoral and popliteal arteries; the pedal arteries are patent and filled by collateral circulation

and netilmicin was added to the treatment regimen after the antibiogram.

An angiogram revealed severe obstruction of the lower right femoral and popliteal arteries (Figure 6.25). Vascular surgeons suggested a femoro-tibial bypass graft after his general condition had been stabilized for several months, or in the case of an

emergency, since no gangrene was present at the time. Pentoxifyllin and buflomedil were prescribed. The ulcer improved after 2 weeks of antibiotic treatment and local care.

Keywords: Neuro-ischemic ulcer; angiography; osteomyelitis

Chapter VII

GANGRENE

- DRY GANGRENE OF TOES
- DRY GANGRENE WITH ISCHEMIC NECROSIS OF THE SKIN
- DRY GANGRENE OF HEEL
- DRY GANGRENE OF ALL TOES
- WET GANGRENE AND SEPSIS
- DRY GANGRENE OF THE TOE
- STENT
- DIGITAL SUBTRACTION ANGIOGRAPHY
- WET GANGRENE OF THE TOES
- WET GANGRENE OF THE FOOT
- WET GANGRENE LEADING TO MID-TARSAL DISARTICULATION
- EXTENSIVE WET GANGRENE OF THE FOOT
- WET GANGRENE OF THE HALLUX

DRY GANGRENE OF THE TOES

A 65-year-old male patient with type 2 diabetes diagnosed at the age of 61 years and treated with sulfonylurea, was admitted to the Vascular Surgery Department. He was a heavy smoker and had a sedentary lifestyle. He had hypertension, background diabetic retinopathy and dyslipidemia (triglycerides: 4 mmol/l; HDL-cholesterol: 0.67 mmol/l). His diabetes control was poor (HBA_{1c}: 8.5%). The patient complained that in the previous 3 weeks he had experienced pain which required analgesia when he was at rest. He had typical symptoms of intermittent claudication for 2 years with progressive worsening.

On examination, extensive dry gangrene was found involving all the toes and with a necrotic area over the dorsum of his left foot (Figure 7.1). The foot arteries and left popliteal artery could not be felt, while the femoral arteries were just palpable bilaterally. Pulses in the right foot arteries were absent; the skin was cold and the right popliteal artery was just palpable. The ankle brachial index was 0.4. The patient had reduced sensation of pain, light touch and temperature. The vibration perception threshold was 35 V on the left and 30 V on the right foot. Critical limb ischemia with dry gangrene was diagnosed; an angiogram showed extensive stenosis of the common iliac, superficial femoral and popliteal arteries of both feet. Aorto-femoral and femoro-popliteal bypass grafts were undertaken 2 days after admission, followed by mid-tarsal disarticulation (at Lisfranc's joint). The postoperative period was without any complications and the wound healed completely.

Gangrene is characterized by the presence of cyanotic, anesthetic tissue associated with

or progressing to necrosis. It occurs when the arterial blood supply falls below minimal metabolic requirements. Gangrene can be described as dry or wet. Wet gangrene is dry gangrene complicated by infection (see below, and Figures 7.24 and 7.25).

Dry gangrene is characterized by its hard, dry texture, usually occurring in the distal aspects of the toes, often with a clear demarcation between viable and necrotic tissue. Once demarcation occurs, as is the case in this patient, the involved toes may be liable to auto-amputation. However, this is a long (several months) and unpleasant process. In addition, many patients do not have an adequate circulation to heal a distal amputation. For these reasons it is common practice to evaluate the arteries angiographically and to carry out a bypass or a percutaneous transluminal angioplasty with concomitant limited distal amputation, in order to improve the chances of wound healing.

CRITICAL LEG ISCHEMIA

Critical leg ischemia is defined — according to the consensus statement on critical limb ischemia — as either of the following two criteria: *persistently recurring ischemic rest pain*, requiring regular adequate analgesia for more than 2 weeks, with an ankle systolic pressure ≤ 50 mmHg and/or a toe pressure ≤ 30 mmHg; or *ulceration or gangrene* of the foot or toes, with an ankle systolic pressure ≤ 50 mmHg and/or a toe pressure ≤ 30 mmHg. In such patients it is important to differentiate neuropathic pain from ischemic rest pain (neuropathic pain typically occurs or worsens at rest in the night). Measurement of the ankle brachial index or toe pressure can easily differentiate the two conditions.

Keywords: Dry gangrene; critical limb ischemia



Figure 7.1 Dry gangrene involving all the toes of the left foot with a necrotic area over the mid-dorsum. Note the hard, dry texture and the clear demarcation between viable and necrotic tissue. (Courtesy of E. Bastounis)

DRY GANGRENE WITH ISCHEMIC NECROSIS OF THE SKIN

Dry gangrene in a female patient with type 2 diabetes, involving the distal parts of the toes of her right foot is illustrated in [Figure 7.2](#). The pedal arteries were not palpable and the ankle brachial index was 0.4. A well-demarcated red area extended up to the ankle and the lateral foot, indicating ischemic necrosis of the skin. Angiography showed the patient to have multilevel severe atherosclerotic disease with involvement of the tibial and pedal arteries. An attempt at mid-tarsal (at Lisfranc's joint) disarticulation was unsuccessful, as it was discovered during the operation that the deep tissues were all necrotic. Finally, the patient sustained a below-knee amputation.

Keywords: Dry gangrene



Figure 7.2 Dry gangrene of the distal areas of the toes of the right foot. The well-demarcated red area extending up to the ankle and the lateral foot indicates ischemic necrosis of the skin. (Courtesy of E. Bastounis)

DRY GANGRENE OF HEEL

A 74-year-old female patient with long-standing type 2 diabetes was admitted to the hospital because of a stroke. She had palsy of her left arm and foot. Her hospitalization was complicated by aspiration pneumonia, which confined the patient to bed for 2 weeks. The patient had a history of ischemic heart disease and hypertension. Peripheral pulses were weak in both feet. On the sixth day of her hospitalization a blister with a black base developed on the posterolateral aspect of her left foot, and it evolved into an ischemic ulcer and dry gangrene ([Figure 7.3](#)). A triplex ultrasonogram revealed extensive severe bilateral stenoses in the superficial femoral and popliteal arteries. Revascularization was not possible

due to the patient's general condition. A heel protector ring was applied so that the heel was completely suspended off the bed and sharp debridement was performed. The ulcer healed after 4 months with daily foot care.

Pressure ulcers are caused by constant pressure over bony heel prominences from an opposing surface such as a mattress. This results in reduced blood flow in the heel with soft tissue necrosis and consequent pressure ulcer development. These ulcers may account for extended hospitalizations and they are recognized as both detrimental to an individual's quality of life and a financial burden to the healthcare system. Pressure ulcers of the heel are preventable by the use of a heel protector ring ([Figure 7.4](#)) or other calf support devices ([Figure 7.5](#)). Since the calf has a large resting surface



Figure 7.3 Dry gangrene due to constant pressure under the bony heel prominence

excessive pressure is avoided. In addition, revascularization should be performed immediately in patients with heel gangrene, since such ulcers heal slowly and may become infected.

Keywords: Heel ulcer; dry gangrene; pressure ulcer; heel protective devices



Figure 7.4 Heel protector ring which keeps the heel suspended and completely off mattress

DRY GANGRENE OF ALL TOES

Dry gangrene in a male diabetic patient involving all the toes is shown in [Figure 7.6](#). In this patient a bypass graft of his leg arteries was not possible because of extensive multilevel disease. The patient sustained a mid-tarsal (at the Lisfranc's joint) disarticulation.

Keywords: Dry gangrene; mid-tarsal disarticulation

WET GANGRENE AND SEPSIS

A 65-year-old male patient who had type 2 diabetes since the age of 45 years and was being treated with sulfonylureas, was brought to the emergency clinic suffering from a fever. He had left paraplegia following a stroke 6 months earlier. One month before admission the toes of his left foot became gradually very painful;

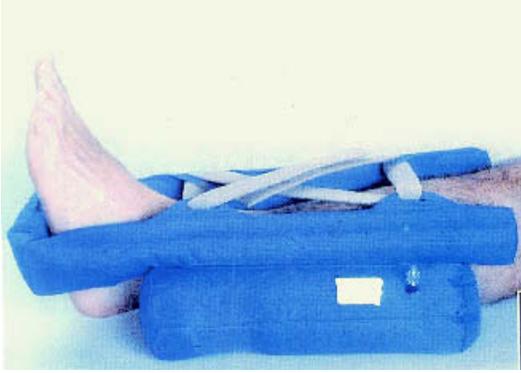


Figure 7.5 Calf support device which provides a larger resting surface thus off-loading pressure from the heel



Figure 7.6 Dry gangrene of all toes



Figure 7.7 A black ischemic ulcer on the dorsum of the left second toe, with edema. Note the whitish tip of the toe due to ischemia. Fungal infection of the thickened nail of hallux with yellowish discoloration and subungual debris is apparent

the patient was usually calm, but occasionally he suffered from bouts of excruciating pain. A general practitioner prescribed cotrimoxazole, pentoxifyllin and fentanyl patches.

On examination, the patient was febrile and his condition was critical. His second left toe was edematous and painful, with a black ischemic ulcer on the dorsum; the tip of the toe was white (Figure 7.7); a gangrenous pressure ulcer was visible on the left heel (Figure 7.8), due to lengthy confinement to bed. Callosity was present under the right fifth metatarsal head, as well as onychodystrophy, due to peripheral vascular disease. No pulses were palpable on his left foot. Both his calves were painful to touch. No other site of infection was found. The patient was classified as Fontaine stage IV. Osteomyelitis was

not found on the radiographs. Swab cultures revealed *Staphylococcus aureus* and *Pseudomonas aeruginosa* and the patient was treated with ciprofloxacin and clindamycin. Blood cultures were negative. On the second day the patient felt better and became afebrile by the third day of hospitalization.

A digital subtraction angiography, carried out 10 days after admission, showed 80% stenosis of both iliac arteries, and almost complete obstruction of both superficial femoral arteries (Figure 7.9), while the popliteal arteries were filled from proximal collateral circulation (Figure 7.10). The peripheral arteries had moderate atheromatous disease. Aorto-iliac intravascular stents were inserted (Figure 7.11).

His second left toe was disarticulated. Surgical debridement of the heel ulcer was



Figure 7.8 Low pressure gangrenous ulcer on the left heel of the foot shown in [Figure 7.7](#)

carried out and calf supportive devices promoted the healing process.

An infected gangrenous area of the foot and particularly on a toe with bounding feet pulses is a condition that is sometimes seen. This is called '*diabetic gangrene*' and it is caused by a thrombosis in the toe arteries which is induced by toxins produced by certain bacteria (mainly staphylococci and streptococci). Plantar abscesses may also result in septic arteritis of the plantar arch and eventually gangrene of the middle toe.

Keywords: Wet gangrene; sepsis; heel ulcer; digital subtraction arteriography; diabetic gangrene

DRY GANGRENE OF THE TOE

A 52-year-old woman with type 2 diabetes mellitus diagnosed at the age of 42 years and being treated with sulfonylureas, was referred to the outpatient diabetic foot clinic



Figure 7.9 Digital subtraction angiography of the foot illustrated in Figures 7.7 and 7.8, showing severe multifocal stenosis of both iliac arteries and almost complete obstruction of both superficial femoral arteries

for dry gangrene of her right fourth toe. No other diabetic complications were reported. She denied intermittent claudication.

A minor painless trauma of the affected toe was reported 1 week previously and the toe became black 24 h later. Edema and redness of the forefoot was reported and she was treated with cotrimoxazole and clindamycin, and bed rest. Within a week the injury became smaller and dried out.

On examination, she had findings of peripheral neuropathy; the pulses in her foot arteries were diminished. The ankle brachial index was 0.5 on the right, and 0.6 on the left side.

The fourth toe was gangrenous and shrunken, and a neuro-ischemic ulcer was noted under the head of the third metatarsal. Scaling of the skin due to edema which had subsided was also observed and onychodystrophy was present (Figure 7.12). Digital

subtraction angiography revealed significant stenosis of both proximal iliac arteries, just after the celiac aortic bifurcation (Figure 7.13). Aortic stents were inserted at the sites of stenosis, by catheterization which was carried out by an experienced radiologist, and the foot circulation was thus restored (Figure 7.14).

An X-ray of her left foot revealed an unknown stress fracture in the proximal phalanx of her fifth toe (Figure 7.15). Osteoarthritis was also apparent in the first and fourth metatarsophalangeal joints.

Ten days after stent insertion the fourth toe was amputated under local anesthesia. No complications occurred postoperatively, and the wound healed completely.

Keywords: Dry gangrene; intravascular stent; stress fracture; revascularization; toe amputation



Figure 7.10 Digital subtraction angiography of the foot illustrated in Figures 7.7–7.9, showing the popliteal arteries being supplied from extensive proximal collateral vessels. (Courtesy of C. Liapis)

STENT

A 71-year-old male patient was admitted because of gangrene in his left foot. Diabetes mellitus was diagnosed on admission and he was treated with low doses of insulin. Antibiotic therapy was initiated, in addition to pentoxifyllin, prostaglandin E1 synthetic analog and phentanyl for the pain. A digital subtraction angiography was carried out which disclosed multiple sites of stenosis in both iliac and superficial femoral arteries (Figure 7.16 upper panel, and Figure 7.17). A suboptimal angioplasty

was carried out on both arteries and stents were inserted (Figure 7.16 lower panel, and Figure 7.18).

During percutaneous transluminal angioplasty a balloon catheter is used to increase the diameter of the lumen of the arteriosclerotic artery. This is a quite safe and minimally invasive technique (as compared to surgery); it preserves saphenous veins, and reduces the length of hospital stay. However, this procedure fails more often in diabetic than in non-diabetic patients due to intimal hyperplasia.

Stents are used to treat suboptimal angioplasty, lesions with severe dissections or

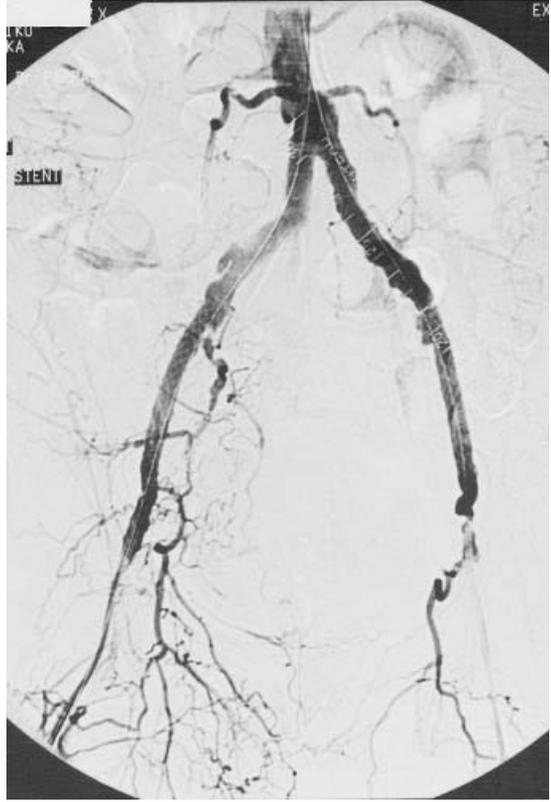


Figure 7.11 Post-stent digital subtraction angiography of foot shown in [Figures 7.7–7.10](#). (Courtesy of C. Liapis)



Figure 7.12 Dry gangrene of right fourth toe. There is a neuro-ischemic ulcer under the third metatarsal head

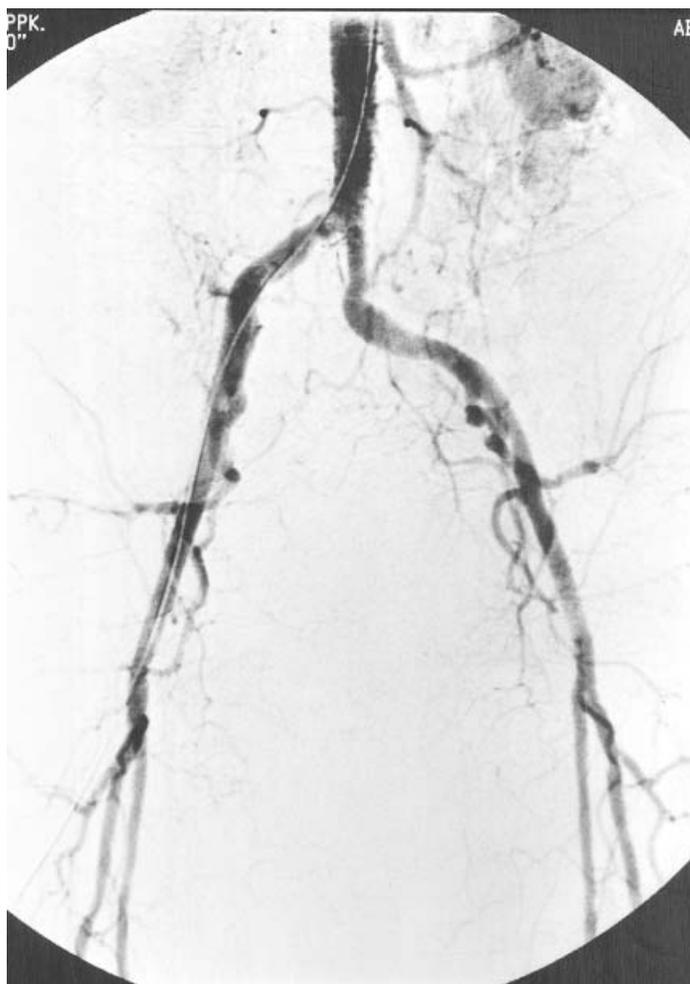


Figure 7.13 Digital subtraction angiography of the foot shown in [Figure 7.12](#). Severe stenosis following the bifurcation of celiac aorta can be seen

significant residual stenosis after angioplasty. The first endovascular stent approved for use in the iliac arteries was the Palmaz stent, a single stainless steel tube, deployed by balloon expansion. The Wall-stent, a flexible self-expanding stent which is available in several different diameter sizes, is also in use. New, covered stents are being evaluated, with the hope that they may mimic surgical grafts and resist re-stenosis.

Keywords: Stents; peripheral vascular disease; angioplasty; digital subtraction angiography

DIGITAL SUBTRACTION ANGIOGRAPHY

A 54-year-old female suffering from type 2 diabetes and being treated with metformin



Figure 7.14 Post-stent digital subtraction angiography of the foot shown in [Figures 7.12](#) and [7.13](#). (Courtesy of C. Liapis)

and insulin, was admitted to the vascular surgery ward; she complained of worsening intermittent claudication in her right leg which had occurred over the previous 2 months. As her ankle brachial index was very low (0.4), a digital subtraction angiography of the abdominal aorta and the arteries of the lower extremities was carried out.

A catheter was inserted through her right brachial artery and the tip of the catheter was advanced into the abdominal aorta.

Advanced stenotic lesions of the abdominal aorta were present with partial stenosis of the lumen. The iliac and common femoral arteries were patent.

Severe stenoses in the superficial femoral arteries were present, predominantly in the right vessel, with a subtotal occlusion of the distal area of the artery ([Figures 7.19](#) and [7.20](#)); extensive collateral vessel development was noted and both popliteal arteries were fairly patent. There was mild atherosclerotic disease in the tibial arteries.

Digital subtraction angiography has replaced film screen angiography since it provides superior contrast resolution and the capability of post-processing the data. It uses less contrast and maximizes guidance for minimally invasive therapy.

Keywords: Peripheral vascular disease; digital subtraction angiography



Figure 7.15 Plain radiograph of the foot shown in [Figures 7.12–7.14](#). A stress fracture of the proximal phalanx of the fifth toe and osteoarthritis in the first and fourth metatarsophalangeal joints can be seen

WET GANGRENE OF THE TOES

A 54-year-old male patient with type 2 diabetes diagnosed at the age of 49 years was admitted to the Vascular Surgery Department because of wet gangrene involving the toes of his left foot. He had been treated with sulfonylurea over the previous 8 years which had led to acceptable diabetes control (HBA_{1c}: 7.5%). The patient was an ex-smoker. During the last 10 years he had also suffered from hypertension which had been treated with an angiotensin converting enzyme inhibitor and a diuretic. He had typical intermittent claudication with

pain in both calves while walking distances of 150 m.

On examination, wet gangrene was noted on the fourth and fifth toes of his left foot. An infected area of ischemic necrosis was also present on the dorsal aspect of his left third toe ([Figure 7.21](#)). The peripheral pulses were absent and the ankle brachial pressure index was 0.4 bilaterally; he also had findings of mild peripheral neuropathy. The patient was in quite severe pain, and he was treated with systemic analgesics and i.v. antibiotics (ticarcillin–clavulanic acid and clindamycin). An angiogram revealed multifocal atherosclerotic lesions of both iliac and superficial femoral arteries ([Figure 7.22](#)), as

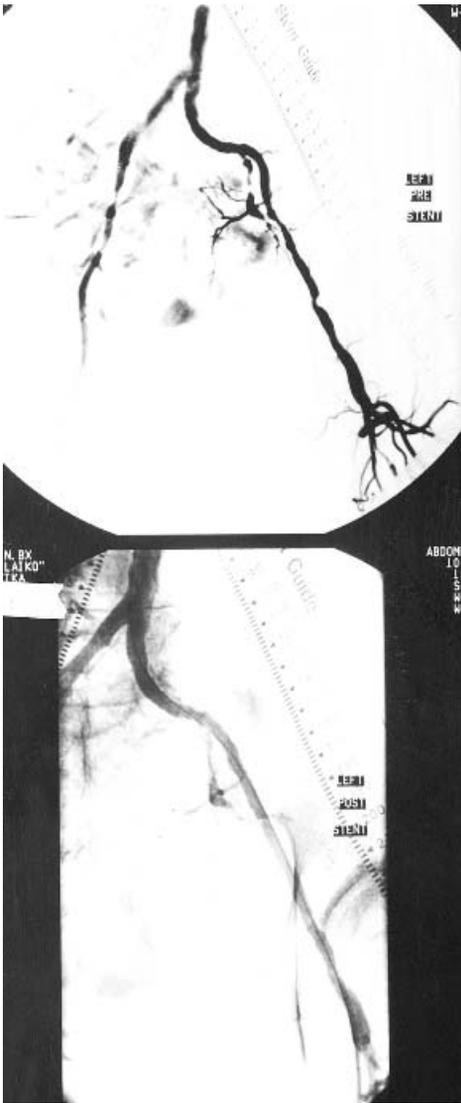


Figure 7.16 Digital subtraction angiography showing multiple sites of stenosis in both iliac and superficial femoral arteries (upper panel). Stent inserted in left superficial femoral artery (lower panel). (Courtesy of C. Liapis)

well as increased development of collateral vessels. A proximal stenosis was noted on both tibial and peroneal arteries. A femoral–popliteal bypass graft and,

eventually, a ray amputation of the last two toes were carried out and the wound was left open for drainage.

Atherosclerotic lesions in diabetic patients occur at sites similar to those in non-diabetics (such as sites of arterial bifurcation), while more advanced disease is common in diabetic patients affecting even collateral vessels. The pathology of the affected arteries is similar in both diabetics and non-diabetics. Typical atherosclerotic lesions of diabetic patients with peripheral vascular disease include diffuse multifocal stenosis. In addition, diabetic peripheral vascular disease has a predilection for the tibioperoneal arteries. All tibial arteries may be occluded with distal reconstitution of a dorsal pedal or common plantar artery. Atherosclerosis begins at a younger age and progresses more rapidly in diabetics than in non-diabetics. While non-diabetic men are affected by peripheral vascular disease much more commonly than non-diabetic women (men-to-women ratio 30:1), the incidence among diabetic men is twice that observed among diabetic women.

Keywords: Peripheral vascular disease; wet gangrene; digital subtraction angiography

WET GANGRENE OF THE FOOT

Gangrene complicated with infection (wet gangrene) in a patient with longstanding type 2 diabetes. Redness and edema, due to infection, extended up to the lower third of the tibia (Figure 7.23). In this patient a below-knee amputation was necessary.

Keywords: Wet gangrene

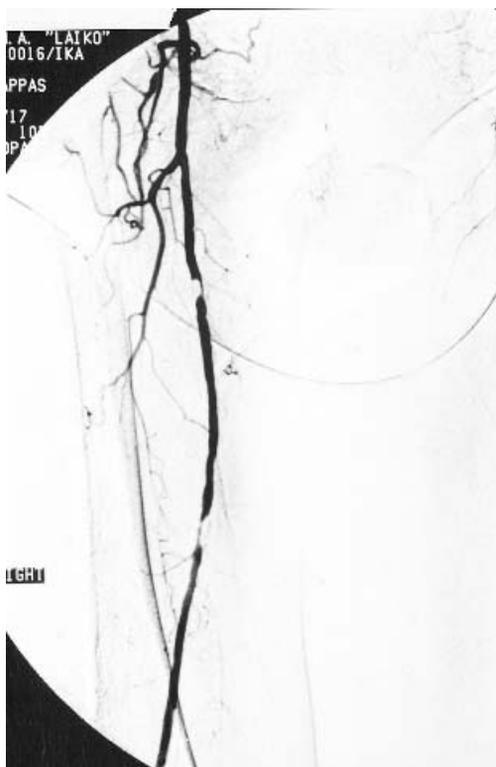


Figure 7.17 Digital subtraction angiography of the foot illustrated in [Figure 7.16](#), showing multiple sites of stenosis in right superficial femoral artery. (Courtesy of C. Liapis)

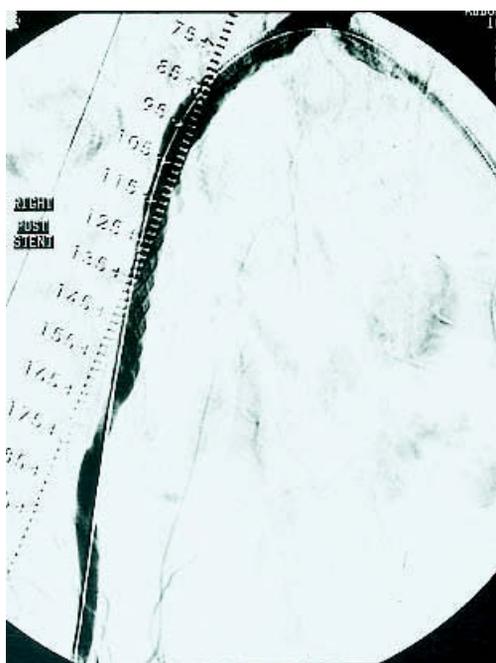


Figure 7.18 Digital subtraction angiography of the foot shown in [Figures 7.16](#) and [7.17](#). Stent inserted in right superficial femoral artery. (Courtesy of C. Liapis)

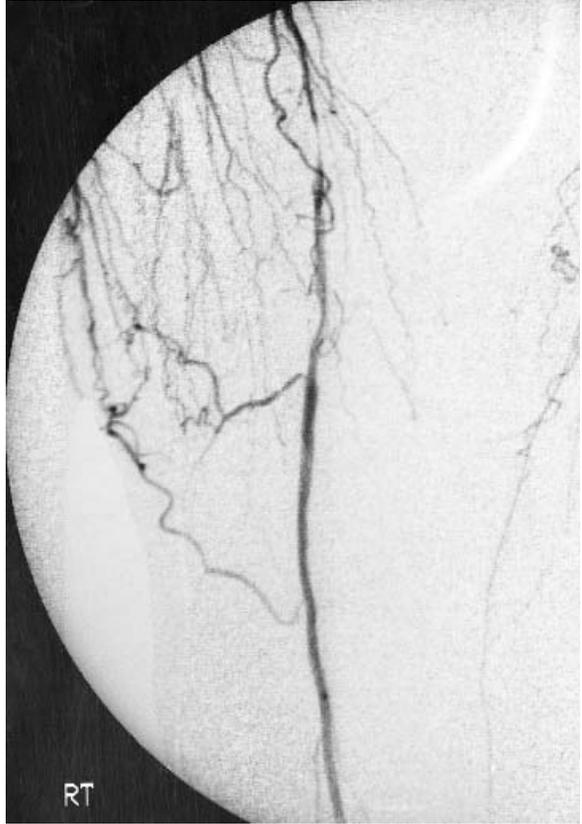


Figure 7.19 Digital subtraction angiography. Severe stenoses in the right superficial femoral artery with extensive collateral vessel development. (Courtesy of C. Liapis)

WET GANGRENE LEADING TO MID-TARSAL DISARTICULATION

A 70-year-old male patient who had type 2 diabetes since the age of 58 years was referred to the outpatient diabetic foot clinic because of wet gangrene of his left foot. He was treated with insulin but his diabetes control was poor. He had hypertension, background diabetic retinopathy and he was a current smoker. The patient noticed black areas on the toes of his foot 7 days previously, but he continued his daily activities since he felt only mild pain.

On examination, he was feverless and his cardiac rhythm was normal. Wet gangrene on his left midfoot and forefoot and an infected necrotic ulcer on the outer aspect of the dorsum were noted (Figure 7.24). An infected ulcer was found under the base of his fifth toe (Figure 7.25), probably the portal of pathogens. Peripheral pulses were absent. He had findings of diabetic neuropathy: loss of sensation of pain, light touch and vibration.

The patient was admitted to the hospital and was treated with i.v. administration of clindamycin plus piperacillin–clavulanic acid. Extensive surgical debridement of the necrotic areas was carried out. An angiogram revealed diffuse peripheral

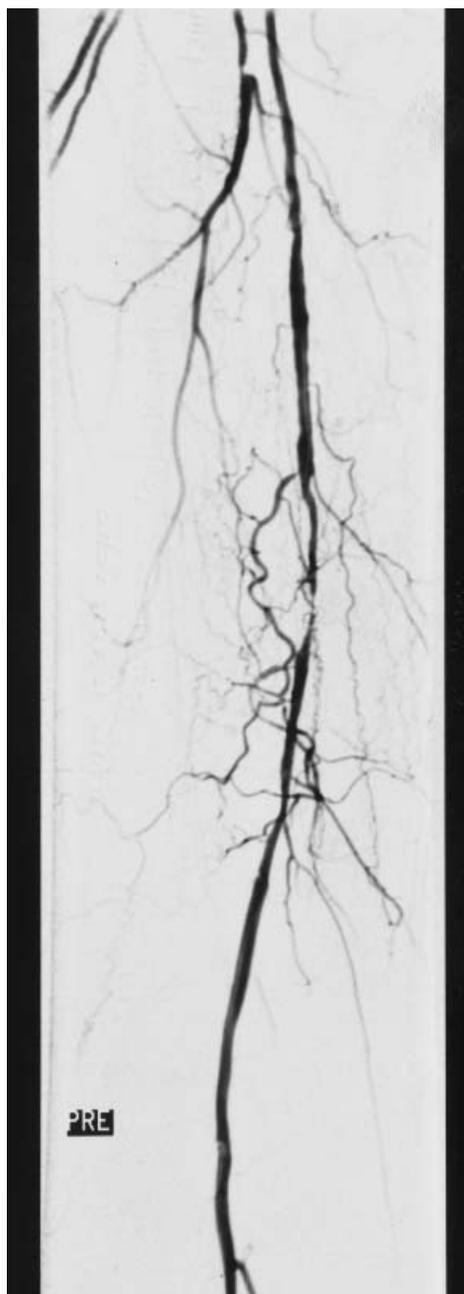


Figure 7.20 Digital subtraction angiography of the foot shown in [Figure 7.19](#). Multilevel stenoses of the left superficial femoral artery. (Courtesy of C. Liapis)

vascular disease with involvement of the pedal arteries. Seven days after admission the patient sustained a mid-tarsal (at Lisfranc's joint) disarticulation.

Wet gangrene is the most common cause of foot amputations in persons with diabetes. It often occurs in patients with severe peripheral vascular disease following infection. Dry gangrene may become infected and progress to wet gangrene. Patients with dry gangrene, awaiting a surgical procedure, should be educated in meticulous foot care. They must be taught to inspect their feet daily, including the interdigital spaces, and wash them twice daily with mild soap and lukewarm water; their feet should be dried thoroughly, particular the web spaces. It is extremely important for patients to avoid wet dressings and debriding agents, as the use of these may convert localized dry gangrene to limb-threatening wet gangrene. The correct footwear is crucial to avoid further injury to the ischemic tissue.

Keywords: Wet gangrene; mid-tarsal disarticulation

EXTENSIVE WET GANGRENE OF THE FOOT

A 51-year-old male patient with type 1 diabetes diagnosed at the age of 25 years was admitted to the Vascular Surgery Department because of extremely painful wet gangrene on his right foot. The patient had proliferative diabetic retinopathy which had been treated with laser, significant loss of his visual acuity (3/10 in both eyes), hypertension and diabetic nephropathy. He had lived in a nursing home. His diabetes control was good (HbA_{1c} : 7%). The patient had complained of pain in his right foot when he was at rest, 4 weeks prior to



Figure 7.21 Wet gangrene of the last two toes. An infected area of ischemic necrosis is also apparent on the dorsal aspect of the third toe. (Courtesy of E. Bastounis)

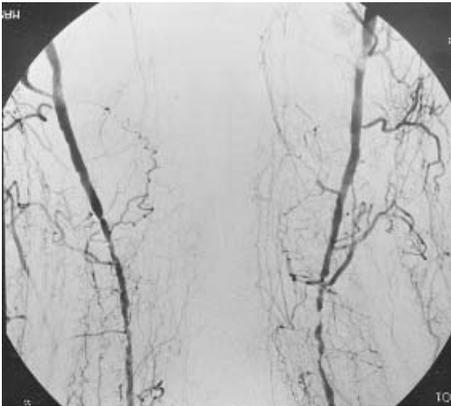


Figure 7.22 Digital subtraction angiography of the foot shown in [Figure 7.21](#). Multifocal atheromatous lesions of both iliac and superficial femoral arteries and increased development of collateral vessels can be seen. This pattern of arterial obstruction is considered typical in diabetes. (Courtesy of E. Bastounis)

admission; the pain worsened progressively and had become refractory to analgesics in the last 2 days. He denied any trauma to his feet. The patient had fever (38.7°C) with rigors and tachycardia; his hemoglobin level was 10 g/l and his white blood cell count was 16,000/l.



Figure 7.23 Wet gangrene of the right foot. Redness and edema due to infection extends as far as the lower third of the tibia. (Courtesy of E. Bastounis)



Figure 7.24 Wet gangrene of midfoot and forefoot in addition to an infected necrotic ulcer on the outer aspect of the dorsum. (Courtesy of E. Bastounis)



Figure 7.25 An infected ulcer under the base of the fifth toe of the patient whose foot is shown in [Figure 7.24](#), probably the portal for pathogens. Gangrene of second toe and mild callus formation under the third metatarsal head can also be seen. (Courtesy of E. Bastounis)



Figure 7.26 Wet gangrene involving the forefoot with cellulitis extending as far as the right ankle. The bone and articular surfaces of the interphalangeal joint of the fourth toe are exposed. Congenital overriding fifth toe and ulceration under the fifth metatarsal is apparent together with onychodystrophy and ingrown nail of hallux. (Courtesy of E. Bastounis)

On examination, he had wet gangrene involving the right forefoot, with cellulitis extending as far as the right ankle (Figure 7.26). The bone and articular surfaces of the interphalangeal joint of the fourth toe were exposed. Ruptured blisters were observed under the right sole (Figure 7.27). The patient was treated with i.v. antibiotics (piperacillin–sulbactam plus metronidazole) while extensive surgical debridement of the necrotic tissue and drainage of the abscess cavities was carried out. *Staphylococcus aureus*, *Escherichia coli* and anaerobic cocci were isolated from a deep tissue culture. An angiograph revealed multilevel atheromatous stenosis of his common femoral, superficial femoral, popliteal and tibial arteries.

The patient had his second and third toes amputated. Extensive longitudinal incisions in the dorsum and the lateral foot were undertaken. Within 2 days his condition worsened rapidly, and he sustained an amputation below his right knee.

Wet gangrene is characterized by a moist appearance, gross swelling and blistering. This is an emergency situation which occurs in patients with severe ischemia who sustain an unrecognized trauma to their toe or foot. Urgent debridement of all affected tissues and use of antibiotics often results in healing if sufficient viable tissue is present to maintain a functional foot together with adequate circulation. If wet gangrene involves an extensive part of the foot, urgent guillotine amputation at a



Figure 7.27 Sole of the foot shown in [Figure 7.26](#) with wet gangrene of the forefoot, ulceration under fifth metatarsal head and ruptured blisters. (Courtesy of E. Bastounis)

level proximal enough to encompass the necrosis and gross infection, may be life saving. At the same time a bypass surgery or a percutaneous transluminal angioplasty should be performed when feasible. Saline gauze dressings, changed every 8 h, work well in open amputations. Revision to a below-knee amputation may be considered 3–5 days later.

Keywords: Wet gangrene; deep tissue infection; onychocryptosis; ingrown nail

WET GANGRENE OF THE HALLUX

A 72-year-old male patient with type 2 diabetes diagnosed at the age of 60 years and being treated with insulin, attended the outpatient diabetic foot clinic because of pain in his right hallux. His diabetes control was poor (HBA_{1c}: 8.7%). He had hypertension and background retinopathy in both eyes. He was an ex-smoker. The patient had



Figure 7.28 Wet gangrene of the right hallux and claw toe deformity. Ischemic changes (loss of hair, redness over toes, dystrophic nail changes) can also be seen

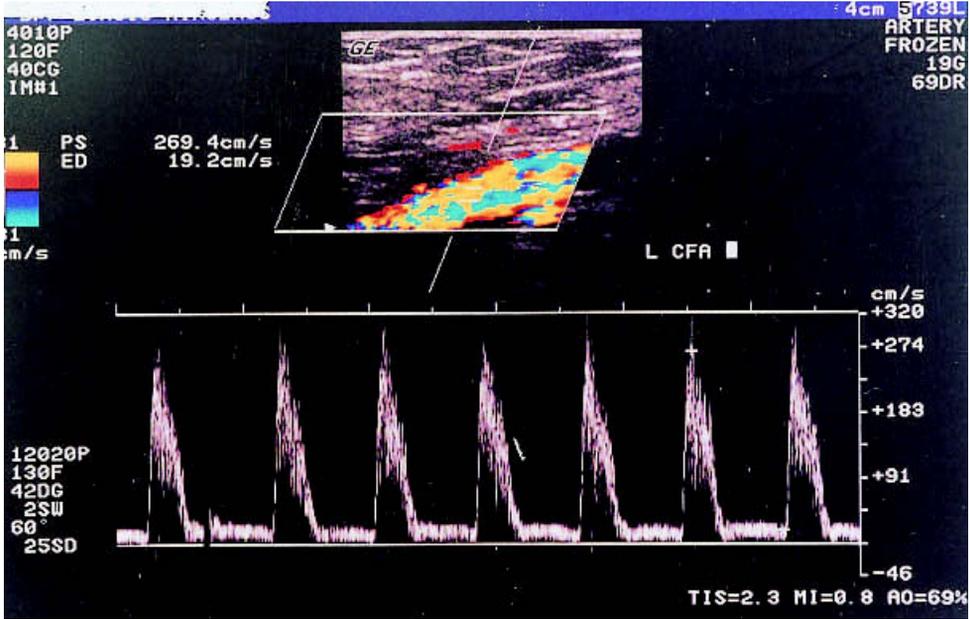


Figure 7.29 Triplex scan of the foot shown in [Figure 7.28](#). Increased peak systolic velocity (PSV) of blood flow (269 cm/s) through the stenotic segment of the left common femoral artery, biphasic flow pattern and widening of the spectral window under systolic peak can be seen (normal PSV in the common femoral artery is approximately 100 cm/s). These findings correspond to a stenosis of the left common femoral artery of 50–60%

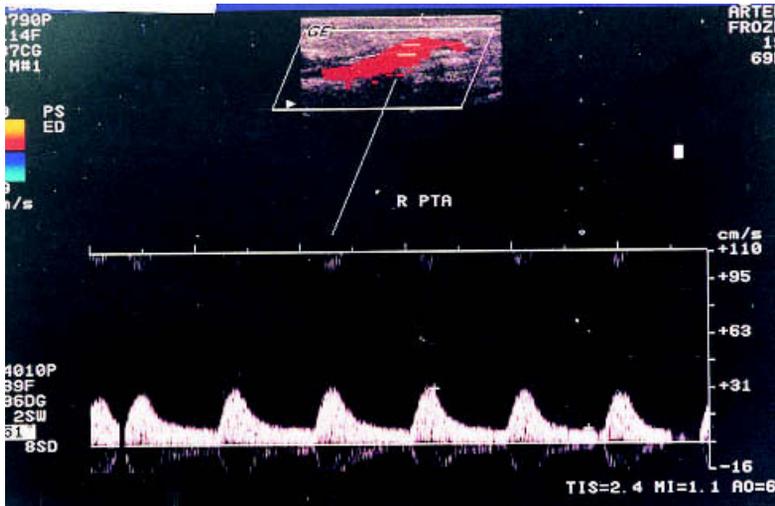


Figure 7.30 Triplex scan of the foot shown in [Figures 7.28](#) and [7.29](#). The spectral window in the right posterior tibial artery is biphasic, the spectrum is wide and the peak systolic velocity (PSV) is reduced (PSV at this level is expected to be about 50 cm/s). These findings denote a proximal stenosis of approximately 60%

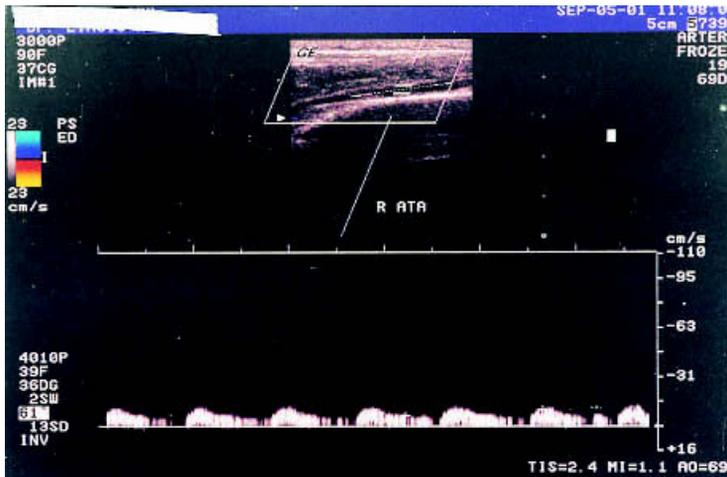


Figure 7.31 Triplex scan of the foot shown in Figures 7.28–7.30. The spectral waveform of the right anterior tibial artery is biphasic, the spectral window is wide, the peak systolic velocity is decreased, the velocity during diastole is increased and the downslope of the waveform is delayed. This pattern of flow is described as *tardus parvus* and corresponds to the presence of a proximal stenosis of 60–70%

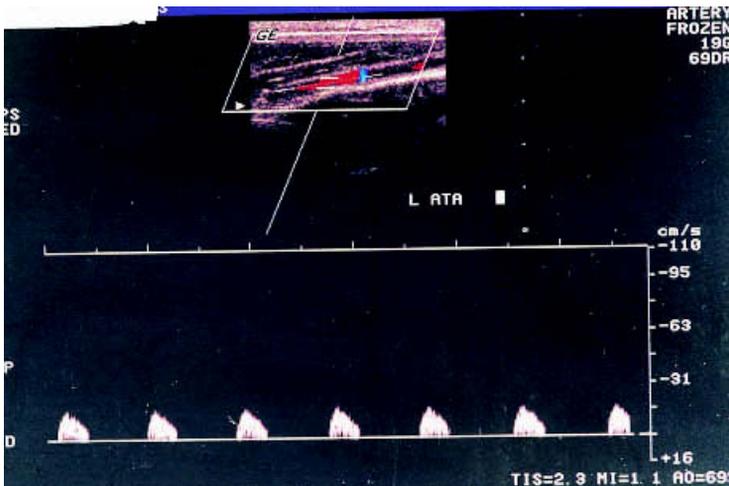


Figure 7.32 Triplex scan of the foot shown in Figures 7.28–7.31. Examination of the left anterior tibial artery shows a monophasic waveform, indicating that a stenosis of greater than 80% is present

ischemic rest pain due to peripheral vascular disease (Fontaine's stage IV). Six days earlier he had become aware of a worsening pain in his right hallux, the onset of which had been acute.

On examination, wet gangrene was noted on the right hallux; peripheral pulses were absent and the ankle brachial index was 0.4 bilaterally. He had severe peripheral neuropathy (no Achilles tendon reflexes, loss of

sensation of 5.07 monofilaments and vibration; the vibration perception threshold was 45 V in both feet) and claw toe deformity. In addition, ischemic changes of his feet were also noted (loss of hair, redness over toes, dystrophic nail changes, and cold feet) (Figure 7.28).

Onychocryptosis was the cause of his gangrene due to inappropriate nail care, resulting in paronychia and localized ischemic necrosis. The patient was treated with amoxicillin–clavulanic acid. A color duplex scan (triplex) of the leg arteries showed mild atheromatous stenosis in his iliac and common femoral arteries (see below), and severe stenosis in his right superficial femoral artery. An angiogram confirmed the ultrasound findings and revealed a >95% stenosis in the middle of his right superficial femoral artery, with the development of collateral circulation. The right anterior tibial artery was almost completely obstructed just after to the popliteal artery trisection; the foot arteries were patent.

The patient underwent a right aorto-popliteal and a popliteal-peripheral bypass. His recovery was good and the infected hallux improved gradually.

Education in foot care was provided. The patient was advised to wear appropriate footwear.

Increased peak systolic velocity (PSV) of blood flow (269 cm/s) through the

stenotic segment of the left common femoral artery, a biphasic flow pattern and widening of the spectral window under the systolic peak were observed (Figure 7.29) (normal PSV in the common femoral artery is approximately 100 cm/s). These findings correspond to a 50–60% stenosis in the left common femoral artery. The spectral window in the right posterior tibial artery (Figure 7.30) was also biphasic, the spectrum was wide and the PSV was reduced (PSV at this level is expected to be about 50 cm/s) indicating that a proximal stenosis of approximately 60% was present. The waveform of the right anterior tibial artery (Figure 7.31) was biphasic, the spectral window was wide, the peak systolic velocity was decreased, the velocity during diastole was increased and the downslope of the waveform was delayed. This pattern of flow is described as *tardus parvus* and corresponds to the presence of a proximal stenosis of 60–70%. Examination of the left anterior tibial artery (Figure 7.32) showed a monophasic waveform, indicating that a stenosis of greater than 80% was present.

Keywords: Dry gangrene; dystrophic nails; onychocryptosis; ingrown nail; triplex scanning; peak systolic velocity; evaluation of arterial stenosis

Chapter VIII

INFECTIONS

- THE INFECTED FOOT ULCER
- SOFT TISSUE INFECTION UNDER CALLUS
- INFECTION UNDER CALLUS OVER FIFTH TOE
- SOFT TISSUE INFECTION
- WEB SPACE INFECTION
- ONYCHOMYCOSIS
- FUNGAL INFECTION WITH MULTIMICROBIAL COLONIZATION
- DEEP TISSUE INFECTION AFTER INTERPHALANGEAL MYCOSIS
- DEEP TISSUE INFECTION
- DEEP TISSUE INFECTION OF A CHARCOT FOOT WITH A NEUROPATHIC ULCER
- OSTEOMYELITIS
- OSTEOMYELITIS OF THE HALLUX
- PHLEGMON
- INFECTED PLANTAR ULCER WITH OSTEOMYELITIS
- NEUROPATHIC ULCER WITH OSTEOMYELITIS
- OSTEOMYELITIS OF THE FIRST METATARSAL HEAD
- CHRONIC NEUROPATHIC ULCER WITH OSTEOMYELITIS
- BONE SCINTIGRAPHY IMAGING
- OSTEOMYELITIS OF THE HEEL

THE INFECTED FOOT ULCER

Invasion of foot tissues by microorganisms, usually accompanied by an inflammatory response, may follow colonization of the skin by initially harmless bacteria, or occur as a primary event.

Diagnosing a foot ulcer infection is based on clinical criteria. A superficial or a full-thickness ulcer, treated inadequately, predisposes to infection, although cellulitis or osteomyelitis can occur without a break in the skin. Infected ulcers are often asymptomatic, especially if the patient feels no pain, due to diabetic polyneuropathy; or they may cause mild discomfort, and produce some drainage, which eventually may become purulent and odorous. Disturbance of blood glucose control may be early evidence of a local infection.

Clinical assessment of any ulcer includes description of location, appearance, extent, depth, temperature, and odor. *Appearance* includes color, type and condition of tissue, presence of drainage, an eschar, necrosis or surrounding callus. Infected wounds may be purple or red, or even brown or black, depending on the pathogen and its etiology, and their drainage may be serous, hemorrhagic or purulent. Induration of the skin and swelling usually denotes infection. *Extent* is measured either directly on a clear film, or by defining length and width of the ulcer. *Depth* is estimated with a sterile blunt probe, which also determines underlying sinus tracts, abscesses or penetration to a bone. *Temperature* is estimated by the hand of the examiner or measured by a dermal thermometer. Underlying infections raise skin temperature. Foul *odor* of an ulcer may denote

infection by a specific pathogen (such as *Proteus*, *Pseudomonas*, anaerobes, a mixed infection, or fungi), or, simply a necrotic process.

Wound infections are categorized as mild, moderate or severe. *Mild* infections are superficial infections confined to the skin and the subcutaneous fat, with minimal or no purulence or cellulitis. *Moderate* infections are deep and may involve fasciae, muscles, tendons, joints or bones. They may present as cellulitis of 0–2 cm in diameter, or a plantar abscess, and they may cause systemic symptoms; they impose a certain risk of amputation. *Severe* infection of a foot ulcer is a deep infection with more than 2 cm of cellulitis, lymphangitis, gangrene, and/or necrotizing fasciitis, threatening limb loss and causing systemic toxicity. Absence of symptoms or signs of systemic illness does not exclude a limb-threatening infection.

Cultures for aerobic and anaerobic pathogens and fungi assist in the management of infection. Curettage of the base of the debrided ulcer, culture of material collected by surgical biopsy of deep tissue or bone, or aspiration of drainage are preferred; superficial swab cultures are not usually helpful, since they often produce different results from cultures of deep tissues. They are also difficult to interpret because of the number of pathogens found on the surface of a wound and they are unsuitable for anaerobes.

Before culturing a wound, any overlying necrotic tissue should be removed by vigorous scrubbing with saline-moistened sterile gauze.

Bacteria that colonize normal skin are coagulase-negative staphylococci, α -hemolytic streptococci and other gram-positive aerobes, and corynebacteria. *Staphylococcus aureus* or β -hemolytic streptococci,

pathogens that colonize the skin of diabetic patients, are the causative agents of acute infections in antibiotic-naïve patients, and are nearly always the cause of cellulitis in non-ulcerated skin; *Staphylococcus aureus* is the most commonly recovered pathogen in most infections in which a single agent is isolated. Polymicrobial cultures, with an average of five or six organisms, are often obtained from patients with chronic lesions, especially when they have been treated with antibiotics for some time; anaerobes, mostly *Bacteroides* sp. and various anaerobic gram-positive cocci are often isolated from deep necroses; *Proteus* spp. and *Escherichia coli* predominate among gram-negative bacilli; and *Pseudomonas* is often isolated from indurated, wet wounds. In severe infections, gram-negative pathogens and anaerobes predominate, versus gram-positive pathogens and enterobacteriaceae, which are usually isolated from mild infections. Severity of infection does not predict the causative microorganism.

Colonization of skin with any bacterium without concomitant infection, does not need treatment; therefore culture results should be interpreted with caution.

Mild or moderate cellulitis may be treated with dicloxacillin, first-generation cephalosporins or clindamycin. Cotrimoxazole is an alternative for treating a proven staphylococcal infection, as well as for enterobacteriaceae.

When infection is mild and the causative pathogens and their susceptibility to antibiotics are predictable, empirical antibiotic therapy is justified. A narrower-spectrum agent may be chosen, such as first-generation cephalosporins and/or clindamycin. One to three weeks of therapy may suffice for soft tissue infections. The more severe the infection and the higher the prevalence of antibiotic resistance, the greater the need

for microbiological information. Second-generation oral cephalosporins, amoxicillin-clavulanic acid or fluoroquinolones, and clindamycin or metronidazole may be effective oral treatment against moderate infections, for patients who do not need hospitalization, when a mixed infection is suspected or before microbiological data are available.

Patients with severe infections, and those in a systemic toxic condition, should be hospitalized and treated with intravenous antibiotics, since they may be unable to swallow or tolerate oral therapy, in addition to which more predictable levels of antibiotics in infected tissues can be achieved with intravenous administration. Such patients are usually treated with broad-spectrum antibiotics, before cultures and antibiograms are available. Imipenem-cilastin, ampicillin-sulbactam, piperacillin-tazobactam, third-generation cephalosporins or fluoroquinolones are usually effective; clindamycin or metronidazole may be added and an agent with a narrower spectrum may be chosen later based on the antibiograms. Urgent surgical drainage or removal of dead tissues may also be needed. Arterial insufficiency may compromise therapy as it prevents the antibiotics from reaching the site of infection. In this case surgical revascularization of the limb is carried out.

Radiographic evaluation is necessary when osteomyelitis is suspected or for wounds of long duration. Subcutaneous gas, foreign bodies, fractures, cortical erosions or neuro-osteopathy may be seen on plain radiographs.

Keywords: Infection, clinical assessment; infection, diagnosis; cultures, curettage; infection, mild; infection, moderate; infection, severe

SOFT TISSUE INFECTION UNDER CALLUS AT THE TIP OF A CLAW TOE

In the case of claw toe deformity of the second toe, callus formation and onychodystrophy was due to exposure of the tip of the toe to high pressure during the propulsion phase of gait.

A purulent discharge was evident after removal of the callus (Figure 8.1). The patient felt no pain or discomfort due to severe peripheral neuropathy. No other signs of infection were present on the toe or the forefoot, therefore drainage of pus was adequate, and no further treatment was needed. Appropriate footwear was prescribed.

Keywords: Soft tissue infection; claw toe; onychodystrophy; subungual hemorrhage; third ray amputation

INFECTION UNDER CALLUS OVER FIFTH TOE

A 66-year-old female patient, with type 2 diabetes which was diagnosed at the age of 51 years was referred to the diabetic foot clinic for chiropody treatment. On physical examination she had severe peripheral neuropathy, and bounding pedal pulses. Superficial vein dilatation on the dorsal foot, dry skin—as a result of neuropathy—and hyperkeratosis over the fifth metatarsal head were noted. Painless hyperkeratosis was seen over the dorsal aspect of the left little toe, caused by inappropriate footwear. After callus removal a purulent discharge was noticed (Figure 8.2). A coagulase-negative *Staphylococcus aureus* was isolated from the pus. A plain radiograph was negative for bone involvement. No antibiotic was given since the pus had drained completely. The ulcer healed within 2 weeks after local treatment.



Figure 8.1 Purulent discharge was evident after removal of callus from the tip of this second claw toe with onychodystrophy. A third ray amputation was carried out 2 years ago. A subungual hemorrhage due to intense trimming of the nail of the fourth toe can be seen



Figure 8.2 Superficial vein dilatation can be seen on the dorsal foot along with dry skin and hyperkeratosis over the fifth metatarsal head. There was painless hyperkeratosis over the dorsal aspect of the left little toe. After callus removal purulent discharge was noted

Non-limb-threatening infections are usually caused by gram-positive cocci, typically *Staphylococcus aureus* and *Streptococcus* spp. In hospitalized patients with diabetic foot infections, methicillin-resistant *Staphylococcus aureus* as well as enterococci are more prevalent.

Keywords: Infection; callus, claw toes

SOFT TISSUE INFECTION

A 58-year-old male patient with type 2 diabetes, diagnosed at the age of 45 years, attended the outpatient diabetic foot clinic for chiropody treatment on a fortnightly basis. He had severe peripheral neuropathy, claw toe deformity and prominent metatarsal heads. He mentioned mild pain

on his left midsole on finding a coin in his shoe after prolonged walking.

On examination, he had a lax blister containing purulent fluid under his left midsole. There was extensive surrounding erythema and callus formation under the second metatarsal head (Figure 8.3). The blister was removed and dressed, and the patient was advised to use crutches and walk on his heel. He was treated with clindamycin for 2 weeks. Debridement of the callus was also carried out.

The blister had developed following an unrecognized trauma to the foot. Such injuries are detrimental to patients with loss of sensation. The coin in the patient's shoe put an additional load under his midsole. All patients with loss of protective sensation should be instructed to inspect and feel the inside of their shoes before they wear them. A selection of objects collected from



Figure 8.3 A lax blister containing purulent fluid under the midsole with extensive surrounding erythema. Injury caused by a coin in the shoe which was not felt by the patient. Callosus formation under the second metatarsal head is apparent

patients' shoes at the outpatient diabetic foot clinic is shown in [Figure 8.4](#).

Diabetic bullae may also cause blisters in diabetic patients. They occur on the lower legs, the dorsum of the feet, hands, and forearms and less commonly, under the soles of the feet. Diabetic bullae more often affect men. They appear suddenly as tense and usually bilateral blisters, with diameters of 0.5 to several cm; they contain clear fluid without any surrounding erythema and heal in a few weeks without scarring. Relapses are common.

Keywords: Trauma; in-shoe foreign objects; diabetic bullae

WEB SPACE INFECTION

A 72-year-old female patient with type 2 diabetes diagnosed at the age of 61 years, was referred to the outpatient diabetic foot clinic because of an infection in her left foot. The patient had poor diabetes control (HBA_{1c}: 8.5%), and was being treated with insulin twice daily. She had background



Figure 8.4 Objects collected from patients' shoes. Loss of protective sensation prevents patients from feeling injurious stimuli

diabetic retinopathy and diabetic nephropathy. She reported itching in the fourth interdigital space 6 months previously, probably due to a fungal infection.

On examination, peripheral pulses were weak and she had severe loss of sensation. There was significant ankle edema. A full-thickness painless, infected neuro-ischemic ulcer was present in the fourth web space, with exposure of the subcutaneous tissue (Figure 8.5). No signs of systemic toxicity were found. The ankle brachial index was 0.7. The ulcer was debrided and dressed. Swab cultures obtained from the base of the ulcer revealed *Staphylococcus aureus* and *Escherichia coli*. A plain radiograph excluded osteomyelitis. She was treated with amoxicillin–clavulanic acid for 2 weeks. Oral furosemide was initiated in order to reduce the ankle edema. The patient had her wound dressed at home daily and attended the outpatient diabetic foot clinic on a weekly basis. A triplex examination of the leg arteries revealed the presence of moderate stenosis in the left superficial femoral and left popliteal arteries. No vascular surgery was

undertaken at that time as the ulcer healed progressively.

Fungal infections develop as a result of poor foot hygiene, hyperhidrosis, and accumulation of moist detritus in the webs (Figure 8.6 shows another patient). *Interdigital tinea pedis* is the most common form of chronic fungal foot infection. Itching, redness, scaling, erosion and soaking of the skin with fluid usually occur, while in the late phase the redness subsides. *Trichophyton metagrophytes*, *Trichophyton rubrum* or *Epidermophyton floccosum* may be found.

Topical terbinafine cream cures most infections caused by *Dermatophyta*, and should be continued for 2 weeks after symptoms subside.

Superficial bacterial infections in the interdigital spaces may cause thrombosis of the adjacent digital arteries and spread to deeper structures through the lumbrical tendons. Furthermore, edema impedes foot circulation, especially in the presence of peripheral vascular disease. Adequate foot hygiene and treatment of the fungal infection could prevent this complication.



Figure 8.5 Full-thickness, infected neuro-ischemic ulcer in the fourth web space with exposure of the subcutaneous tissue. Ankle edema due to diabetic nephropathy, and onychodystrophy can be seen



Figure 8.6 Fungal infection in the interdigital space with claw toe deformity

Keywords: Web space; infection; fungus infection; chronic tinea pedis; *Trichophyton metagrophytes* *Trichophyton rubrum* or *Epidermophyton floccosum*.

ONYCHOMYCOSIS

The patient whose feet are shown in [Figures 5.12–5.16](#) was suffering from onychomycosis and the nails and nail beds were completely destroyed by fungus as shown in [Figure 8.7](#). Onychomycosis *per se* does not cause foot problems, but when it affects the proximal nail (proximal subungual onychomycosis) it may cause chronic paronychia and serve as a portal for bacteria, resulting in deep tissue infection. It often co-exists with mycosis of the web spaces and it may be superinfected by bacteria, leading to deep tissue infection as well.

This patient was treated with terbinafine hydrochloride, both systemic (tablets 250 mg once daily) and topical (cream), for 3 months and was instructed in the appropriate foot care.



Figure 8.7 Fungal infection affecting all nails in addition to claw toes. Patient whose feet are shown in [Figures 5.12–5.16](#)

Chronic onychomycosis is classified into three clinical types: *distal subungual onychomycosis* is the most common form. The distal edge of the nail becomes infected and a yellow discoloration, onycholysis

and subungual debris develop. In *proximal subungual fungal infection*, the second commonest form, *Trichophyton rubrum* accumulates hyperkeratotic debris under the nail plate and loosens the nail, eventually separating it from its bed. This fungus infects the underlying matrix and nail plate leaving the nail surface intact. *Leuconychia mycotica*, caused by *Trichophyton metagrophytes*, infects the nail superficially. The nail surface becomes dry, soft and friable but the nail remains attached to its bed. In addition to these fungi *Epidermophyton floccosum* may also be isolated from infected areas.

Itraconazole and fluconazole are also effective in the treatment of chronic onychomycosis.

Keywords: Onychomycosis; distal subungual onychomycosis; proximal subungual fungal infection; leuconychia mycotica; *Trichophyton metagrophytes*, *Trichophyton rubrum* or *Epidermophyton floccosum*; terbinafine; itraconazole; fluconazole

FUNGAL INFECTION WITH MULTIMICROBIAL COLONIZATION

Superficial ulcers of 10 days' duration on the facing sides of the left first and second toe of a 70-year-old type 2 diabetic lady with diabetic neuropathy, before debridement are shown in [Figures 8.8](#) and [8.9](#). Note soaking of the skin. An X-ray excluded osteomyelitis. Staphylococcus coagulase-negative, *Pseudomonas aeruginosa* and enterobacteriaceae were recovered after swab cultures in addition to *Candida albicans*. She was treated successfully with itraconazole for 5 weeks. The patient used a clear gauze in order to keep her toes apart, together with local hygiene procedures twice daily. Weekly debridement was carried out and no antimicrobial agent was needed.

Keywords: Fungal infection



Figure 8.8 Neuro-ischemic ulcers facing each other on the first and second toe with fungal infection and soaked skin in addition to claw toes. Foot shown from the plantar aspect



Figure 8.9 Neuro-ischemic ulcers facing each other on first and second toe, with fungal infection. Front aspect of [Figure 8.8](#)

DEEP TISSUE INFECTION AFTER INTERPHALANGEAL MYCOSIS

A 60-year-old female patient with type 2 diabetes diagnosed at the age of 47 years and treated with sulfonylurea and metformin and with poor glycemic control, was referred to the diabetic foot outpatient clinic because of a severe foot infection.

The patient had known mycosis between the fourth and the fifth toes of her right foot. Three days before her visit she noticed redness and mild pain on the dorsum of her toes. Her family doctor gave her cefaclor, but she became febrile and her foot became swollen, red and painful. No trauma was reported.

On examination, her foot was red, warm and edematous with pustules on its dorsum ([Figure 8.10](#)). The peripheral arteries were normal on palpation and peripheral neuropathy was present. Pathogen entry was probably via the area of the mycosis.

The patient was admitted to the hospital and treated with intravenous ciprofloxacin and clindamycin. No osteomyelitis was found on repeated radiographs. Extensive surgical debridement was carried out. Deep tissue cultures revealed *Staphylococcus aureus*, *Escherichia coli* and anaerobes. The patient was discharged in fair condition after a stay of 1 month.

Keywords: Mycosis; deep tissue infection

DEEP TISSUE INFECTION

A 50-year-old type 1 male diabetic patient with known diabetes since the age of 25 years was referred to the outpatient diabetic foot clinic for a large infected neuro-ischemic ulcer.

The patient suffered from retinopathy—treated with laser—established diabetic nephropathy, hypertension—treated with enalapril and furosemide—and severe neuropathy.



Figure 8.10 Deep tissue infection of the foot following web space mycosis. Redness and edema of the whole foot with pustules on the dorsum can be seen along with claw toes

Six months before visiting a surgeon, the patient had noticed a painless superficial ulcer caused by a new pair of shoes. Hoping it would subside quickly, he did not seek a doctor's advice and continued his daily activities although the ulcer became larger with surrounding erythema and eventually became purulent and odorous. Fever developed. A deep tissue culture revealed *Staphylococcus aureus*, *Klebsiella* spp. and anaerobes. Surgical debridement was carried out, and amoxicillin–clavulanic acid treatment was initiated. After 1 month of stabilization, with dressings being changed daily, the patient noticed increased purulent discharge and an intense foul odor.

On examination at the diabetic foot clinic, the patient was febrile and weak. He had complete loss of sensation. Peripheral pulses were palpable. Gross ankle and forefoot edema was noted and the short extensor of the toes and anterior tibial tendons was exposed (Figure 8.11). The common tendon sheath and subcutaneous tissue were necrosed. An acrid odor emanated from the foot even before the bandages were removed. A seropurulent discharge was being emitted from deeper structures. The patient was referred back to his surgeon; admission to the hospital and intravenous antibiotics together with extensive debridement followed, and due to abiding



Figure 8.11 Deep tissue infection of the foot with gross ankle and forefoot edema. The short extensor of the toes and the anterior tibial tendons are exposed, while the common tendon sheath and subcutaneous tissue are necrosed

septic fever and the critical condition of the patient, a below-knee amputation was undertaken 2 days later.

Keywords: Deep tissue infection; amputation

DEEP TISSUE INFECTION OF A CHARCOT FOOT WITH A NEUROPATHIC ULCER

A 65-year-old female patient with type 2 diabetes mellitus since the age of 40 years attended the diabetic foot clinic because of a large ulcer of the sole of her left foot. She was being treated with insulin resulting in acceptable diabetes control (HbA_{1c}:

7.28%). She had a history of hypothyroidism as well as a history of ulcers under her right foot at the age of 63 years, which had healed completely.

The present ulcer had developed after a minor trauma to the sole of her foot while walking barefoot during the summer. It evolved within a month together with a fast progressing gross deformity of the foot. The patient complained of mild discomfort but no pain, so she kept on using both feet without any means of reducing the pressure on her ulcerated foot. She was treated with amoxicillin–clavulanic acid and clindamycin for 20 days.

On examination, her left foot was swollen, with midfoot collapse; it was warm (2.5°C temperature difference to the contralateral foot), and crepitus was heard on passive movement. A large neuropathic



Figure 8.12 Neuro-osteoarthropathy. A large neuropathic non-infected ulcer surrounded by callus occupies the midsole



Figure 8.13 Right foot of the patient whose left foot is shown in [Figure 8.12](#). A small, full-thickness neuropathic ulcer within an area of callus is present over the right first metatarsal head

non-infected ulcer of size $8 \times 7 \times 0.4$ cm occupied the midsole surrounded by callus ([Figure 8.12](#)). A small, full-thickness neuropathic ulcer was present within an area of callus formation over the right first metatarsal head ([Figure 8.13](#)). The skin on both her feet was dry and the peripheral pulses were palpable. The vibration perception threshold was 20 V in both feet. Monofilament sensation was absent, as were sensations of light touch, pain and temperature perception.

Debridement was carried out; an X-ray showed disruption of the tarsometatarsal joint (Lisfranc's joint), bone absorption of the first and second cuneiforms and dislocation of the cuboid bone ([Figure 8.14](#)). A diagnosis of acute neuro-osteoarthropathy

was made and a single dose of 90 mg of pamidronate was administered. The presence of ulcers prevented the use of a total-contact cast since daily changes of dressings were needed. The patient was instructed to refrain from walking and to visit the diabetic foot clinic on a weekly basis. After 1 month the midsole ulcer was smaller compared to its initial size ([Figure 8.15](#)) and showed no signs of infection. The ulcer under her right sole healed. There was no difference in the temperature between the two feet.

After an absence of 3 weeks the patient visited the clinic with acute foot infection and fever. The midsole ulcer was

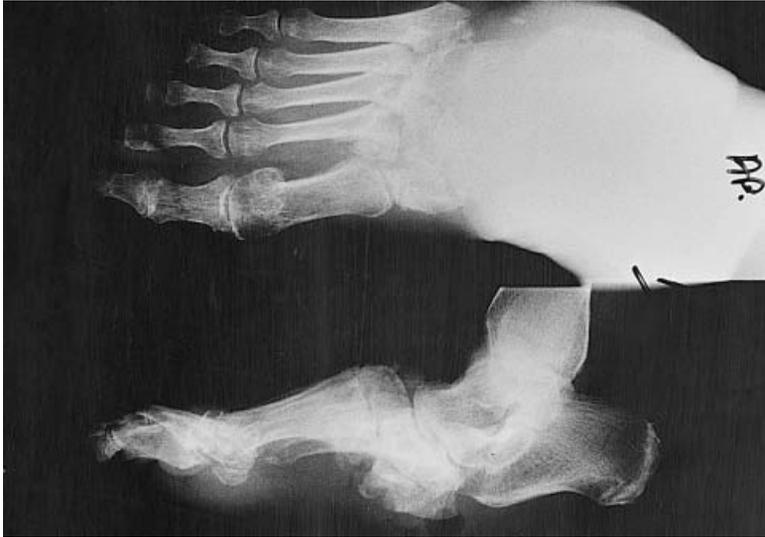


Figure 8.14 Plain radiographs showing neuro-osteoarthropathy in the left foot of the patient whose feet are illustrated in [Figures 8.12](#) and [8.13](#). Disruption of the tarsometatarsal joint (Lisfranc's joint), resorption of the first and second cuneiforms and midfoot collapse can be seen



Figure 8.15 Left neuro-osteoarthropathic foot of the patient whose feet are shown in [Figures 8.12–8.14](#). Progress of the plantar neuropathic ulcer after 1 month of chiropody treatment. Healthy granulated tissue covers the bed of the ulcer



Figure 8.16 Left neuro-osteoarthropathic foot of the patient whose feet are shown in [Figures 8.12–8.15](#), 3 weeks after the photograph shown in [Figure 8.15](#) was taken. Signs of infection (cellulitis, blisters and edema) are present

much smaller ([Figure 8.16](#)), surrounded by cellulitis, and a new infected ulcer was present on the lateral aspect of the hind-foot ([Figure 8.17](#)). The patient insisted that she had complied with the instructions, except for the last week, when she felt confident that the ulcer had healed. She was admitted to the hospital and underwent extensive surgical debridement. Intravenous antibiotics (ciprofloxacin, penicillin and clindamycin) were administered but the

high fever persisted despite treatment; the infection spread to the lower tibia and the patient became septic. On the 10th day of hospitalization, the critical condition of the patient necessitated a below-knee amputation. She was discharged in good clinical condition after 1 week.

Keywords: Deep tissue infection; acute neuro-osteoarthropathy; neuropathic ulcer; below-knee amputation



Figure 8.17 Lateral aspect of the foot shown in [Figure 8.16](#). Infection has spread to the whole foot and the lower tibia. The superficial ulcer on the lateral aspect of the hindfoot may have been caused by rupture of a blister

OSTEOMYELITIS

A 69-year-old female patient with type 2 diabetes diagnosed at the age of 54 years and treated with sulfonylurea, was referred to the outpatient diabetic foot clinic for an infection of her right second toe. She had background diabetic retinopathy and hypertension. She complained of numbness and a sensation of pins and needles in her feet at night.

On examination, she had findings of severe neuropathy (no feeling of light touch, pain, temperature, vibration or a 5.08 monofilament; Achilles tendon reflexes were absent; the vibration perception threshold was >50 V in both feet). Peripheral pulses were weak and the ankle brachial index was 0.7. Dry skin and nail dystrophies were present. A superficial ulcer with a sloughy base was seen on the dorsum of

her right second toe which was red, swollen and painful, having a sausage-like appearance ([Figure 8.18](#)). She did not mention any trauma, but inspection of her shoes revealed a prominent seam inside the toe box of her right shoe.

The sausage-like appearance of a toe usually denotes osteomyelitis. Bone infection was confirmed on X-ray, showing osteolysis of the first and second phalanges. *Staphylococcus aureus* and *Klebsiella pneumoniae* were cultured from the base of the ulcer. The patient was treated with cotrimoxazole and clindamycin for 2 months. She was also referred to the Vascular Surgery Department for a percutaneous transluminal angioplasty of her right popliteal artery. After 2 months the ulcer was still active and the patient had local extension of osteomyelitis despite the restoration of the circulation in the periphery. She eventually had her second



Figure 8.18 Sausage-like toe deformity usually denotes underlying osteomyelitis

ray amputated. A bone culture revealed the presence of *Staphylococcus aureus*. She continued with cotrimoxazole for two more weeks.

Keywords: Osteomyelitis; painful–painless feet

OSTEOMYELITIS OF THE HALLUX

A 30-year-old male patient with type 1 diabetes diagnosed at the age of 11 years was admitted because of infected foot ulcers on his right hallux. He had a mild fever and a history of proliferative diabetic retinopathy and microalbuminuria. Diabetes control was poor (HBA_{1c}: 9.5%). He reported a trauma to his left foot 2 months earlier when an object fell on his feet while working. A superficial ulcer had developed on the dorsal aspect of his right great toe; the ulcer had become infected because the patient felt no pain and therefore did not seek medical advice.

On examination, pedal pulses were normal. Severe peripheral neuropathy was found and the vibration perception threshold was 30 V in both feet. An infected right hallux with purulent discharge, necrotic tissue at the tip, and cellulitis were observed (Figure 8.19). A plain radiograph showed osteomyelitis involving both distal phalanges (Figure 8.20).

A culture of the pus revealed *Pseudomonas maltophilia*, *Enterobacter cloacae* and



Figure 8.19 Infection of the hallux with purulent discharge, necrotic tissue at the tip and cellulitis



Figure 8.20 Osteolysis of the distal phalanx and condyle of the proximal phalanx due to osteomyelitis of the hallux. Plain radiograph of the foot shown in [Figure 8.19](#)

anaerobes, and the patient was treated with ciprofloxacin and ampicillin–sulbactam for 2 weeks, based on the antibiogram. An amputation of the right great toe was undertaken due to persistent osteomyelitis.

Keywords: Hallux; osteomyelitis; amputation

PHLEGMON

A 62-year-old male diabetic patient with type 2 diabetes diagnosed at the age of 42 years and treated with sulfonylurea,

biguanide and acarbose and whose diabetes control was acceptable, visited the outpatient diabetic foot clinic due to infection of the sole of his right foot. He had hypertension and coronary heart disease treated with metoprolol and aspirin. He had no previous history of foot problems.

On examination, the patient had fever, severe diabetic neuropathy, and bounding pedal pulses. He had hallux valgus, claw toes, prominent metatarsal heads, onychodystrophy and dry skin. Callus formation superimposed on a neuropathic ulcer over his third metatarsal head was present; a callus was also noted over his fifth metatarsal head. A superficial, painless,

infected ulcer with purulent discharge was present under Lisfranc's joint (Figure 8.21). This infection progressed to a phlegmon 2 days after a minor shear trauma.

The patient was admitted, and intravenous amoxicillin–clavulanate was initiated. A plain radiograph excluded osteomyelitis or gas collection within the soft tissues. Computerized tomography revealed a phlegmonous subcutaneous mass under

the base of the metatarsals (Figure 8.22). A sterile probe was used to detect any sinuses or abscesses, but none was found. The patient remained bedridden for 1 week and the infection subsided. He continued antibiotics for one more week with limited mobilization and he was released from hospital in excellent condition. Oral antibiotics were continued for two more weeks. Preventive footwear was prescribed and the



Figure 8.21 Superficial infected ulcer with purulent discharge under Lisfranc's joint. Callus formation is superimposed on neuropathic ulcer over the third metatarsal head with callus formation over the fifth metatarsal head. Hallux valgus, claw toes, prominent metatarsal heads, onychodystrophy and dry skin can be seen

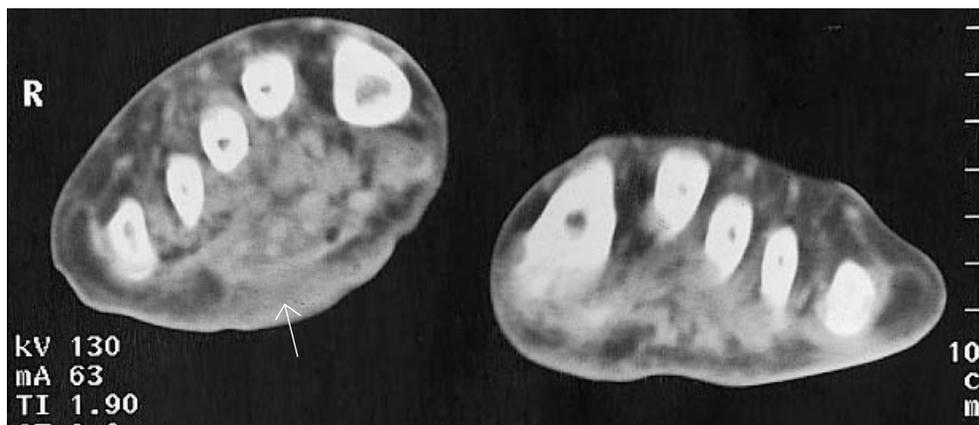


Figure 8.22 Computerized tomography of the feet of the patient whose right foot is shown in [Figure 8.21](#). A phlegmonous subcutaneous mass is present under the base of the metatarsals (arrow)

patient continued to visit the outpatient diabetic foot clinic on a regular basis.

Computerized tomography is useful in identifying areas of phlegmon within the soft tissues. It may provide information about the exact anatomic location and extent, so that aspiration or surgical drainage can be undertaken. Magnetic resonance imaging and ultrasound studies are also helpful in this respect.

Keywords: Neuropathic ulcer; computerized tomography; phlegmon

INFECTED PLANTAR ULCER WITH OSTEOMYELITIS

A 50-year-old female diabetic patient with type 2 diabetes diagnosed at the age of 44 years and treated with sulfonylurea, was referred to the outpatient diabetic foot clinic because of a chronic infected ulcer on her left foot. The patient lived alone and she was being treated for depression; she had good diabetes control. A minor

trauma under her left foot was reported to have occurred 2 years previously. She had treated the injury with different types of gauzes and creams, but it failed to heal. She presented to the clinic with a large, painless, infected ulcer under her left foot ([Figure 8.23](#)).

On examination, an irregular, soaked, foul-smelling ulcer with sloughy bed, and surrounding cellulitis of 3 cm in diameter was found; body temperature was normal. Diabetic neuropathy was diagnosed, while peripheral pulses were normal. Signs of osteomyelitis (osteolysis of the first metatarsal head, and the base of proximal phalanx of the hallux, with periosteal reaction) were noted on the radiograph ([Figure 8.24](#)). A post-debridement swab culture from the base of the ulcer revealed methicillin-resistant *Staphylococcus aureus* and *Escherichia coli*. The patient was admitted to the hospital. The white blood cell count was 14,700/mm³, anemia (Hb: 9.8 g/dl) characteristic of chronic disease was found, the erythrocyte sedimentation rate was 90 mm/h and the level of C-reactive protein was 70 mg/dl. She was treated with 600 mg teicoplanin



Figure 8.23 A large, irregular, soaked and infected neuropathic ulcer with sloughy bed and surrounding cellulitis of 3 cm in diameter is shown here. A minor trauma reported to have occurred 2 years earlier was the cause of this ulcer

intravenously once daily and the ulcer was debrided and dressed. The cellulitis progressively subsided, the ulcer became clear and healthy granulating tissue began to cover the ulcerated area (Figure 8.25). The patient was discharged from the hospital in good clinical condition. She continued treatment with intramuscular teicoplanin for three more months and attended the outpatient diabetic foot clinic on a weekly basis. Complete offloading of pressure from the ulcerated area was achieved by the use of a wheelchair for most of her activities. Platelet-derived growth factor- β (becaplermin) was



Figure 8.24 Osteolysis of the first metatarsal head and the base of proximal phalanx of the hallux with periosteal reaction due to osteomyelitis are shown on this plain radiograph of the foot illustrated in Figure 8.23

applied once daily. The ulcer diminished progressively (Figure 8.26) and healed in 4 months; no relapse occurred.

All patients with deep or long-standing ulcers should be evaluated for osteomyelitis. The possibility of an ulcer being complicated by osteomyelitis increases when the diameter of the ulcer exceeds 2 cm and the depth is greater than 3 mm; the possibility of complications becomes even higher when the white blood cell count, the erythrocyte sedimentation rate and the C-reactive protein levels are high.

Treatment of acute osteomyelitis includes parenteral administration of antibiotics for 2 weeks initially, and the continuation of oral treatment for a prolonged period (at least 6 weeks).



Figure 8.25 Clear ulcer with healthy granulating tissue after 1 month of appropriate treatment in the patient whose foot is shown in [Figures 8.23 and 8.24](#)

Keywords: Neuropathic ulcer; acute osteomyelitis; platelet-derived growth factor- β (PDGF- β , becaplermin)

NEUROPATHIC ULCER WITH OSTEOMYELITIS

A 57-year-old obese male patient with type 2 diabetes diagnosed at the age of 40 years was referred to the outpatient diabetic foot clinic because of a chronic ulcer under his right foot. He was being treated with insulin and metformin with acceptable diabetes control (HBA_{1c}: 7.8%). He had a history of background retinopathy and cataract in both eyes. He reported a severe deep tissue infection 5 years earlier after a burn sustained under his right foot. At that time he was hospitalized for about 1 month and treated with intravenous antibiotics and surgical debridement.

On examination, the patient had severe diabetic neuropathy with loss of sensation of pain, light touch, temperature, vibration,



Figure 8.26 Healing of the ulcer affecting the foot shown in [Figures 8.23–8.25](#). This photograph was taken 3 months after that shown in [Figure 8.25](#)



Figure 8.27 Full-thickness neuropathic ulcer post-debridement under a prominent fourth metatarsal head



Figure 8.28 Commercially-available extra depth therapeutic shoe

and 5.07 monofilaments. Achilles tendon reflexes were absent. The vibration perception threshold was above 50 V bilaterally, while the peripheral pulses were normal. A scar was noted on the dorsum of his right foot which had an overriding fourth toe, as a result of past surgical procedures. A full-thickness neuropathic ulcer was present under his fourth metatarsal head surrounded by callus (Figure 8.27).

A bony prominence could also be felt under the ulcerated area. A plain radiograph did not show osteomyelitis or neuro-osteoarthropathy. Debridement of the ulcer was carried out and extra depth therapeutic shoes with a flat insole were prescribed (Figure 8.28); a window was made in the insole in order to offload pressure on the ulcerated area; the ulcer began to heal well (Figure 8.29).



Figure 8.29 Healing of the neuropathic ulcer shown in [Figure 8.27](#) pre-debridement

The patient kept himself very active. He returned to the clinic after 3 weeks absence with a deeper ulcer involving the tendons ([Figure 8.30](#)). The underlying bone could not be detected with a sterile metal probe and a plain radiograph did not show osteomyelitis. An elevated erythrocyte sedimentation rate (74 mm/h) and mild leukocytosis were found, therefore the possibility of osteomyelitis was high. A magnetic resonance imaging-T1-weighted sagittal image of the foot was obtained, showing a phlegmonous mass starting from the skin and extending to the deeper tissues causing erosion of the fourth metatarsal head ([Figure 8.31](#)). The patient was hospitalized so that offloading pressure from the ulcerated area was enforced, and intravenous antibiotics were administered. Two weeks later the size of the ulcer had decreased by almost 50%.

Several methods are used for the diagnosis of osteomyelitis. *Probe-to-bone tests* (contacting the bone with a sterile metal probe) have a sensitivity of more than 90% and they are carried out at the bedside. *Plain radiographs* have a sensitivity of 55%, but when repeated—usually

2 weeks later—the sensitivity is higher, making this the most cost-effective diagnostic procedure. *Computerized tomography* may reveal areas with subtle abnormalities such as periosteal reactions, small cortex erosions and soft tissue abnormalities. *Magnetic resonance imaging* has a sensitivity of almost 100% and a specificity of over 80% and has the potential to reveal abscesses. Therefore this is the preferred method for the diagnosis of osteomyelitis in many centers in cases where the plain radiographs do not provide sufficient information to make a conclusive diagnosis. However, the specificity of MRI decreases in the presence of neuro-osteoarthropathy, prior bone biopsy, recent bone fracture or recent surgery. *Magnification radiography* is also a very useful method for the detection of early osteomyelitis and it is used to follow up the disease.

Bone scintigraphy imaging is explained in [Figure 8.37](#).

Keywords: Neuropathic ulcer; magnetic resonance imaging; MRI; osteomyelitis; diagnostic methods for osteomyelitis



Figure 8.30 The neuropathic ulcer shown in [Figures 8.27](#) and [8.29](#) has been aggravated by the patient's refusal to reduce activity levels and poor compliance with measures to offload pressure from the affected area

OSTEOMYELITIS OF THE FIRST METATARSAL HEAD

A 74-year-old male patient with type 2 diabetes attended the outpatient diabetic foot clinic because of a chronic painless ulcer on the medial aspect of the right first metatarsal head ([Figure 8.32](#)). The ulcer

developed over a bunion deformity, and had persisted for 10 months.

On examination, the peripheral pulses were palpable and the patient had severe peripheral neuropathy. He could not feel pain, light touch, vibration or 5.07 monofilaments. The vibration perception threshold was above 50 V in both feet. After debridement, the underlying bone could be felt by means of a sterile probe. A plain radiograph revealed osteomyelitis of the first metatarsal head and the proximal phalanx of the right great toe ([Figure 8.33](#)). The patient sustained a first ray amputation.

Chronic osteomyelitis needs surgical removal of the infected bone. However, recent data suggest that prolonged treatment with antibiotics (for 1 or 2 years) may eradicate chronic osteomyelitis. However, no consensus on this issue exists at present.

Keywords: Chronic osteomyelitis; first ray amputation; neuropathic ulcer

CHRONIC NEUROPATHIC ULCER WITH OSTEOMYELITIS

A 46-year-old male patient with type 1 diabetes diagnosed at the age of 27 years was referred to the outpatient diabetes foot clinic because of a chronic ulcer under his right fifth metatarsal head. He had acceptable diabetes control (HbA_{1c} : 7.7%), proliferative diabetic retinopathy treated with laser in both eyes, but no nephropathy. He complained of muscle cramps during the night and chronic constipation interrupted by episodes of nocturnal diarrhea. The patient had a history of painless diabetic foot ulceration for 3 years under his right foot after a burn injury. He had attended the surgery department of a country hospital,



Figure 8.31 MRI image showing osteomyelitis. A magnetic resonance imaging-T1-weighted sagittal image of the foot illustrated in [Figure 8.30](#) showing a phlegmonous mass (arrow) extending from the skin into the deeper tissues and causing erosion of the fourth metatarsal head



Figure 8.32 Chronic neuropathic ulcer over a bunion deformity



Figure 8.33 Plain radiograph of the foot illustrated in [Figure 8.32](#) showing bone resorption, periosteal reaction and destruction of metatarsophalangeal joint of the hallux due to osteomyelitis

where he had his foot dressed and several courses of antibiotics were prescribed. The patient continued to keep himself active, without any special footwear since he felt no discomfort or pain.

On examination, severe diabetic neuropathy was found. The peripheral pulses were palpable and a full-thickness neuropathic ulcer with gross callus formation was observed under his right fifth metatarsal head ([Figure 8.34](#)). Sharp debridement was carried out and the underlying bone was probed with a sterile probe. A plain radiograph revealed pseudoarthrosis of a stress fracture of the upper third of his fifth metatarsal, bone resorption in the metatarsophalangeal joint, and osteolytic lesions in the fifth metatarsal epiphysis ([Figures 8.35](#) and [8.36](#)). Post-debridement cultures from the base of the ulcer revealed *Staphylococcus aureus*, *Proteus vulgaris* and *Enterococcus* spp. The patient was treated with amoxicillin–clavulanic acid 625 mg three times daily for 2 weeks. He was advised to rest and appropriate footwear and insoles were prescribed. A fifth ray amputation was undertaken and antibiotics continued for two more weeks. A bone culture revealed

Staphylococcus aureus. The wound healed completely in 2 weeks.

A ray amputation consists of removal of a toe together with its metatarsal. The uninvolved half of the fifth metatarsal shaft was preserved, so that it retained the insertion of the short peroneal muscle. Ray amputation results in narrowing of the forefoot, but the cosmetic and functional result is excellent. However, the biomechanics of the foot are disturbed after such an operation and this leads to the exertion of high pressure under the metatarsal heads of the adjacent rays.

Keywords: Neuropathic ulcer; osteomyelitis; ray amputation

BONE SCINTIGRAPHY IMAGING

The scintigraphy findings of a patient with possible osteomyelitis are discussed below and the history of this patient is illustrated in [Figures 9.3](#) to [9.5](#) in Chapter 9.

A plain radiograph showed findings compatible with osteomyelitis or neuro-osteoarthropathy of the second and third



Figure 8.34 Full-thickness chronic neuropathic ulcer with gross callus formation under the right fifth metatarsal head

metatarsal heads of this female patient. She was referred for a technetium-99m (^{99}Tc) phosphonate scan. Images obtained during the *flow phase* are shown in the left upper panel of [Figure 8.37](#); during this phase a series of 3-s image acquisitions of the site in question is obtained. They showed increased radionuclide uptake by the tarsometatarsal area of her left foot. A static blood pool image (*blood pool phase*) obtained 5 min later is shown in the right upper panel. A static delayed

image (*delayed phase*) obtained 3 h later is shown in the left lower panel. All images showed increased uptake in the same areas. A gallium-67 citrate study performed on the same day (right lower panel of [Figure 8.37](#)), showed increased radionuclide uptake at the tarsometatarsal area. Based on the results of bone scintigraphy the patient was diagnosed as having osteomyelitis in the tarsometatarsal area.

^{99}Tc scintigraphy is useful in cases of questionable osteomyelitis. It has a high sensitivity (over 90%) but a low specificity (33%), particularly in the presence of neuro-osteoarthropathy. Although increased radionuclide uptake during the flow and pool phase is not specific to the diagnosis of osteomyelitis (it may mean soft tissue, bone infection or both), delayed images of the ^{99}Tc scintigraphy showed increased blood flow to the bones only, thus increasing the specificity of the method in the diagnosis of bone infection. Patients with neuro-osteoarthropathy have increased bone blood flow in the absence of osteomyelitis.

Like ^{99}Tc scintigraphy, gallium-67 citrate accumulates in both osteomyelitis and neuro-osteoarthropathy. This is the reason for its low specificity in the diagnosis of osteomyelitis in diabetic patients. Indium-111 white blood cell imaging (^{111}In WBCs) is expensive, time consuming, has poor spatial resolution and does not distinguish soft tissue from bone infection.

Keywords: Scintigraphy; bone scans; diagnosis of osteomyelitis

OSTEOMYELITIS OF THE HEEL

A 71-year-old female patient with type 2 diabetes was admitted to the hospital



Figure 8.35 Anteroposterior plain radiograph of patient of [Figure 8.34](#). Osteomyelitis. Pseudoarthrosis of a stress fracture of the upper third of the fifth metatarsal, bone resorption at the metatarsophalangeal joint, and osteolytic lesions at the fifth metatarsal epiphysis

because of a severe infection of her right foot. She had a history of type 2 diabetes diagnosed at the age of 51 years, diabetic nephropathy, background diabetic retinopathy—treated with laser—hypertension and ischemic heart disease. She also had a history of stroke at the age of 69 years. A heel ulcer caused after the rupture of a



Figure 8.36 Lateral plain radiograph of foot shown in [Figures 8.34](#) and [8.35](#), focused on the osteomyelitic lesion in the fifth metatarsal. Note that the phalanx of fifth toe continues over fourth metatarsal head

blister under her right heel, which developed after walking in tight new shoes, had persisted for about 1 year. The ulcer progressively became deeper and larger. The patient reported two septic episodes with infection at the same site, for which she was hospitalized for prolonged periods.

On examination, her body temperature was 39.2°C, blood pressure 90/50 mmHg, heart rate 120 beats/min and weak, and she was anuric. Her right foot and the tibia were warm, red and swollen. A large, foul-smelling, neuro-ischemic ulcer with gross purulent discharge was seen on the posterior surface of her right heel ([Figure 8.38](#)). The calcaneus was exposed.

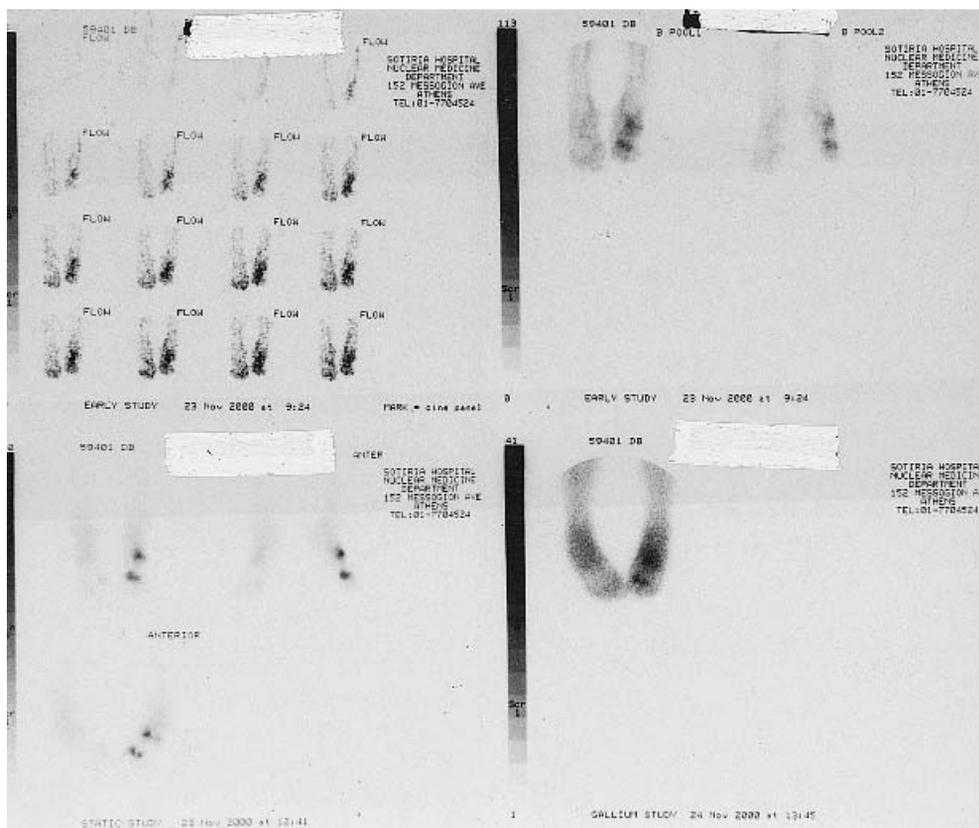


Figure 8.37 Increased radionuclide uptake by the tarsometatarsal bones, possibly due to osteomyelitis. Technetium-99m (^{99}Tc) phosphonates scan: *flow phase* (left upper panel); *blood pool phase* (right upper panel); *delayed phase* (left lower panel). Gallium-67 citrate study (right lower panel)

A plain radiograph showed a large skin defect on the posterioplantar aspect of her heel and bone resorption of the posterior calcaneus (Figure 8.39). Extensive calcinosis of the posterior tibial artery and medial plantar branch artery was also noted. After surgical debridement, bone and deep tissue cultures were obtained. Immediate support with i.v. fluids and antibiotic administration was commenced (ciprofloxacin 400 mg \times 3 and clindamycin 600 mg \times 3) and her situation improved within 12 h.

Tissue cultures revealed *Enterococcus* spp., *Acinetobacter baumannii*, and *Proteus mirabilis*. Based on an antibiogram, treatment was changed to ampicillin-sulbactam and continued for 2 weeks. Disarticulation through the ankle joint (Syme ankle disarticulation) was not feasible; a healthy heel flap and the heel pad is a prerequisite for this procedure so that the end of the stump is capable of bearing the patient's weight. Two weeks after her admission the patient sustained a below-knee amputation.



Figure 8.38 Large, neuro-ischemic ulcer with gross purulent discharge on the posterior surface of right heel. The calcaneus is exposed

Empirical treatment with antibiotics in severe foot infections should always include agents against staphylococci, enterobacteriaceae and anaerobes. In this case, two agents with good bone bioavailability were used since osteomyelitis was present. Therapeutic options in patients with severe foot infections include:

- Fluoroquinolone plus metronidazole or clindamycin. This combination is effective against *Staphylococcus aureus* (only methicillin-susceptible strains), enterobacteriaceae, and anaerobes.
- β -lactam and β -lactamase inhibitor combinations (ticarcilline–clavulanic acid, piperacillin–tazobactam). Ampicillin–sulbactam is particularly active against *Enterococcus* spp. For patients who have received extensive antibiotic therapy, ticarcilline–clavulanic acid or piperacillin–tazobactam may be preferred because of their increased activity against nosocomial gram-negative bacilli. Such regimens are also effective against



Figure 8.39 Plain radiograph of the foot illustrated in [Figure 8.38](#) showing a large skin defect on the posterio-plantar aspect of the heel and bone resorption of the posterior calcaneus. Calcification of the posterior tibial artery and medial plantar branch artery is also apparent

Staphylococcus aureus (only methicillin sodium-susceptible strains), *Streptococcus* spp. and most anaerobes.

- In patients who have severe penicillin allergy, combination therapy with aztreonam and clindamycin, or a fluoroquinolone and clindamycin is effective.
- Imipenem–cilastin or meropenem as monotherapy.

Doctors should always consider that:

- Modification of the treatment may be necessary according to the results of cultures.
- Vancomycin or teicoplanin are indicated in cases of infection with methicillin-resistant staphylococcal strains.

- Third generation cephalosporins should be used only in combination with other agents, as they have moderate anti-staphylococcal activity and lack significant activity against anaerobes.
- Aminoglycosides are nephrotoxic and they are inactivated in the acidic environment of the soft tissue infection and have poor penetration into bone.

Keywords: Osteomyelitis; heel ulceration; calcaneus; severe foot infection treatment; below-knee amputation

Chapter IX

NEURO-OSTEOARTHROPATHY. THE CHARCOT FOOT

- CLASSIFICATION
- ACUTE NEURO-OSTEOARTHROPATHY
- DIFFERENTIAL DIAGNOSIS BETWEEN ACUTE NEURO-OSTEOARTHROPATHY AND OSTEOMYELITIS
- PATTERNS OF NEURO-OSTEOARTHROPATHY
- NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERNS II AND III; DOUNIS TYPE II: INVOLVEMENT OF THE FIFTH METATARSAL HEAD
- NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERNS II AND III; DOUNIS TYPE II: PARTIAL RESORPTION OF LISFRANC'S JOINT
- ACUTE NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERN II; DOUNIS TYPE II
- NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERNS II AND III; DOUNIS TYPE II: FRAGMENTATION OF THE CUBOID BONE
- NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERNS II AND III; DOUNIS TYPE II: COLLAPSED PLANTAR ARCH
- NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERNS II AND III; DOUNIS TYPE II: MIDFOOT COLLAPSE

- NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERNS II AND III; DOUNIS TYPE II: ULCER OVER A BONY PROMINENCE
- ACUTE NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERN IV; DOUNIS TYPE IIIa
- NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERN IV; DOUNIS TYPE III (a, b, and c)
- NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERN IV; DOUNIS TYPE IIIa
- NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERNS IV AND V; DOUNIS TYPE III (a, b and c): INVOLVEMENT OF THE HINDFOOT
- NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERNS IV AND V; DOUNIS TYPE III (a, b and c)
- BIBLIOGRAPHY

CLASSIFICATION OF NEURO- OSTEOARTHROPATHY

Neuro-osteoarthropathy (Charcot arthropathy, Charcot osteoarthropathy, neuropathic osteoarthropathy) represents one of the most serious complications of diabetes. Its prevalence is between 1 and 7.5%; bilateral involvement has been reported to occur in 6–40% of patients in several series. The development of this complication depends on peripheral somatic and autonomic neuropathy, together with adequate blood supply to the foot. A minor trauma, often unrecognized by the patient, may initiate the process of joint and bone destruction. Some cases of neuro-osteoarthropathy have been reported after infection of the foot, surgery to the ipsilateral or the contralateral foot, or restoration of foot circulation. Mean age of presentation is approximately 60 years and the majority of the patients have diabetes of more than 15 years' duration. Men and women are affected equally.

CLASSIFICATION OF NEURO-OSTEOARTHROPATHY, BASED ON CHARACTERISTIC ANATOMIC PATTERNS OF BONE AND JOINT DESTRUCTION

Classification Proposed by Sanders and Frykberg (1991)

Pattern I: Forefoot (involvement of interphalangeal joints, phalanges, metatarsophalangeal joints, distal metatarsal bones). The frequency of this pattern is 26–67%, and it is often associated with ulceration over the metatarsal heads.

Pattern II: Tarsometatarsal joints. The frequency of this pattern is 15–48%; it often causes collapse of the midfoot and a rocker-bottom foot deformity.

Pattern III: Naviculocuneiform, talonavicular and calcaneocuboid joints. The frequency of this pattern is 32%; it often causes collapse of the midfoot and a rocker bottom foot deformity, particularly when it is combined with pattern II.

Pattern IV: Ankle and subtalar joints. Although this pattern accounts for only 3–10% of the cases of neuro-osteoarthropathy, it invariably causes severe structural deformity and functional instability of the ankle.

Pattern V: Calcaneus. Avulsion fracture of the posterior tubercle of the calcaneus. This pattern is not in fact neuro-osteoarthropathy, since no joint involvement occurs. This pattern is rare.

Classification Proposed by Dounis (1997)

According to the classification proposed by Dounis in 1997, there are three main types of neuro-osteoarthropathy (Figure 9.1):

Type I: This type is similar to pattern I as in the above classification proposed by Sanders and Frykberg, and involves the forefoot.

Type II: Type II involves the midfoot (tarsometatarsal, naviculocuneiform, talonavicular and calcaneocuboid joints); its main consequence is the collapse of the midfoot and development of rocker-bottom foot deformity.

Type III: Type III involves the rearfoot and is subclassified as:

IIIa (ankle joint): Main consequence is instability.

IIIb (subtalar joint): Main consequence is instability and development of varus deformity of the foot.

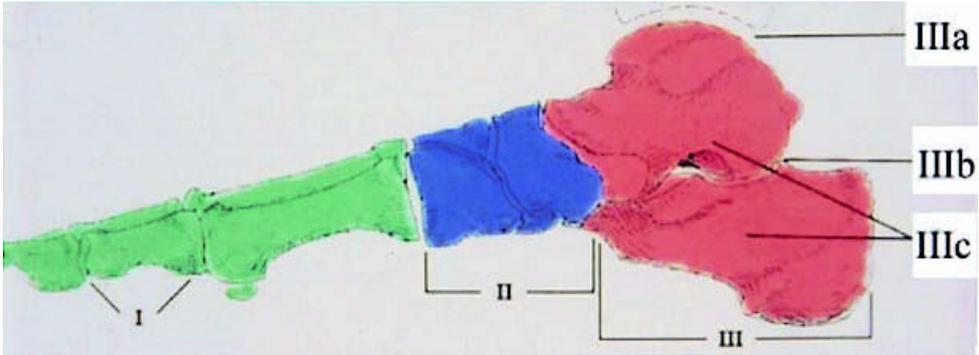


Figure 9.1 Dounis classification of neuro-osteoarthropathy. Refer to text

IIIc (resorption of talus and/or calcaneus): This type is associated with the inability to bear weight.

The *IIIc* subcategory is similar to pattern V as proposed by Sanders and Frykberg, but it includes some cases with resorption either of the talus or the calcaneus or both bones. The classification proposed by Dounis is less complex than that suggested by Sanders and Frykberg as it is based on the three anatomic areas of the foot.

Other classifications have been also described (Harris and Brand, 1966; Lennox, 1974; Horibe *et al.*, 1988; Barjon, 1993; Brodsky and Rouse, 1993; Johnson, 1995). Detailed descriptions of these classification systems can be found in the literature.

CLINICAL PRESENTATION AND LABORATORY FINDINGS

A typical clinical presentation is a patient with a swollen, warm and red foot with mild pain or discomfort. Usually there is a difference in skin temperature of more than 2°C compared to the unaffected foot. Most patients do not report any trauma, although some may recall a minor injury

such as a mild ankle sprain. On examination, pedal pulses are bounding and findings of peripheral neuropathy are constantly present. The white blood cell count is normal and the erythrocyte sedimentation rate may be slightly increased (20–40 mm/h).

RADIOLOGICAL FINDINGS

Radiological findings depend on the stage of the disease. Eichenholtz (1966) described three clinico-radiologically distinct stages. (a) The development stage, characterized by soft tissue swelling, hydrarthrosis, subluxations, cartilage debris (detritus), erosion of the cartilage and subchondral bone, diffuse osteopenia, thinning of the joint space and bone fragmentation. (b) The *coalescence* stage, characterized by evidence of restoration of the tissue damage: inflammation subsides, fine debris is absorbed, periosteal bone is formed, bone fragments fuse to the adjacent bones and the affected joints are stabilized. (c) The *reconstructive* stage, characterized by subchondral osteosclerosis, periarticular spurring, intra-articular and marginal exuberant osteophytes and ossification of ligaments and joint cartilage. Joint mobility is reduced and fusion and rounding of large bone fragments may be seen (Onvlee, 1998).

DIFFERENTIAL DIAGNOSIS

Diagnosis of acute neuro-osteoarthropathy requires a high level of vigilance for the disease. The acute development of foot swelling in a patient with long-standing diabetes and peripheral neuropathy is a clue to the presence of acute neuro-osteoarthropathy. In the early stages, plain radiographs may be normal and serial radiographic examination of the affected foot may be warranted. Acute infections (osteomyelitis, cellulitis) and crystal deposition disease should be excluded. Exclusion of osteomyelitis in such patients is not always easy. Scintigraphy studies and magnetic resonance imaging or computed tomography may not distinguish neuro-osteoarthropathy from osteomyelitis (Shaw and Boulton, 1995).

Keywords: Classification of neuro-osteoarthropathy; Charcot foot; Sanders and Frykberg classification; Dounis classification; clinical presentation of neuro-osteoarthropathy; radiological findings of neuro-osteoarthropathy; differential diagnosis of neuro-osteoarthropathy; Eichenholtz stage of neuro-osteoarthropathy

ACUTE NEURO-OSTEOARTHROPATHY: SANDERS AND FRYKBERG PATTERN I; DOUNIS TYPE I

A 56-year-old female patient with type 2 diabetes mellitus diagnosed at the age of 43 years and treated with sulfonylureas, was referred to the outpatient diabetic foot clinic for a forefoot ulcer and possible osteomyelitis. Diabetes control was acceptable (HBA_{1c}: 7.6%). She had background diabetic retinopathy and hypertension. On

examination the forefoot was red, swollen, warm and painful; she had severe peripheral neuropathy and a clear ulcer under her right fifth metatarsal head of 2 weeks' duration; peripheral pulses on both feet were normal. The patient denied any trauma. An anteroposterior radiograph showed osteolytic destruction of her third and fourth metatarsal heads, widening of the third metatarsophalangeal joints and subluxation of the second metatarsophalangeal joint (Figure 9.2). The white blood cell count (WBC) was within the normal range and the erythrocyte sedimentation rate (ESR) was 25 mm/h. The patient was diagnosed as a case of acute neuro-osteoarthropathy and, after debridement of the ulcer, a total-contact cast was fitted and bed rest was advised. She had her cast changed on a weekly basis for 1 month and every 2 weeks thereafter for two more months. The ulcer healed completely in 4 weeks and she had a good recovery. Plain radiographs followed 2 weeks later in order to exclude osteomyelitis, but no further bone destruction was seen.

This type of bone destruction is quite similar to that seen in osteomyelitis. However, in this patient osteomyelitis was less possible due to the short duration of the ulcer and lack of infection which must be present to cause extensive bone destruction. Bone destruction due to osteomyelitis takes at least 2 weeks to become visible on plain radiographs. Involvement of bones and joints is typical in acute neuro-osteoarthropathy. An increase in the ESR (greater than 70 mm/h) and WBC is a common feature of acute osteomyelitis. Mild elevation of the ESR (usually less than 40 mm/h) is common in acute neuro-osteoarthropathy.

Other roentgenographic findings in pattern I neuro-osteoarthropathy include concentric resorption of phalanges and



Figure 9.2 Radiograph of acute neuro-osteoarthropathy showing osteolytic destruction of the third and fourth metatarsal heads, widening of the third metatarsophalangeal joint and subluxation of the second metatarsophalangeal joint

broadening of the bases of proximal phalanges with formation of a cup around the metatarsal heads. Osteolytic destruction of the metatarsophalangeal joints with a

pencil-like tapering of the metatarsal shafts, epiphyseal absorption, thinning of the joint space and subluxation of the metatarsophalangeal and the phalangophalangeal joints,



Figure 9.3 Neuro-osteoarthropathy: concentric resorption of the phalanges of the three lesser toes, osteolytic destruction of the metatarsophalangeal joints and severe epiphyseal absorption are evident

may also be seen (Figure 9.3 exemplified by another patient). Pattern I-type neuro-osteoarthropathy is often complicated by plantar ulceration.

Keywords: Acute neuro-osteoarthropathy; plantar ulceration

DIFFERENTIAL DIAGNOSIS BETWEEN ACUTE TYPE I NEURO-OSTEOARTHROP- ATHY AND ACUTE OSTEOMYELITIS

A 62-year-old lady with type 2 diabetes diagnosed at the age of 48 years was



Figure 9.4 Right first ray amputation. Medial displacement and an ulcer on the tip of the second toe due to repeated trauma of the clawed toe can be seen

referred to the outpatient diabetic foot clinic for possible acute osteomyelitis of her right foot. The patient had had a first ray amputation on the right side due to osteomyelitis 2 years earlier. Eventually second and third claw toe deformity developed and a chronic ulcer formed at the tip of her right second toe due to repeated trauma (Figure 9.4). During the previous 6 months the patient had been the subject of several scintigraphic studies which suggested osteomyelitis of her right second and third metatarsals, she had therefore been treated with ciprofloxacin and clindamycin.

On examination, claw toe deformity was observed; the dorsum of her right fore-foot was red, swollen, painful and warm; she had severe peripheral neuropathy and bounding feet pulses. A clear non-infected



Figure 9.5 Radiograph of acute neuro-osteoarthropathy as shown in the patient whose foot is illustrated in Figure 9.4. Osteolytic destruction of the second and third metatarsal heads, widening of the third metatarsophalangeal joint and subluxation of the second metatarsophalangeal joint are evident

ulcer was seen at the tip of her second toe. A plain radiograph (Figure 9.5) showed disintegration of her right second and third metatarsal heads and an avulsion fracture between her second and third proximal phalanges. Her white blood cell count (WBC) was 14,500, the erythrocyte sedimentation rate (ESR) was 104 mm/h and the C-reactive protein level was 45 mg/dl. The patient's foot was immobilized by the use of a total-contact cast and she continued with antibiotics as the probability of osteomyelitis was high. She continued using the cast and the antibiotic treatment for 3 months. At that time the WBC was normal and the ESR and C-reactive protein levels were mildly elevated. One year later, a plain radiograph (Figure 9.6) revealed broadening of her second metatarsal head,

proliferative changes of her third metatarsal head and lateral exostosis of the proximal phalanx of her second toe. These findings correspond to the *reconstructive* stage in the evolution of neuro-osteoarthropathy.

Differential diagnosis in this case included osteomyelitis and acute neuro-osteoarthropathy. Scintigraphy and hematology studies suggested the presence of osteomyelitis. Radiographic findings are similar in both acute Charcot foot and osteomyelitis (see Figure 8.37 which shows scintigraphy studies of the same patient). It is also possible that both conditions co-existed for some time, as an acute infection may initiate acute neuro-osteoarthropathy. Whatever was the case, the patient had a good outcome and no further foot deformity developed.



Figure 9.6 X-ray showing the progression of neuro-osteoarthropathy in the patient whose foot is illustrated in Figures 9.4 and 9.5. This radiograph was taken 1 year after that shown in Figure 9.4. Broadening of the second metatarsal head, proliferative changes of the third metatarsal head and lateral exostosis of the proximal phalanx of the second toe are all evident

Keywords: Acute neuro-osteoarthropathy; type I neuro-osteoarthropathy; acute osteomyelitis

**NEURO-OSTEO-
ARTHROPATHY: SANDERS
AND FRYKBERG
PATTERNS II AND III;
DOUNIS TYPE II:
INVOLVEMENT
OF THE FIFTH
METATARSAL HEAD**

A 40-year-old male patient with type 1 diabetes diagnosed at the age of 18 years was

referred to the outpatient orthopedic department of our hospital for acute osteomyelitis in his left foot. The patient had fair diabetes control (HbA_{1c} : 7.2%) and background diabetic retinopathy.

On examination, redness, edema and warmth were noted on the dorsolateral aspect of his left foot (Figure 9.7), but no ulceration. A large ecchymosis was seen below the external malleolus, but the patient denied any trauma. He had diabetic neuropathy with severe loss of sensation of pain, light touch and temperature perception, but he could feel vibration. The vibration perception threshold was 10 V in both feet. The difference in temperature between the two feet was 3.5°C. Peripheral pulses

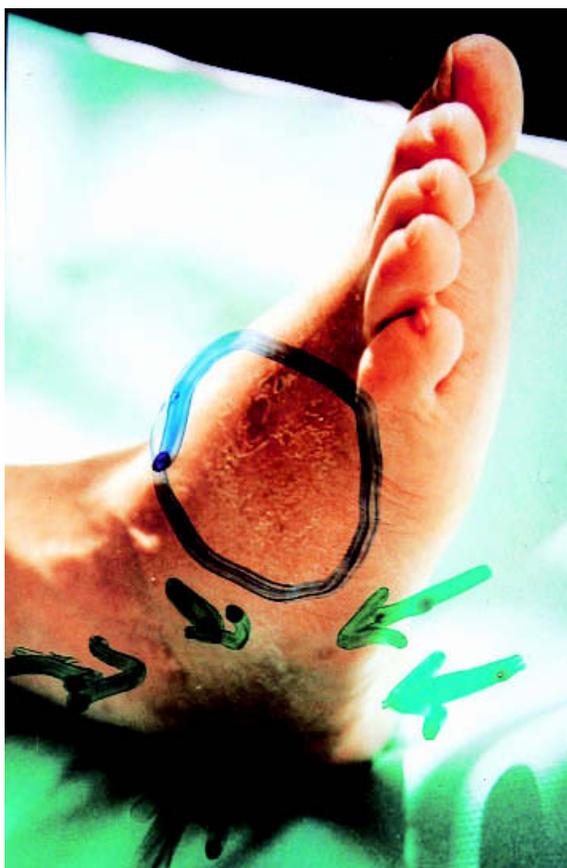


Figure 9.7 Redness and edema on the dorsolateral aspect of this foot is due to acute neuro-osteoarthropathy. A large ecchymosis below the external malleolus is due to an avulsion fracture of the base of the fifth metatarsal

were palpable and the ankle brachial index was 1.2 bilaterally.

An anteroposterior radiograph showed an avulsion fracture of the tubercle of his left fifth metatarsal base together with mild erosion of his left cuboid bone (Figure 9.8). Acute neuro-osteoarthropathy was diagnosed; a total-contact cast was applied and the patient was advised to limit his activity to a minimal level. He had the cast changed on a weekly basis initially and every 2 weeks thereafter for 3 months. He

had a good recovery with minimal foot deformity.

This patient had preserved function of the large myelinated fibers as evidenced by normal sensation of vibration as well as the vibration perception thresholds, and damage to the small nerve fibers responsible for sensations of pain, light touch and temperature. The pattern of nerve fiber damage may vary considerably in diabetes. The most common pattern is impairment of all nerve fibers; however, damage of the large myelinated fibers with preservation of the small unmyelinated fibers and vice versa may also be seen.

Pattern II (or Dounis' type II) is the commonest pattern of neuro-osteoarthropathy, characterized by involvement of the tarsometatarsal joints (Lisfranc's joint). Osteolytic destruction at this site may result in collapse of cuneiforms and/or cuboid bone and a rocker-bottom foot deformity. Ulcers may develop at the apex of collapsed bones.

The patient's ecchymosis was probably due to an avulsion fracture of the base of his fifth metatarsal bone.

Keywords: Pattern II neuro-osteoarthropathy; fracture; cuboid bone; fifth metatarsal



Figure 9.8 Radiograph of acute neuro-osteoarthropathy in the patient whose foot is shown in Figure 9.7. An avulsion fracture of the tubercle of the left fifth metatarsal base, together with mild erosion of the left cuboid bone can be seen

**NEURO-OSTEO-
ARTHROPATHY: SANDERS
AND FRYKBERG
PATTERNS II AND III;
DOUNIS TYPE II: PARTIAL
RESORPTION
OF LISFRANC'S JOINT**

A 38-year-old lady with type 1 diabetes diagnosed at the age of 19 years attended the outpatient diabetic foot clinic because of mild discomfort in her right midfoot.

Symptoms persisted for 1 week and the patient denied any trauma.

On examination, the right midfoot was red and swollen. She had claw toe deformity, and a small superficial neuropathic ulcer on the dorsum of the second right toe (Figure 9.9). Peripheral pulses were normal. Severe peripheral neuropathy was found with loss of sensation of light touch, pain, temperature and vibration. She could not feel 5.07 monofilaments. The vibration perception threshold was 40 V at the tip of the halluxes. A plain radiograph (Figure 9.10) revealed a partial disruption of the Lisfranc's joint with mild subluxation of the second metatarsal bone and mild lateral displacement of the last three metatarsals. Diagnosis of acute neuro-osteopathy was made; a total-contact cast was fitted and 3 months later a prefabricated walker was fitted and used for a further 6 months (Figure 9.11). The outcome

was good and no significant foot deformity developed.

Minimal second metatarsal dislocation may be easily overlooked in patients with mild symptoms. This type of neuro-osteopathy should be diagnosed and managed early as it invariably results in collapse of the midfoot. A minimal (of a few millimeters) lateral deviation or a fracture of the base of the second metatarsal may be an early sign of acute neuro-osteopathy. If the foot is not immobilized, dislocation of all metatarsals develops. Lateral displacement of the metatarsal bases on the cuneiform and cuboid bones occurs and eventually the midfoot collapses.

Prefabricated walkers are suitable alternatives to a total-contact cast, although they do not provide total contact. Application of inflatable pads improves contact. They are indicated in patients with impaired vascular circulation or in patients who require



Figure 9.9 Claw toe deformity and a small superficial neuropathic ulcer on the dorsum of the second right toe. No other apparent foot deformity is visible in this patient with early acute neuro-osteopathy

Figure 9.10 Radiograph of acute neuro-osteoarthropathy in the patient whose foot is shown in [Figure 9.9](#). Partial disruption of the Lisfranc's joint with mild subluxation of the second metatarsal bone and mild lateral displacement of the last three metatarsals can be seen



Figure 9.11 Prefabricated walker for the patient whose foot is illustrated in [Figures 9.9](#) and [9.10](#). Prefabricated walkers are suitable alternatives to the total-contact cast. As they do not provide total contact, inflatable pads are used to improve contact. They are indicated in patients with impaired vascular circulation or in patients who require frequent removal of the cast for the treatment of concurrent ulcers



frequent removal of the cast for treatment of concurrent ulcers.

Keywords: Second metatarsal subluxation; pattern II neuro-osteoarthropathy

**ACUTE NEURO-
OSTEOARTHROPATHY:
SANDERS AND FRYKBERG
PATTERN II; DOUNIS
TYPE II**

A 54-year-old male patient with long-standing type 1 diabetes was referred to the orthopedic department because of mild pain, foot swelling and deformity, which developed 2 weeks after a foot sprain. He had a history of an intermediate amputation of the fifth metatarsal bone due to osteomyelitis resulting from a perforated ulcer on his fifth metatarsal head and metaphysis, which had occurred 2 years earlier.

On examination he had bounding feet pulses and severe diabetic neuropathy; the

vibration perception threshold was above 50 V bilaterally. His left foot was painful, swollen and warm. A prominence on the dorsal aspect of his first metatarsophalangeal joint was visible and crepitus could be heard on passive foot flexion and extension. A radiograph revealed signs of acute neuro-osteoarthropathy involving the midfoot: osteolysis and fragmentation of the cuneiforms, tarsometatarsal joint involvement and dislocation of the first metatarsal joint (Figure 9.12). The patient was hospitalized and arthrodesis of the dislocated metatarsal bone was carried out by means of Steinmann pins (Figure 9.13). A non-weight-bearing total-contact cast was applied for 3 months, followed by 6 months in a weight-bearing total-contact cast. Two months later no major deformity was visible (Figure 9.14, immediately after cast removal).

As a rule, reconstructive surgery of neuro-osteoarthropathy is contraindicated in the acute dissolution phase (Eichenholtz stage I). The main reconstructive procedures carried out in patients with neuro-osteoarthropathy are osteotomy of a bony



Figure 9.12 Radiograph of acute neuro-osteoarthropathy involving the midfoot: osteolysis and fragmentation of the cuneiforms, tarsometatarsal joint involvement and dislocation of the first metatarsal joint are evident



Figure 9.13 Postoperative radiographs of the condition shown in [Figure 9.12](#); fusion of the tarsometatarsophalangeal joints (arthrodesis) with the placement of Steinmann pins can be seen



Figure 9.14 Postoperative photograph of the patient whose condition is illustrated in [Figures 9.12](#) and [9.13](#), 9 months after arthrodesis and use of a total-contact cast. No major foot deformity is seen

prominence and arthrodesis. When indicated, surgery is undertaken during the *reconstructive* stage (Eichenholtz stage III). During the acute phase surgery results in high rates of fixation failure, recurrent foot deformity and infection. Other contraindications to arthrodesis in such patients include soft tissue and bone infection, insufficient bone stock to achieve rigid fixation and non-compliance with postoperative regimens. However, in this patient arthrodesis in the acute phase was necessary, as major foot deformity and functional dysfunction was anticipated if the metatarsal head was left untreated. In addition, a prerequisite for successful arthrodesis is the presence of a sufficient bone stock for rigid fixation. If the operation had been postponed, this procedure might have been impossible as the cuneiforms and navicular bones were fragmented and collapsed. After the operation, patients with neuro-osteopathy need long-term immobilization, irrespective of stage. In general, the immobilization period following an arthrodesis is double that required by patients without neuropathy.

Keywords: Acute pattern II neuro-osteopathy; arthrodesis; reconstructive surgery; Steinmann pins

**NEURO-OSTEO-
ARTHROPATHY: SANDERS
AND FRYKBERG
PATTERNS II AND III;
DOUNIS TYPE II:
FRAGMENTATION
OF THE CUBOID BONE**

A 64-year-old female patient with long-standing type 2 diabetes attended the outpatient diabetic foot clinic because of redness, edema, swelling and mild pain on the dorsum of the right midfoot, which had been present for the previous 2 months. She did not report any trauma. The feet pulses were normal but she had severe diabetic neuropathy. A plain radiograph showed fragmentation of the cuboid bone, a pseudoarthrosis of an old fracture at the base of the fifth metatarsal and bone



Figure 9.15 Plain radiograph showing neuro-osteopathy. Fragmentation of the cuboid, a pseudoarthrosis of an old fracture at the base of the fifth metatarsal and bone fragments at the talonavicular joint dorsally can be seen

fragments in the talonavicular joint dorsally (Figure 9.15). Neuro-osteoarthropathy was diagnosed, and the foot was put into a total-contact cast for 8 weeks. A fibrous union was present despite the absence of radiographic signs of healing of the fractured fifth metatarsal. The patient wore high-arched custom-made shoes. No further bone destruction was found during the next 6 months.

Keywords: Neuro-osteoarthropathy; patterns II and III; type II; cuboid fragmentation

**NEURO-OSTEO-
ARTHROPATHY: SANDERS
AND FRYKBERG
PATTERNS II AND III;
DOUNIS TYPE II: COL-
LAPSED PLANTAR ARCH**

A 56-year-old insulin-treated female patient with type 2 diabetes since the age of 45 years attended the outpatient diabetic foot clinic. She had background retinopathy—treated with laser—hypertension,

diabetic nephropathy and dyslipidemia, as well as a left foot deformity which she had had since the age of 52 years. She was a heavy smoker. She had an ulcer on the outer aspect of her left foot which had developed when she was 53 years old and had started as a bulla following a trauma caused by her shoe.

On examination, a collapsed left mid-foot and claw toe deformity were present. She had a painful irregular ulcer on the lateral aspect of her left foot with local edema, erythema and purulent discharge (Figure 9.16). A hemorrhagic callus was noted on the dorsum of her fifth left toe. Peripheral pulses were weak. The vibration perception threshold was over 50 V and she had reduced sensation of pain, light touch and temperature. Achilles tendon reflexes were absent.

Triplex ultrasonography of her leg arteries showed severe obstruction of her common femoral arteries on both sides. A methicillin-resistant *Staphylococcus aureus* was isolated from the base of the ulcer and the patient was treated with intramuscular teicoplanin and ciprofloxacin. The



Figure 9.16 Chronic neuro-osteoarthropathy with collapsed plantar arch and claw toe deformity. An irregular neuro-ischemic ulcer is present over the lateral aspect of the fifth metatarsal head with local edema, erythema and a sloughy bed superimposing osteomyelitis. A hemorrhagic callus is seen on the dorsum of the fifth left toe

patient was referred to the Vascular Surgery Department for bypass surgery. Appropriate footwear was prescribed and the ulcer healed in 3 months.

A plain radiograph revealed cuneiform fragmentation and disruption of the tarsometatarsal joint (Lisfranc's joint). Osteomyelitis of the head of the fifth metatarsal was also present (Figure 9.17). Calcification of the digital arteries was also noted, a common finding in patients with diabetes.

Adequate blood supply is a prerequisite for neuro-osteopathy. In this patient,



Figure 9.17 Plain radiograph showing chronic neuro-osteopathy in the patient whose foot is illustrated in Figure 9.16. Fragmentation of the cuneiforms and disruption at the tarsometatarsal joint (Lisfranc's joint) with osteomyelitis of the head of the fifth metatarsal and calcification of the digital arteries, a common finding in patients with diabetes, are all evident

peripheral vascular disease occurred after neuro-osteopathy.

Keywords: Neuro-ischemic ulcer; neuro-osteopathy

**NEURO-
OSTEOARTHROPATHY:
SANDERS AND FRYKBERG
PATTERNS II AND III;
DOUNIS TYPE II: MIDFOOT
COLLAPSE**

A lateral radiograph shows midfoot collapse (collapse of naviculocuneiform, talonavicular and calcaneocuboid joints) due to chronic neuro-osteopathy of combined patterns II and III (Figure 9.18). In addition, localized bone resorption at the naviculocuneiform joints can be seen.

Osteotomy of protruding bone is recommended if recurrent ulceration occurs despite the use of custom-made shoes and insoles.

Keywords: Midfoot collapse; neuro-osteopathy

**NEURO-
OSTEOARTHROPATHY:
SANDERS AND FRYKBERG
PATTERNS II AND III;
DOUNIS TYPE II: ULCER
OVER A BONY
PROMINENCE**

A 64-year-old insulin treated male patient with type 2 diabetes diagnosed at the age of 46 years and acceptable diabetes control (HBA_{1c}: 7.4%), was referred to the outpatient diabetic foot clinic for a chronic plantar ulcer on his left midfoot. The

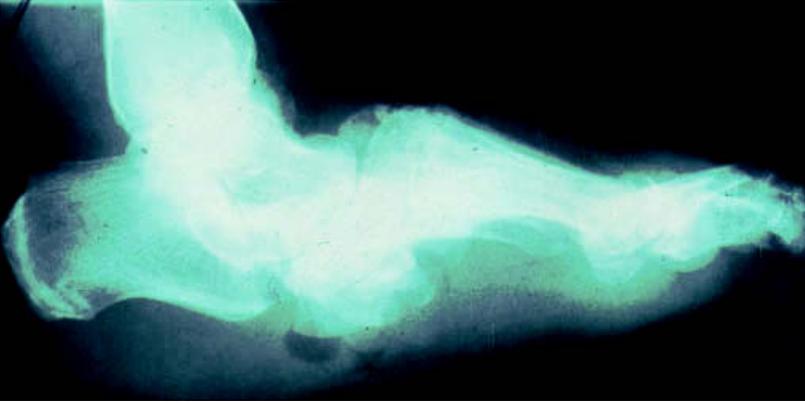


Figure 9.18 Plain radiograph of a midfoot collapse (collapse of naviculocuneiform, talonavicular and calcaneocuboid joints) due to chronic neuro-osteoarthropathy. Localized bone resorption at the naviculocuneiform joints is also seen



Figure 9.19 Chronic neuro-osteoarthropathy with collapsed midfoot, hallux valgus deformity and amputation of the second toe. Gross callus formation is seen at the borders of the neuropathic ulcer. Healthy granulating tissue can be seen on its bed

patient had background retinopathy, hypertension and diabetic nephropathy (proteinuria of 1.5 g/24 h).

One year earlier he had been hospitalized for almost 3 months because the ulcer was complicated by a severe deep tissue infection. During hospitalization he had extensive surgical debridement and was treated with intravenous antibiotics. A below-knee amputation was suggested, but the patient did not consent and he sustained a second toe amputation instead, due to osteomyelitis. The patient wore his usual shoes and refused the total-contact cast which had been suggested.

On examination, he had severe peripheral neuropathy (the vibration perception threshold was above 50 V bilaterally) and bounding peripheral pulses. He had chronic neuro-osteoarthropathy of his left foot. Hallux valgus deformity and a collapsed mid-foot were observed. Bony prominences could be palpated at the base of the neuropathic ulcer (Figure 9.19).

An anteroposterior radiograph showed collapsed cuboid and navicular bones (Figure 9.19), extensive destruction and

resorption of the cuneiforms with osteosclerotic changes and complete destruction of the tarsometatarsal, naviculocuneiform and talonavicular joints (Figures 9.20–9.22). Extensive resorption of the metatarsal diaphyses with a pencil-like appearance of the fifth metatarsal could be seen (Figure 9.21).

Debridement of the ulcer was carried out and the patient was advised to rest at home. Custom-made shoes were prescribed in order to offload the pressure from the ulcerated area and to accommodate the deformity. The ulcer healed in 12 weeks (Figure 9.23). Within the next 2 years the patient suffered two relapses of the foot ulcer at the same site.

In this case, chronic neuro-osteoarthropathy involves the Chopart's joint (talonavicular and/or calcaneocuboid) or naviculocuneiform joints, as well as Lisfranc's joint and, if left untreated, results in collapse of the midfoot and a rocker-bottom foot deformity. Recurrent foot ulceration at the apex of the collapsed bones is a common complication.

Keywords: Chronic neuro-osteoarthropathy; neuropathic ulcer



Figure 9.20 Plain radiograph of chronic neuro-osteoarthropathy in the patient whose foot is shown in Figure 9.19. Collapsed cuboid and navicular bones, extensive destruction and resorption of the cuneiforms and destruction of the talonavicular joint are evident



Figure 9.21 Plain radiograph of chronic neuro-osteoarthropathy in the patient whose foot is illustrated in [Figures 9.19](#) and [9.20](#). Osteosclerotic changes and complete destruction of the tarsometatarsal and naviculocuneiform joints can be seen in addition to the pencil-like appearance of the fifth metatarsal



Figure 9.22 Plain radiograph of chronic neuro-osteoarthropathy in the patient whose foot is shown in [Figures 9.19–9.21](#). Osteosclerotic changes and complete destruction of the tarsometatarsal joints together with bone resorption of the metatarsal shafts and osteophyte formation can be seen

**ACUTE NEURO-
OSTEOARTHROPATHY:
SANDERS AND FRYKBERG
PATTERN IV; DOUNIS
TYPE IIIa**

A 41-year-old male patient with type 1 diabetes diagnosed at the age of 19 years

was referred to the orthopedic department of the hospital because of erythema and swelling of his right ankle, the onset of which had occurred rapidly some days earlier. No history of trauma was reported.

On examination, he had severe peripheral neuropathy and normal feet pulses. His right ankle was red, warm and swollen ([Figure 9.24](#)). A radiograph showed erosion



Figure 9.23 Healed ulcer in the patient whose foot is shown in [Figures 9.19–9.22](#). Recurrent ulceration of the midsole in a patient with midfoot collapse is an indication of osteotomy in the protruding bones



Figure 9.24 Clinical presentation of acute neuro-osteoarthritis of the right ankle which is red, warm and swollen

of the articular surfaces of the right tibia and talus. Bone fragments protruded medially (Figure 9.25). A diagnosis of acute neuro-osteoarthropathy was made and the patient was advised to rest, with his right foot in a total-contact cast. The cast was changed fortnightly for the first month and monthly for the next year. After this time osteoarthritic changes remained only in the affected joint and no major deformity was sustained.

Neuro-osteoarthropathy in the ankle is the third most common pattern of this

condition (frequency of 13%) and may result in severe structural deformity and instability. An extensive period of immobilization is required in order to prevent deformities.

Keywords: Acute neuro-osteoarthropathy

**NEURO-
OSTEOARTHROPATHY:
SANDERS AND FRYKBERG
PATTERN IV; DOUNIS
TYPE III (a, b, and c)**



Figure 9.25 Plain radiograph of chronic neuro-osteoarthropathy of the right ankle and foot as illustrated in Figure 9.24. There is erosion of the articular surfaces of the right tibia and talus and bone fragments protruding medially

A 67-year-old patient with type 2 diabetes diagnosed at the age of 41 years attended the outpatient orthopedic clinic because of worsening painful ankle swelling after a strain in his right ankle 2 weeks previously. He had severe peripheral neuropathy and normal feet pulses.

A plain film showed resorption of the distal parts of the tibial and peroneal bones and involvement of the ankle joint (Figure 9.26). Pattern IV neuro-osteoarthropathy was diagnosed and the foot was placed in a total-contact cast and bed rest was advised. The patient did not comply with the advice and continued to be active while wearing the cast. One month later extensive resorption and fragmentation of the talus and resorption of the distal areas of the tibia and fibula was observed on a second radiograph. A bone fragment protruded posteriorly (Figure 9.27). Six months later a plain film showed extensive resorption of the talus, subchondral osteosclerosis of the tibia and calcaneus and extensive ligament ossification (the *reconstructive* stage of neuro-osteoarthropathy). Bone fragments protruded laterally (Figure 9.28). The patient admitted that during this time he had been active. He had significant



Figure 9.26 Plain radiograph showing acute neuro-osteoarthropathy. Resorption of the distal areas of the tibia and fibula and involvement of the ankle joint are evident



Figure 9.27 Plain radiograph showing progress of neuro-osteoarthropathy 1 month after the X-ray shown in [Figure 9.25](#) was taken. There is extensive resorption and fragmentation of the talus and resorption of distal areas of the tibia and fibula and a bone fragment protrudes posteriorly

instability and varus foot deformity. Eventually the patient sustained a below-knee amputation.

A major problem in this pattern of neuro-osteoarthropathy is functional instability and foot deformity. Reconstructive



Figure 9.28 Plain radiograph showing progress of neuro-osteoarthropathy 6 months after the X-ray shown in [Figure 9.27](#) was taken. There is extensive resorption of the talus, subchondral osteosclerosis of the tibia and calcaneus and extensive ligament ossification. Bone fragments can be seen to protrude laterally

procedures (such as arthrodesis) were not possible due to extensive bone absorption. With this type of articular destruction rehabilitation will be more successful if the patient uses a below-knee prosthesis rather than a patellar-tibial-bearing orthosis.

Keywords: Ankle neuro-osteoarthropathy; talus resorption; reconstructive stage

**NEURO-
OSTEOARTHROPATHY:
SANDERS AND FRYKBERG
PATTERN IV; DOUNIS
TYPE IIIa**

A type 2 diabetic female patient with bilateral chronic neuro-osteoarthropathy (in the

reconstructive stage) resulting in marked bilateral varus foot deformity (Figures 9.29 and 9.30), attended the outpatient orthopedic clinic. She was unable to walk without crutches due to significant instability. On a plain radiograph complete destruction of the ankle joint and subchondral osteosclerosis at the distal ends of the tibia and fibula were seen, together with lateral resorption of the talus. Bone fragments were observed laterally in the ankle joint as were medial exuberant osteophytes (Figure 9.31). The patient underwent a realignment arthrodesis of the ankle joint by lateral ankle incisions and the ankle joint was fixed with a Huckstep nail (Figure 9.32). The postoperative results were excellent (Figure 9.33).

Significant deformity and instability is the main indication for arthrodesis in



Figure 9.29 Bilateral varus deformity of the feet due to chronic neuro-osteoarthropathy. Significant instability resulted in the patient's inability to walk without crutches



Figure 9.30 Lateral view of [Figure 9.29](#)



Figure 9.31 Plain radiograph of neuro-osteoarthropathy of the right foot of the patient whose feet are shown in [Figures 9.29](#) and [9.30](#). There is complete destruction of the ankle joint, subchondral osteosclerosis in the distal areas of the tibia and fibula, together with lateral resorption of talus. Bone fragments are seen laterally in the ankle joint and exuberant osteophytes medially



Figure 9.32 Plain postoperative radiograph of the right foot of the patient whose feet are illustrated in [Figures 9.29–9.31](#). Arthrodesis of the ankle joint with the use of a Huckstep nail has been carried out



Figure 9.33 Postoperative photograph of the right foot of the patient whose feet are shown in [Figures 9.29–9.32](#) after successful arthrodesis of the ankle joint

patients with neuro-osteoarthropathy. In experienced hands it is possible in almost 80% of cases to achieve the goal of a stable and shoeable foot after an arthrodesis in patients with neuro-osteoarthropathy. The use of modern techniques of internal fixation has significantly improved prognosis in these patients. The period of immobilization after an arthrodesis in patients with neuro-osteoarthropathy is prolonged, usually more than 4 months.

Keywords: Neuro-osteoarthropathy; arthrodesis; Huckstep nail

**NEURO-OSTEO-
ARTHROPATHY: SANDERS
AND FRYKBERG PATTERNS
IV AND V; DOUNIS TYPE III
(a, b and c): INVOLVE-
MENT OF THE HINDFOOT**

Chronic neuro-osteoarthropathy often leads to extensive resorption of the hindfoot (talus and calcaneus), navicular and cuboid bones ([Figure 9.34](#)). The patient whose



Figure 9.34 Plain radiograph showing chronic neuro-osteopathy. Extensive resorption of the hindfoot (talus and calcaneus), navicular and cuboid bones is evident

X-ray is shown in [Figure 9.34](#) is a 45-year-old female with long-standing type 1 diabetes who developed this complication after a severe ankle sprain. She suffered complete loss of sensation in her feet and symptomatic autonomic neuropathy

(gastroparesis, diabetic diarrhea and orthostatic hypotension). Gait instability developed within 8 months, to the point where the patient was unable to walk without crutches. Although she used a total-contact cast, bone resorption was rapid and



Figure 9.35 Plain radiograph showing extensive resorption of most of the talus and calcaneus and of the distal end of the tibia-fibula in a patient with chronic neuro-osteopathy. Osteolysis in the lower part of the calcaneus is due to osteomyelitis following a perforated ulcer

relentless, so that eventually the patient succumbed to a below-knee amputation.

Keywords: Chronic neuro-osteoarthropathy

**NEURO-
OSTEOARTHROPATHY:
SANDERS AND FRYKBERG
PATTERNS IV AND V;
DOUNIS TYPE III (a, b and c)**

Figure 9.35 shows extensive resorption of most of the talus and calcaneus, in addition to the distal end of the tibia–fibula in a patient with neuro-osteoarthropathy. The osteolysis in the lower part of the calcaneus is due to osteomyelitis. A chronic



Figure 9.36 Chronic neuro-osteoarthropathy. The osteomyelitis in the heel has been superimposed with a deep neuropathic ulcer in the patient whose X-rays are illustrated in Figure 9.35

neuropathic heel ulcer is present, caused by a foreign body (Figure 9.36). Eventually the patient, who had long-standing diabetes and severe diabetic neuropathy, sustained a below-knee amputation.

Keywords: neuro-osteoarthropathy; heel ulcer; osteomyelitis

BIBLIOGRAPHY

1. Sanders LJ, Frykberg RG. Diabetic neuropathic osteoarthropathy: the Charcot foot. In Frykberg RG (Ed.), *The High Risk Foot in Diabetes Mellitus*. New York: Churchill Livingstone, 1991.
2. Dounis E. Charcot neuropathic osteoarthropathy of the foot. *Acta Orthopaed Hellenica* 1997; **48**: 281–295.
3. Harris JR, Brand PW. Patterns of disintegration of the tarsus in the anaesthetic foot. *J Bone Joint Surg* 1966; **5**: 95–97.
4. Lennox WM. Surgical treatment of chronic deformities of the anaesthetic foot. In McDowell F, Enna CD (Eds), *Surgical Rehabilitation in Leprosy, and in Other Peripheral Nerve Disorders*. Baltimore: Williams and Wilkins, 1974; 350–372.
5. Horibe S, Tada K, Nagano J. Neuroarthropathy of the foot in leprosy. *J Bone Joint Surg (Br)* 1988; **70-B**: 481–485.
6. Brodsky JW, Rouse AM. Exostectomy for symptomatic bony prominences in diabetic Charcot foot. *Clin Orthop* 1993; **296**: 21–26.
7. Barjon MC. Les ostéoarthropathies destructrices du pied diabétique. In Hérisson C, Simon L (Eds), *Le Pied Diabétique*. Paris: Masson, 1993; 77–91.
8. Johnson JE. Neuropathic (Charcot) arthropathy of the foot and ankle. AAOS 1995 Instructional course #349. Handoutcover.
9. Eichenholtz SN. *Charcot Joints*. Springfield, IL: Charles C Thomas, 1966.
10. Onvlee GJ. The Charcot foot. A critical review and an observational study of 60 patients. Thesis, Universiteit van Amsterdam, 1998.
11. Shaw JE, Boulton AJM. The Charcot foot. *Foot* 1995; **5**: 65–70.

Appendix 1

ANATOMY OF THE FOOT

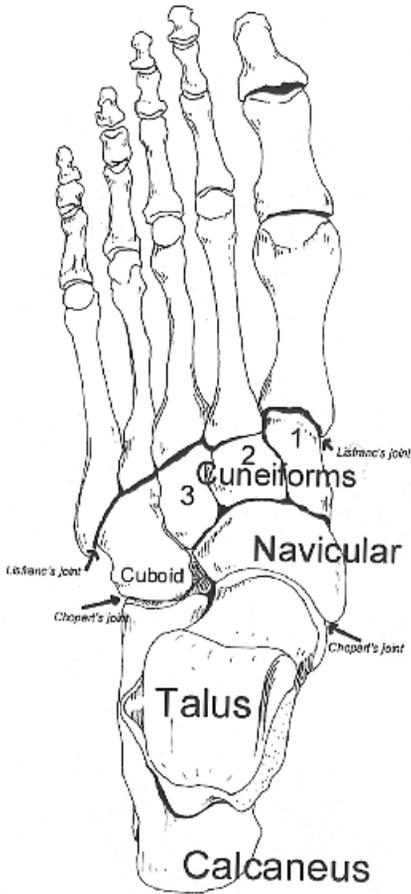


Figure A1 Dorsal aspect of the bones in the foot

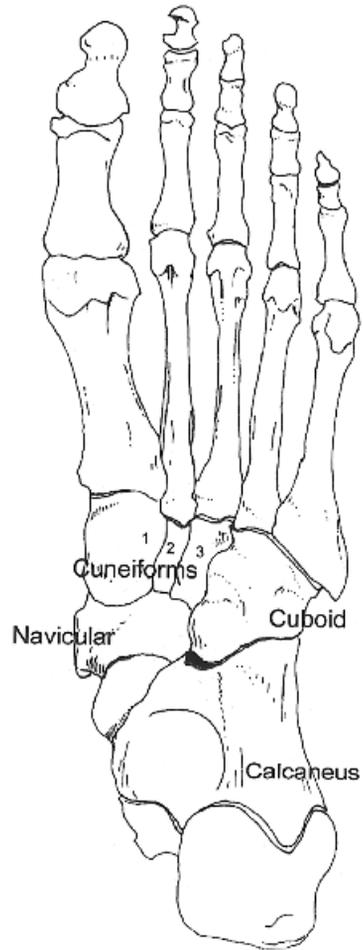


Figure A2 Plantar aspects of the bones in the foot

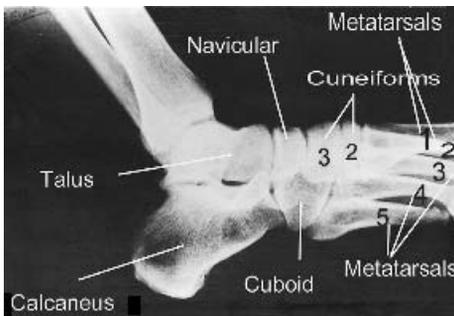


Figure A3 Plain radiograph of the foot shown in lateral view

Appendix 2

MANUFACTURERS OF PREVENTIVE AND THERAPEUTIC FOOTWEAR

The therapeutic and preventive footwear and insoles described in this book are products of various companies including:

Acor Orthopedic, USA
Aircast, Inc., USA
AliMed, Inc., USA
Buratto Advanced Technology, Italy
Darco International, Inc., USA
F. W. Kraemer KG, Germany
Orthopaedic Systems, UK

Index

Note: page numbers in *italics* refer to figures and tables

- abscess
 - drainage 146
 - plantar 133
- Achilles tendon reflexes 5
- Acinetobacter baumannii* 181
- amputation
 - arteriography prior to 20
 - below-knee 129, 140, 147
 - chronic neuro-osteoarthropathy 212
 - deep-tissue infection 163, 166
 - osteomyelitis of heel 181
 - biomechanics of foot 69
 - Charcot foot 95
 - Chopart disarticulation 68–9
 - foot deformities 31
 - hallux 109, 110, 169
 - heel ulcers 98
 - limited distal 127
 - prevalence 3
 - ray 140, 178
 - fifth 178
 - first 61–3, 176
 - fourth 70
 - risk 3
 - toe 140, 146
 - fifth 121, 122
 - fourth 134
 - second 203
 - wet gangrene 143, 146–7
- angiography 29
 - digital subtraction 132, 134, 136, 137–8
 - stenosis location 135, 140, 141
- angioplasty
 - suboptimal 135
 - see also* percutaneous transluminal angioplasty
- anhidrosis 3
- ankle
 - arthrodesis 208, 210
 - bone fragments 208, 209
 - disarticulation 181
 - edema 53, 110, 112
 - deep-tissue infection 162, 163
 - neuro-osteoarthropathy 204, 205, 206, 207
 - osteophytes 208, 209
 - pressure 8, 20
 - swelling 204, 205, 206
- ankle brachial index
 - calculation 20
 - cardiovascular risk 18
 - foot ulcer classification 26
 - ischemic ulcers 28
 - monitoring 18, 20
 - neuropathic ulcer 28
 - peripheral vascular disease detection 16
- antibiotics 154
 - bone bioavailability 182
 - broad-spectrum 154
 - intravenous 154
 - osteomyelitis of heel 182–3
 - resistance 154
- antifungal drugs 160
- aorta, abdominal, stenosis 138
- aorto—femoral bypass graft 127
- aorto—popliteal bypass graft 150
- Apligraf® *see* Graftskin
- arterial calcification 9, 10
- arterial insufficiency, antibiotic therapy 154
- arterial stenosis 11
 - criteria in spectral analysis 13
 - ultrasonography 12
 - see also* named arteries
- arteriography 20
- arthrodesis, realignment 208, 210

- aspirin 16
- autolytic debridement 119
- becaplermin *see* platelet-derived growth factor β (PDGF- β)
- bedridden patients 97–8
- biosurgery 119, 121
- biothesiometer 6
- blisters
 - rupture 146, 167
 - soft tissue infection 156, 157
- blood pressure control 16
- bone demineralization 92
- bone scintigraphy imaging 175, 178–9, 181
 - phases 179, 181
- bony prominence
 - osteotomy 197, 199
 - ulcer 201, 202, 203, 204–5
- bunion 48–9
 - chronic neuropathic ulcer 177
 - ulcers 49
- bunionette formation 43–4, 46
 - ulceration 46
 - varus deformity 48
- burns
 - forefoot 98, 99, 100, 101, 102
 - squamous cell carcinoma in scars 81
 - toes 98, 99, 100, 101, 102
- bursitis 48–9
- bypass grafting 127, 201
 - neuro-ischemic ulcers 113
 - wet gangrene 147
- calcaneocuboid joint
 - Chopart dislocation 69
 - collapse 201, 202
 - neuro-osteoarthropathy 203
- calcaneus
 - bone resorption 183
 - exposure 180
 - osteolysis 211, 212
 - resorption 210, 211, 212
 - subchondral osteosclerosis 206, 207
- calcium pyrophosphate dihydrate (CPPD)
 - deposition disease 77–8
- calf support device 129, 131
- callus formation 3
 - under bone prominence 63–4
 - claw toe 155
 - hemorrhagic 65–6, 67, 68, 88, 200
 - metatarsal heads 45, 50–1
 - neuropathic ulcers 89, 94
 - prominent 64–5
 - second toe removal 59, 60, 61, 62
 - plantar pressure 88
 - pressure loading 87–8
 - prevention 67
 - removal 65
 - ulcer under 66–7
- Candida albicans* 71
 - fungal infection with multimicrobial colonization 160
 - wound colonization 121
- cardiovascular risk factors 16
- cast, total-contact 34, 195–7, 198, 200, 206, 211
 - contraindications 34
- cellulitis
 - deep-tissue infection 166
 - infected foot ulcer 153
 - infected plantar ulcer with osteomyelitis 172
 - neuro-ischemic ulcers 114, 115
 - non-ulcerated skin 154
 - treatment 154
 - wet gangrene 146
 - wound infections 153
- Charcot foot 27
 - amputation 95
 - with neuropathic ulcer and deep-tissue infection 163–4, 165, 166, 167
 - radiography 192
 - ulcers 95–6
- Chopart dislocation, bilateral 68–9
- Chopart's joint 203
- claw toe 30, 31, 46–8
 - bunionette formation 46
 - callus formation 155
 - under bone prominence 64
 - hemorrhagic 65–6
 - convex triangular foot 51
 - fungal infection 159
 - hallux valgus with overriding toes 52–4, 56
 - with heloma durum 48–9
 - infection
 - under callus at tip 155
 - soft-tissue 156
 - muscle atrophy 103
 - neuro-ischemic ulcers 120, 121
 - neuro-osteoarthropathy 191, 195
 - neuropathic ulcers of metatarsal heads 93–4
 - onychodystrophy 155
 - pes cavus 45
 - phlegmon 169
 - plantar arch collapse 200
 - prominent metatarsal heads 58–9

- second 99
- ulcers 58, 59, 93–4, 120, 121
 - under callus area 66–7
 - wet gangrene 150
- cocked-up toes *see* claw toe
- coeliac aortic bifurcation stenosis 134, 137
- collagen bundles, hyalinized 80, 81
- collagenase 119
- collateral circulation 8, 15, 19, 122
 - development 138, 150
 - popliteal artery 132, 135
- collateral vessel development 142
- compliance with medical instructions 3
- computed tomography (CT)
 - osteomyelitis diagnosis 175
 - phlegmon imaging 171
 - spiral 14
- convex triangular foot 51–2
- corns 49–50
 - see also* heloma durum
- critical leg ischemia 127
- cuboid bone
 - collapsed 203
 - fragmentation 199–200
 - resorption 210, 211
- cuneiform bone
 - fragmentation 201
 - osteolysis 197
 - resorption 203
- curly toe deformity 48
 - hemorrhagic callus formation 65–6
- debridement 119, 121
- depth—ischemia classification 26
- Dermagraf® 38
- dermatofibrosarcoma protruberans 82–3
- dermis, bioengineered 38
- dextranomers 119
- diabetes 3
- diabetic bullae 157
- diabetic neuropathy 3–6
 - definition 4
 - painful—painless foot 90
 - peripheral vascular disease co-existence 7
- digital arteries
 - calcification 201
 - thrombosis 158
- digital subtraction angiography *see* angiography, digital subtraction
- dorsalis pedis palpation 18
- dressings 36, 37–8
- ecchymosis 193, 194
- eczema, hyperkeratotic 78, 79
- edema
 - ankle 53, 110, 112
 - deep-tissue infection 162, 163
 - foot 158
 - forefoot deep-tissue infection 162, 163
 - neuro-osteoarthropathy 193
 - wet gangrene 140
- Enterobacter* 103, 104
 - fungal infection with multimicrobial colonization 160
- Enterobacter cloacae* 168
- Enterococci 156, 178, 181
- enzymatic debridement 119
- Epidermophyton floccosum* 158, 160
- Escherichia coli* 62, 98
 - deep-tissue after interphalangeal mycosis 161
 - infected ulcers 154
 - osteomyelitis 104, 171
 - web space infection 158
 - wet gangrene 146
- femoral artery
 - bruits 18
 - obstruction 122, 123
 - stenosis 7, 132, 134, 138
- femoral artery, common
 - atheromatous disease 114, 146
 - obstruction 200
 - peak systolic velocity 148, 150
 - stenosis 17, 148, 150
- femoral artery, superficial 19
 - atheromatous disease 139, 144, 146
 - atherosclerosis 117
 - spectral waveform 13, 16
 - stenosis 7, 10, 15, 17, 140, 141
 - collateral vessel development 142
 - dry gangrene 127
 - neuro-ischemic ulcers 114
 - stents 135
 - wet gangrene 150
 - stents 135, 141
- femoro—popliteal bypass graft 117, 127, 140
 - neuro-osteoarthropathy 95
- femoro—tibial bypass graft 123
- fibroblasts 39
- fibula
 - resorption 206, 207, 211, 212
 - subchondral osteosclerosis 208, 209
- flat foot *see* pes planus
- Fontaine clinical staging 7–8
 - neuro-ischemic ulcers 116

- foot
- anatomy 215
 - see also* forefoot; heel; hindfoot; midfoot; sole of foot; toe(s)
- foot care, patient education 31–2
- foot deformities 3
- definition 31
 - see also* pes cavus; pes planovalgus; pes planus; varus deformity
- foot ischemia, hyperbaric oxygen 38
- foot pulses
- palpation 7
 - peripheral 28
- footwear, preventive 29, 31, 111, 219
- commercially-available 120
 - prescription 117
 - pressure relief shoes 35–6
 - refusal to wear 116
 - see also* shoes
- forefoot
- burns 98, 99, 100, 101, 102
 - edema with deep-tissue infection 162, 163
 - neuro-ischemic ulcers with osteomyelitis 114, 116–17
- foreign objects
- neuropathic heel ulcer 212
 - in shoes 156, 157
- fractures
- avulsion of metatarsal base 193
 - see also* stress fractures
- fungal infection 158
- claw toe 159
 - interphalangeal deep-tissue infection 161, 162
 - with multimicrobial colonization 160
 - palmoplantar keratoderma 76–7
 - web space 158
 - see also* onychomycosis
- gait instability 211
- gallium-67 citrate 179, 181
- gangrene 7–8
- bedridden patients 97–8
 - critical limb ischemia 18
 - diabetic 133
 - dry
 - heel 129–30
 - with ischemic necrosis of skin 129
 - patient education 143
 - toes 127, 128, 130, 131, 133–4, 136–8
 - infection 127
 - ischemic ulcers 29
 - necrosis 127, 128, 139
 - transcutaneous oxygen pressure 10
- wet 127
- amputation 143, 146–7
 - blistering 146
 - cellulitis 146
 - Escherichia coli* 146
 - extensive of foot 143–4, 146–7
 - foot 140, 144
 - hallux 147, 148, 149–50
 - leading to mid-tarsal disarticulation 142–3, 145
 - necrotic tissue debridement 146
 - sepsis 130, 132–3, 134, 135, 136
 - Staphylococcus aureus* 132, 146
 - toes 139–40, 144
 - wound infections 153
- Graftskin 38–9
- granulocyte colony stimulating factor (G-CSF) 39
- hallux
- amputation 109, 110, 169
 - disarticulation 63, 95, 99, 102, 109, 110
 - hemorrhagic callus 55
 - infection 103
 - ingrown nails 146
 - neuro-ischemic ulcers 107, 108
 - osteomyelitis 107–9, 110, 117–21
 - osteomyelitis 168–9, 176, 178
 - neuro-ischemic ulcers 107–9, 110, 117–21
 - removal 63
 - subungual hematoma 110
 - ulcer under 67, 68
 - wet gangrene 147, 148, 149–50
- hallux valgus 31, 45
- after second toe removal 59, 60, 61, 62
 - bunionette formation 46
 - callus over prominent metatarsal heads 65
 - collapsed midfoot 202, 203
 - hammer toes 57
 - onychomycosis 54–6, 57
 - with overriding toe 50–1, 52–4, 56, 57
 - phlegmon 169
 - with quintus varus 51–2
- hammer toe 31, 47, 58
- hallux valgus 57
 - onychomycosis 54–6, 57
- hands, hyperkeratosis of palms 76
- heel
- cracks 65, 67–8
 - dry gangrene 129–30
 - osteomyelitis 179–83
 - ulcers
 - debridement 132–3

- neuro-ischemic 114, 115
- neuropathic 96–8, 212
- heel protector ring 129, 130
- heloma durum 48–9, 50
- heloma molle 49–50
 - hemorrhagic callus formation 66
 - kissing 50
- hindfoot
 - preservation 69
 - resorption 210, 211
- Huckstep nail 208, 210
- Hyaff® 39
- hydrocolloids 119
- hydrogels 119
- hydrotherapy 119
- hyperhidrosis 158
- hyperkeratosis
 - eczema 78, 79
 - fifth toe 155
 - metatarsal heads 95
- hypoesthesia
 - neuropathic ulcers 27
 - zone 5
- iliac artery
 - atheromatous disease 114, 139, 144
 - common, stenosis 127
 - proximal, stenosis 134, 137
 - stenosis 7, 132, 134, 135, 140
- infection
 - under callus
 - over fifth toe 155–6
 - at tip of claw toe 155
 - deep-tissue 161–3
 - after interphalangeal mycosis 161, 162
 - Charcot foot with neuropathic ulcer 163–4, 165, 166, 167
 - gangrene 127
 - ulceration 153–4
 - web space 157–9
 - see also* abscess; cellulitis; fungal infection; sepsis; soft-tissue infection
- insoles, flat 174
- insoles, shock-absorbing 31, 32
 - convex triangular foot 52
 - custom-molded 32, 33
 - pes cavus 46
- intermittent claudication 7, 16
 - digital subtraction angiography 138
 - ischemic ulcers 28
- internal fixation 210
- interphalangeal joint, distal
 - calcium pyrophosphate dihydrate deposition disease 77, 78
 - plantar flexion 47, 56
- interphalangeal joint, proximal
 - osteomyelitis 109
 - plantar flexion 47
- intrinsic muscles of foot, atrophy 47
- joint mobility, limited 3
 - plantar pressure 33
- keratinocytes 38
- keratoderma, palmoplantar 75–7
- Klebsiella*, deep-tissue 162
- Klebsiella pneumoniae* 168
- larval therapy 119, 121
- latissimus dorsi musculocutaneous flap 81, 82, 83
- leuconychia mycotica 160
- lichenification 78
- ligament ossification 206, 207
- limb ischemia, critical 18
- lipid level reduction 16
- Lisfranc joint *see* tarsometatarsal joint
- lymphangitis 153
- maggot debridement 119, 121
- magnetic resonance angiography (MRA) 14, 16
- magnetic resonance imaging (MRI)
 - osteomyelitis diagnosis 175, 177
 - phlegmon imaging 171
- male patients, foot ulcers 3
- malleolus, ulcer 43
- mallet toe 56, 58
- Marjolin's ulcer 81
- mechanical debridement 119
- medial arterial calcification 8
- Meggitt—Wagner classification of ulcers 25
 - modification 26
- metastases, dermatofibrosarcoma protruberans 83
- metatarsal bone
 - avulsion fracture 193
 - diaphysis resorption 203, 204
 - fifth
 - pencil-like appearance 203, 204
 - pseudoarthrosis 199
 - lateral displacement 195, 196
 - osteomyelitis 191
 - phlegmon under base 170, 171
 - subluxation of second 195, 196
- metatarsal epiphysis, osteolytic lesions 178
- metatarsal heads
 - broadening 192

- metatarsal heads (*continued*)
 callus formation 45, 50–1, 53
 hallux disarticulation 99
 second toe removal 59, 60, 61, 62
 cup formation 190
 erosion in osteomyelitis 103
 fat pad
 displacement 47, 65, 90, 91, 103
 reduced thickness 91–2
 fifth
 callosity 132
 exostosis 46
 neuro-ischemic ulcer with osteomyelitis 121–3
 neuro-osteoarthropathy 193–4
 neuropathic ulcer 99
 osteomyelitis 121–3, 201
 pseudoarthrosis in stress fractures 178
 ulcer 51
 first
 neuro-ischemic ulcer with osteomyelitis 113–14
 neuropathic ulcer 87–8
 osteomyelitis 62, 113–14, 176, 177, 178
 fourth
 erosion 175, 177
 neuropathic ulcer 69–71, 90–2, 174
 hyperkeratosis 43, 95
 necrosis 61
 osteolysis 172, 191
 proliferative changes 192
 prominent 3, 5, 31, 58–9
 callus 64–5
 neuropathic ulcer 87–90, 93–5
 phlegmon 169
 plantar pressure 66
 soft tissue infection 156
 ulcer under callus area 66–7
 stress fractures 92
 third and hemorrhagic callus 65–6
 ulcer formation 67
 metatarsal joint, dislocation of first 197
 metatarsophalangeal joint
 arthrodesis 61, 62
 bone resorption 178
 dorsiflexion 47
 fourth toe disarticulation 70–1
 hallux disarticulation 63, 95
 osteoarthritis 134, 139
 osteolytic destruction 190
 subluxation 190
 methicillin-resistant *Staphylococcus aureus* (MRSA) 121, 156
 infected plantar ulcer with osteomyelitis 171
 painful ulcer 200
 mid-tarsal disarticulation 127, 129, 130
 wet gangrene 142–3, 145
 midfoot
 chronic ulcer 201, 202, 203
 collapsed 90, 200–1, 202
 midsole, neuro-ischemic ulcers 114, 115
 Moh's micrographic surgery 83
 monofilaments 5–6
 muscles, small 5
 musculocutaneous flap, latissimus dorsi 81, 82, 83
 mycosis, interphalangeal 161, 162
 nails
 deformities 75, 76
 ingrown 71, 146
 repetitive trauma 75
 navicular bone
 collapsed 203
 resorption 210, 211
 naviculocuneiform joint
 bone resorption 201, 202
 collapse 201, 202
 destruction 203, 204
 neuro-osteoarthropathy 203
 necrobiosis lipoidica 78–80, 81
 necrotizing fasciitis 153
 nephropathy, diabetic 53
 neuro-osteoarthropathy 3, 164, 165, 166
 acute 189–91, 193, 197, 206
 acute osteomyelitis differential diagnosis 191–3
 acute dissolution phase 197
 ankle 204, 205, 206, 207
 arthrodesis 199
 blood supply 201
 bone destruction 189
 bone/joint destruction anatomic patterns 187–8
 calcaneocuboid joint 203
 classification 187–9
 claw toe 191
 clinical presentation 188
 cuboid bone fragmentation 199–200
 differential diagnosis 189
 Dounis classification 187–8
 type I 189–91
 type II 193–203, 204–5
 type III (a, b and c) 206–8, 210–12

- neuro-osteoarthropathy (*continued*)
 type IIIa 208, 209, 210
 type III α 204, 205, 206
 edema 193
 Eichenholtz stage I 197
 Eichenholtz stage III 199
 femoral—popliteal bypass graft 95
 foot deformities 31, 207
 functional instability 207
 immobilization 199, 206, 210
 internal fixation 210
 metatarsal heads 193–4
 midfoot ulcers 36
 naviculocuneiform joint 203
 osteophyte formation 204
 radiological findings 188
 reconstructive stage 206, 208
 Sanders and Frykberg classification 187
 pattern I 189–91
 pattern II and III 193–203, 204–5
 pattern IV 204, 205, 206–8, 209, 210
 pattern IV and V 210–12
 stages 188
 talonavicular joint 203
 tarsometatarsal joint 193, 197, 203
 neurothesiometer 6
- onychocryptosis 71
 wet gangrene 150
 onychodystrophy 134, 136
 claw toe 155
 ischemic ulcers 28
 peripheral vascular disease 132
 phlegmon 169
 wet gangrene 146
 onychogryposis 75, 76
 onychomycosis 54–6, 57, 159–60
 callus under bone prominence 64
 distal subungual 159–60
 neuropathic ulcers of metatarsal heads 94
 proximal subungual 160
 orthosis, patellar-tibial-bearing 208
 osteoarthritis, metatarsophalangeal joint 134, 139
 osteomyelitis 95, 96, 167–8
 bone destruction 189
 bone scintigraphy imaging 179, 181
 burns 102
 calcaneus osteolysis 211, 212
 chronic neuro-osteoarthropathy 211
 chronic ulcer 203
 diagnosis 175
- Escherichia coli* 104
 hallux 168–9, 176, 178
 with neuro-ischemic ulcers 117, 118, 119, 120, 121
 heel 179–83
 infected ulcer 153
 plantar 171–3
Klebsiella pneumoniae 168
 metatarsal head
 fifth 201
 first 62, 176, 177, 178
 metatarsals 191
 neuro-ischemic ulcer
 fifth metatarsal head 121–3
 first metatarsal 113–14
 forefoot 114, 116–17
 hallux 117, 118, 119, 120, 121
 neuropathic ulcer complication 102–4, 173–5, 176, 177
 chronic 176, 178, 180
 pathogens 103, 104
 proximal phalanx 63
Pseudomonas aeruginosa 119
 radiographic evaluation 154
 radiography 192
Staphylococcus aureus 103, 104, 108, 113–14, 116, 168–9
 treatment regimens 104
 acute infection 172
- osteophytes 204
 ankle 208, 209
 oxygen
 hyperbaric 36, 38
 transcutaneous pressure (TcPO₂) 9–10
- pain
 neuropathic 4
 plantar heel spur 43
see also rest pain, ischemic
 painful—painless foot 90
 Palmaz stent 137
 papain 119
 papules, violaceous 79
 paronychia 71
 progression to gangrene 7–8
 patellar-tibial-bearing orthosis 208
 peak systolic velocity (PSV) ratio 12, 14, 15, 16, 17
 wet gangrene 148, 150
 pedal arteries
 calcification 112
 pressure index 10
 reconstitution 140

- percutaneous transluminal angioplasty 135
 gangrene 127
 neuro-ischemic ulcers 113
 popliteal artery 168
 wet gangrene 147
 peripheral neuropathy 3
 claw toes 47
 painless ulcer 30
 prevalence 4
 symptoms 5
 tests 5–6
 peripheral vascular disease 3, 6–14, 15, 16,
 17, 18, 19, 20
 detection and follow-up 16, 18, 20
 gender differences 140
 neuropathy co-existence 7
 prevalence 6–7
 peroneal artery stenosis 140
 pes cavus 44–6
 callus over prominent metatarsal heads 65
 pes planovalgus 51
 pes planus 43, 112
 bunionette 43–4
 phalanges, concentric resorption 189–90
 phalangophalangeal joint
 hemorrhagic callus 67, 68
 subluxation 190
 phalanx, proximal, osteomyelitis 63
 phlegmon 169–71
 photoplethysmography 11
 plantar abscesses 133
 plantar arch
 collapse 200–1
 high 45
 plantar arteries, reconstitution 140
 plantar branch artery, medial 181, 183
 plantar cracks 30
 plantar heel spur pain 43
 plantar ligament preservation 90
 plantar pressure
 callus formation 88
 convex triangular foot 52
 fat pad thickness reduction 91–2
 high 3
 in-shoe measurement 52, 54, 91, 92
 limited joint mobility 33
 offloading 33–6
 prominent metatarsal heads 66
 redistribution 32
 reduction 31, 32
 ulcer under callus area 67
 platelet-derived growth factor β (PDGF- β)
 38, 172
 plethysmography, segmental 11
 ischemic ulcers 29
 popliteal artery
 atheromatous disease 146
 collateral supply 132, 135
 obstruction 122, 123
 percutaneous transluminal angioplasty 168
 stenosis 114, 127
 popliteal—peripheral bypass 117, 150
 pre-ulcer formation 88
 pressure loading 87–8
 pressure offloading 103, 107, 114
 heel 131
 neuropathic ulcer 174
 pressure perception 5–6
 pressure ulcers 129
 gangrenous 132, 133
 probe-to-bone tests 175
 prosthesis, below-knee 208
 Proteus infection 153
 Proteus mirabilis 181
 Proteus vulgaris 178
 pseudo-gout *see* calcium pyrophosphate
 dihydrate (CPPD) deposition disease
 Pseudomonas 153
 infected ulcers 154
 Pseudomonas aeruginosa 104
 fungal infection with multimicrobial
 colonization 160
 osteomyelitis 119
 wet gangrene 132
 wound colonization 121
 Pseudomonas maltophilia 168
 pulsed Doppler imaging 12
 quality of life 3
 quintus varus 51–2
 radiography
 Charcot foot 192
 magnification 175
 neuro-osteoarthropathy 188
 osteomyelitis 154, 175, 192
 Regranex® *see* platelet-derived growth factor
 β (PDGF- β)
 rest pain, ischemic 7
 persistent recurring 127
 toe pressure 9
 transcutaneous oxygen pressure 10
 revascularization procedures 114
 heel gangrene 130
 rocker bottom deformity 90
 scars, squamous cell carcinoma formation 81
 scintigraphy *see* bone scintigraphy imaging
 Scotch-cast boot 35

- segmental pressure measurement 10–11
 ischemic ulcers 29
- Semmes—Weinstein monofilaments 5–6
- sensation loss 4, 5
 footwear 31
- sensory deficit 5
 neuropathic ulcers 27
- sepsis, wet gangrene 130, 132–3, 134, 135, 136
- sesamoid bones 63
- shear stress
 reduction 32
 ulcers 54
- shoes
 athletic 31
 foreign objects 156, 157
 half 35, 36, 66
 heel-free 36, 97
 manufacturers 219
 pressure relief 35–6
 rocker-style 33, 35, 88
 roller-style 33
 selection 29–30
 therapeutic for neuropathic ulcers 87, 88
 trauma 3
 ulcer on dorsum of foot 110–11
see also footwear, preventive; insoles
- silicone ring 111, 112
- skin
 atrophy 112
 changes
 critical limb ischemia 18
 ischemic ulcers 28
 ischemic necrosis with dry gangrene 129
 lesions
 shoe friction 111
 toe pressure 9
 pathogens 154
 scaling 134
see also ulcers/ulceration; wound(s); wound healing
- skin, dry 3
 callus
 under bone prominence 64
 over prominent metatarsal heads 65
 heel 79
 cracks 67–8
 phlegmon 169
- smoking cessation 16
- socks, padded 31
- soft-tissue infection 102, 156–7
 under callus at tip of claw toe 155
- soft-tissue sarcoma 82–3
- sole of foot
 hyperkeratosis 76
 midsole neuro-ischemic ulcers 114, 115
- squamous cell carcinoma 81–2
- S(AD)SAD classification for foot ulcers 26
- staphylococcal toxins 133
- Staphylococcus aureus* 62, 97, 98
 antibiotics 182, 183
 callus over fifth toe 155
 deep-tissue 162
 after interphalangeal mycosis 161
 fungal infection with multimicrobial colonization 160
 infected ulcers 154
 non-limb-threatening infections 156
 osteomyelitis 103, 104, 108, 113–14, 116, 168–9
 chronic neuropathic ulcer 178
 web space infection 158
 wet gangrene 132, 146
see also methicillin-resistant *Staphylococcus aureus* (MRSA)
- Staphylococcus epidermidis* 104
- Steinmann pins 197
- stents 135, 137, 140, 141
 aortic 134, 138
 aorto-iliac intravascular 132, 136
- Streptopomonas maltophilia* 122
- streptococci
 non-limb-threatening infections 156
 toxins 133
- stress fractures
 metatarsal heads 92, 178
 pseudoarthrosis in fifth metatarsal 178
 toes 134, 139
- surgical debridement 119
- sweating inhibition 3
- Syme ankle disarticulation 181
- tailor's bunion *see* bunionette formation
- talonavicular joint
 Chopart dislocation 69
 collapse 201, 202
 destruction 203, 204
 fragments 199, 200
 neuro-osteoarthropathy 203
- talus resorption 206, 207, 208, 209, 210, 211, 212
- tardus parvus waveform 19, 149, 150
- tarsometatarsal joint
 destruction 203, 204
 disruption 164, 165, 201
 Lisfranc joint line 43
 neuro-osteoarthropathy 193, 197, 203
 partial resorption 194–5, 196, 197
 phlegmon 170

- tarsometatarsal joint line, front transverse
convexity 43
- tarsometatarsophalangeal joint fusion 198
- technetium-99m 179, 181
- thermal injury *see* burns
- tibia
resorption 206, 207, 211, 212
subchondral osteosclerosis 206, 207, 208,
209
- tibial artery
atheromatous disease 138, 146
occlusion 140
stenosis 117, 140
- tibial artery, anterior
obstruction 150
spectral waveform 149
stenosis 16, 18
- tibial artery, posterior 14, 16
calcification 112
calcinosis 181, 183
palpation 18
stenosis 18, 148
- tibioperoneal artery 140
- tinea pedis 76
interdigital 158
- toe(s)
amputation 121, 122, 134, 146, 202, 203
ray 140
burns 98, 99, 100, 101, 102
deformities 30, 31
incidence 56
varus deformity 46, 48
dry gangrene 127, 128, 130, 131, 133–4,
136–8
- fifth
amputation 121, 122
hemorrhagic callus 200
infection under callus 155–6
stress fracture 134, 139
- fourth
amputation 134
disarticulation 70–1
ulceration by pressure from little toe 111
- overriding 50–1
causes 55
hallux valgus 52–4, 56, 57
pressures 8–9
sausage-like appearance 167, 168
- second
amputation 202, 203
disarticulation 116, 132
lateral exostosis 192
medial displacement 99
removal 59, 60, 61, 62
- silicone ring 111, 112
- ulcer formation 67
- wet gangrene 139–40, 144
see also claw toe; curly toe deformity;
hammer toe; mallet toe; nails
- toe, great *see* hallux
- transcutaneous oximetry 9–10
- transcutaneous oxygen pressure (TcPO₂)
9–10
- transparent films 119
- trauma 3
progression to gangrene 7–8
- Trichophyton* 158, 160
- triplex scan, wet gangrene of foot 148, 149,
150
- trypsin 119
- ulcers/ulceration 29
under callus area 66–7
cellulitis 114, 115
Charcot foot 95–6
classification 25–6
critical limb ischemia 18
curettage 153
depth—ischemia classification 26
under hallux 67, 68
infection 25–6, 27, 153–4
clinical assessment 153
cultures 153
diagnosis 153
plantar with osteomyelitis 171–3
wet gangrene 142–3, 145
- ischemia 25–6
clinical presentation 26, 28–9
load reduction 34
midfoot 201, 202, 203
necrobiosis lipoidica 80
neuro-ischemic 26, 30
claw toes 120, 121
clinical presentation 29
dorsum of foot 109–11
fifth metatarsal with osteomyelitis 121–3
first metatarsal with osteomyelitis
113–14
forefoot with osteomyelitis 114, 116–17
under hallux 107, 108
hallux with osteomyelitis 117, 118, 119,
120, 121
under hallux with osteomyelitis 107–9,
110
heel 114, 115, 180, 182
interdigital 111, 112
medial side of foot 112–13
midsole 114, 115

- neuropathic 4, 26, 69–71
 - bunion 177
 - callus formation 169
 - Charcot foot and deep-tissue infection 163–4, 165, 166, 167
 - chronic and osteomyelitis complication 176, 178, 180
 - clinical presentation 26–8
 - collapsed midfoot 90
 - under fifth metatarsal head 99
 - first metatarsal head 87–8
 - foreign body 212
 - under fourth metatarsal head 90–2
 - under heel 96–8
 - neuro-osteoarthropathy 195
 - osteomyelitis complication 102–4, 173–5, 176, 177
 - prominent metatarsal heads 87–90
 - under prominent metatarsal heads 93–5
- new treatments 36, 38–9
- pathways to 4
- prevalence 3, 67
- prevention 29–33
- progression to gangrene 7–8
- recurrence 116–17
- risk categories 30, 31–3
- risk factors 3–4
 - reduction 117
- total-contact cast 34
- see also* pressure ulcers
- ultrasonography
 - arterial 11–14, 16, 17, 18, 19
 - color frequency mapping 12
 - duplex scanning 12
 - ischemic ulcers 28–9
 - phlegmon imaging 171
 - pulsed Doppler 12
 - sensitivity 14
 - waveform analysis 12–14
 - wet gangrene of foot 148, 149, 150
- University of Texas classification system for diabetic foot wounds 25–6
- varus deformity 208
 - ulcer under callus area 66–7
- vascular status assessment 6–14, 15, 16, 17, 18, 19, 20
 - invasive 20
 - noninvasive 8–14, 15, 16, 17, 18, 19, 20
- vascular surgery 113, 121
 - femoro—tibial bypass graft 123
- vibration perception 5
 - thresholds 6
- walkers
 - prefabricated 195, 196, 197
 - removable cast 34–5
- walking, Chopart disarticulation 69
- web space infection 157–9
- wet-to-dry dressings 119
- windlass mechanism maintenance 63
- wound(s)
 - bacterial contamination 121
 - classification 25–6
 - infection 153
 - irrigation 119
- wound healing
 - accelerating factors 38–9
 - failure 10