A PRACTICAL MANUAL OF

Diabetic Foot Care

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To our families: Audrey, Stephen and
Susie Edmonds; John, Julien, William and Dennis Foster
and Debra, Rebecca, Douglas and Lauren Sanders.

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We refer to patients throughout the book as 'he' simply because more men than women seem to develop diabetic foot ulcers.

This is a practical hands-on manual, uninterrupted by references. At the end of each chapter, we have given a classified reading list which should provide further information for our readers.

Prologue

Time's the king of men;
He's both their parent and he is their grave,
And gives them what he will, not what they crave.
*(*Pericles, Prince of Tyre, *II, iii, William Shakespeare)*

THE SCOPE OF THE PROBLEM

Diabetic foot complications are a major global public health problem. Amputation rates vary throughout the world but are always increased in people with diabetes compared to those without diabetes. Amputations are increasing in diabetic patients. Throughout the world, health-care systems, both public and private, have been unsuccessful in managing the overwhelming problems of patients suffering with diabetic foot complications. The results of this failure are shown in the following case histories, illustrated in Figs 1–5.

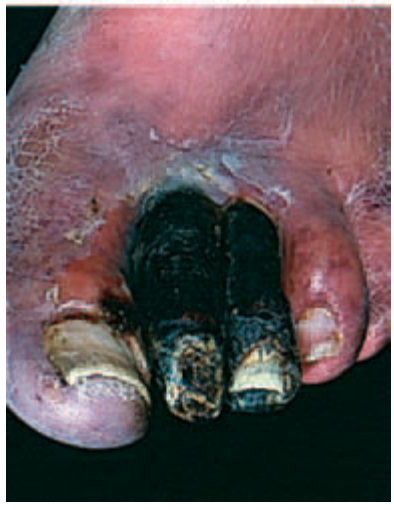


Fig. 1 Foot from the UK. This 85-year-old man with type 2 diabetes of 8 years' duration received regular dressings of his ulcerated ischaemic foot for 9 months, but was not referred until extensive gangrene had developed.

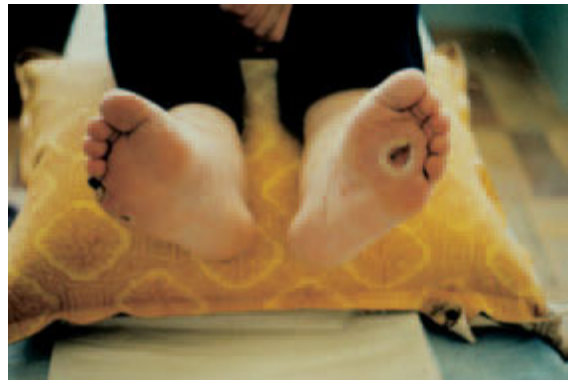


Fig. 2 Foot from Ukraine. This 48-year-old man with type 2 diabetes of 12 years' duration trod on a nail and developed severe infection with wet gangrene of the right 5th toe. He had a longstanding neuropathic ulcer of the left foot.

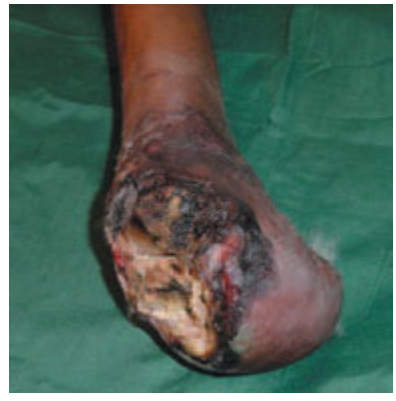


Fig. 3 Foot from Sudan. This 80-year-old lady with type 2 diabetes of 15 years' duration and neuropathic feet sustained a puncture wound through the thin sole of her sandal. She did not seek advice and developed profound sepsis.

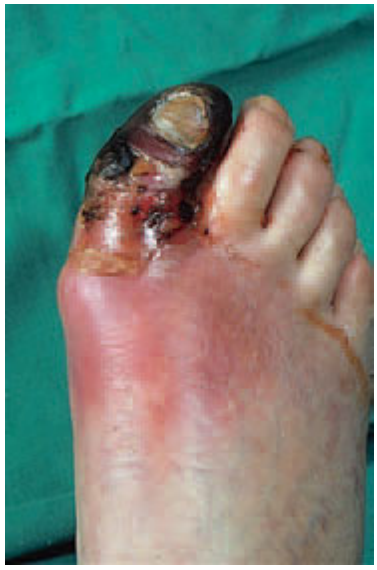


Fig. 4 Foot from the USA. This 58-year-old woman with type 2 diabetes of 9 years' duration, wore a tight shoe which rubbed a blister. She did not seek help because the blister was not painful and presented late—with gangrene.

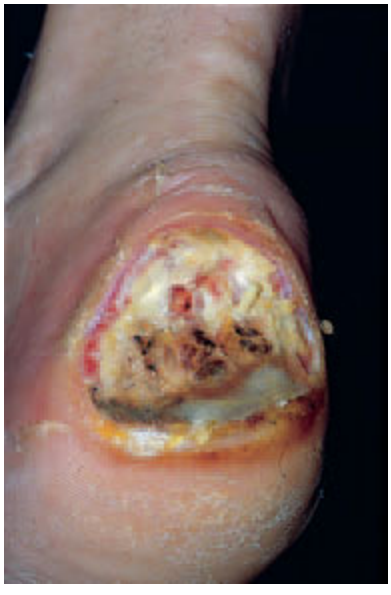


Fig. 5 Foot from Australia. This 75-year-old man with type 2 diabetes of 20 years' duration developed fissures round his heel which were a portal of entry for severe infection.

These pictures show authentic diabetic feet from five continents of the world. Foot catastrophes such as these, as Elliott Joslin pointed out, do not strike like lightning out of heaven, but are too often due to ignorance and apathy, which prevent patients from detecting problems early and seeking treatment, and which prevent health-care professionals from organizing rapid and effective care. In nearly every case there are warning signs which, if acted upon, could prevent tragedy. However, because of local barriers to effective care, patients often do not receive help in time to save their feet.

Diabetic patients in the real world are often perceived as the poor relations, the 'lepers of our time'. Indeed, diabetic foot patients have more in common with lepers than just neuropathy: in many quarters they are regarded with disgust and antipathy as dirty, smelly, 'unclean', socially unacceptable feet belonging to patients who take up hospital beds for unacceptably long periods of time.

Equally, diabetic foot patients may be regarded by inexperienced staff as 'feckless' patients, who fail to look after themselves and are directly responsible for their problems. Health-care systems are 'symptom-led' and thus fail to respond to the needs of the diabetic foot patient, who usually has neuropathy, numb feet and no complaints.

In addition, diabetic foot problems are frequently underestimated. Just as there is no such entity as 'mild' diabetes, there is no such thing as a 'trivial' lesion of the diabetic foot.

Sadly, there are many areas of the world where people with diabetes are unable to obtain good foot care, or where the provision of such care is dependent upon the patient being able to pay for it. A recent tragic case involved a diabetic man with indolent neuropathic ulceration, who amputated his own leg (using a railway line and a passing train) because he could not pay for medical care.

However, amputations are not inevitable. The aim of this book is to help readers to achieve good care for patients with diabetic foot problems and so avoid preventable amputations. Progression down the road to amputation is not inevitable and relentless. Patients can be rescued.

HISTORICAL BACKGROUND

The last century made great inroads into improving the management of diabetes. The early work of pioneers such as Nicolas Paulesco in Rumania and Georg Zuelzer in Germany culminated in the work of Banting, Best, Collip and Macleod in Canada who produced a pancreatic extract which was used successfully in patients and ended the

inevitable 'death sentence' hitherto attached to a diagnosis of type 1 diabetes. Insulin became widely available, and the subsequent development of oral hypoglycaemic agents and blood glucose monitoring also led to improved outcomes for type 2 patients. However, many diabetic patients lived longer only to develop diabetic complications, including peripheral neuropathy, peripheral vascular disease, ulceration, foot sepsis and gangrene.

The work of Elliott Joslin (USA) and R.D. Lawrence (UK) during the first half of the twentieth century was concerned with all aspects of diabetes management including the management of the foot. Since then there has been activity on all continents to attempt to reduce amputations and improve outcomes in diabetic foot patients, which have been particularly evident over the past 15 years.

ADVANCES IN DIABETIC FOOT CARE

The diabetic foot has become a major area of interest, and insight has been gained into the reasons why diabetic feet go wrong and the ways in which patients can be helped. Of all the complications of diabetes, the diabetic foot is probably the easiest to prevent and treat.

The groundswell of interest in the diabetic foot surged in the 1980s, and developments in foot care included the setting up of multidisciplinary diabetic foot clinics (Fig. 6) and the pioneering educational work of Jean Philippe Assal in Geneva, Switzerland. Paul Brand, Frank Tovey and Grace Warren worked in India as medical missionaries with leprosy patients and subsequently applied their knowledge to the management of diabetic neuropathic patients, spreading the word to Carville, USA, Basingstoke, UK, and Sydney, Australia. The popular biennial Malvern diabetic foot conferences began in 1986, and in 1991 Karel Bakker of The Netherlands established the regular International Symposia on the Diabetic Foot at Noordwijkerhout in The Netherlands. Bakker's work led to the establishment of International Working Group of the Diabetic Foot, which produced the *International Consensus on the Diabetic Foot*, published in 1999 (and now translated into 20 languages). Lee Sanders, who has contributed the chapter on surgery to this book, was the first podiatrist to be elected President for Healthcare and Education of the American Diabetic Association.

Fig. 6 International visitors at the King's Diabetic Foot Clinic: left to right, Dr Kamenov (Bulgaria), the Authors, Dr Harkless (USA) and Dr Plamen (Bulgaria).

Fig. 7 The Khartoum Diabetic Foot Clinic.

Professor Mohammed Rasheid, a surgeon, established one of the first African diabetic foot clinics in Khartoum, Sudan, in 1998 (Fig. 7). Dr Hermelinda Pedrosa in Brazil has organized a national programme of diabetic foot care. Dr Theresa Que established the first diabetic foot clinic in The Philippines. The fall of the 'Iron Curtain' across Eastern Europe in the 1990s enabled the setting up of diabetic foot clinics in many countries including East Germany (Fig. 8) Russia, Romania and Lithuania (Fig. 9) The late Jacquie Lloyd Roberts, a UK podiatrist, established a successful chain of diabetic foot clinics in Ukraine before her untimely death.

The work of diabetic foot clinics, operating in different parts of the world, in very different conditions, has clearly demonstrated that outcomes for diabetic foot patients can be improved when dedicated and enthusiastic clinicians organize a multidisciplinary diabetic foot service. With a flexible approach, most problems can be overcome.

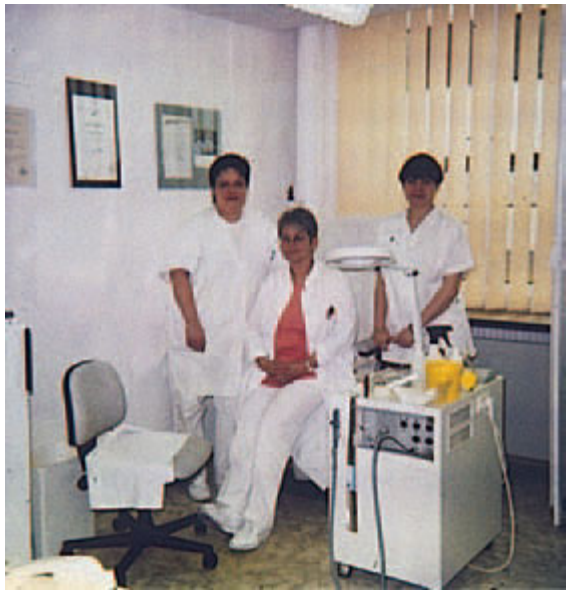


Fig. 8 Foot clinic in Magdeborg, East Germany—Dr Karola Zemlin's team.

Fig. 9 Foot care nurses in Kaunas who learned foot care from an English podiatrist, Abigail Clarke.

If key members of the team, such as podiatrists, are not available in certain countries, then doctors or nurses can take on many aspects of the role of the podiatrist. Indeed, we have learnt from the work of Dr Grace Warren with Hansen's disease patients that neuropathic patients and their families can be taught safe self-care techniques for removing callus to prevent ulceration if no other help is available.

One of the most important messages from these workers is that successful interventions in the real world do not depend on the possession of high-technology

Fig. 10 International consensus and practical guidelines on the diabetic foot.

equipment and vast financial budgets. Barriers to care which at first glance appear to be insurmountable can usually be overcome if we learn lessons from our own and other people's experiences, and, in the words of E. M. Forster, 'only connect' with each other.

'Experts' are sometimes called in to help to set up systems for managing the diabetic foot. However, experts are often outsiders from other regions or countries, who may lack information about local conditions and should refrain from being too dictatorial to local practitioners who may have expert and first-hand knowledge of local conditions and problems. While we always welcome visitors from abroad to our foot clinics, we try to avoid being too dogmatic about how they should manage feet back home: they observe what we do and extract what is relevant to their own situation but should not regard our messages as being cast in stone.

There is a dearth of evidence for treatments applied to the diabetic feet. One of the problems encountered in developing guidelines for management of the diabetic foot is that evidence is often lacking, and rarely comes from large, properly conducted, randomized controlled trials. Our recommendations are based either upon first-hand experience gained over the past 22 years working in the outpatient diabetic foot clinic and on the wards or on the published work of other groups, throughout the world.

In the spirit of the International Consensus on the Diabetic Foot (Fig. 10), we have tried to make this book relevant to all practitioners who want to set up a diabetic foot service, no matter where or under whatever conditions they labour: we hope that there is something here for everybody.

These are heady times for devotees of the diabetic foot, and we hope and believe that a new generation of young and enthusiastic practitioners will be there to take up the baton for the diabetic foot in the future. We hope that they will find this book useful and practical.

PRACTICE POINTS

- Throughout the world, health-care systems are symptom-led and they often fail to respond to the needs of the diabetic foot patient
- Diabetic foot problems are frequently underestimated: there is no such thing as a trivial lesion of the diabetic foot
- Diabetic foot patients are unfairly regarded as 'feckless'
- Increased interest in the diabetic foot over the last 25 years has resulted in major advances in the care of the diabetic foot patient
- The multidisciplinary diabetic foot service has been developed as a successful model of care throughout the world.

FURTHER READING

The scope of the problem

Boulton AJM, Vileikyte L. Diabetic foot problems and their management around the world. In Bowker JH, Pfeifer MA (eds). *Levin and O'Neal's The Diabetic Foot.* Mosby Inc., St Louis, USA, 2001, pp. 261–72.

The Global Lower Extremity Amputation Group. Comparing the incidence of lower extremity amputations across the world: the Global Lower Extremity Amputation Study. *Diabetic Medicine* 1995; **12**: 14–18.

Historical background

Bliss M. *The Discovery of Insulin.* University of Toronto Press, Toronto, Canada, 2000.

Joslin EP. The menace of diabetic gangrene. *N Engl J Med* 1934; **211**: 16–20.

Lawrence RD. *The Diabetic Life.* JA Churchill, London, UK, 1925.

McKeown KC. History of the diabetic foot. *Foot* 1992; **2**: 179–82.

Advances in diabetic foot care

Assal JP, Muhlhauser I, Pernet A *et al.* Patient education as the basis for diabetes care in clinical practice and research. *Diabetologia* 1985; **28**: 602–13.

Boulton AJM, Connor H, Cavanagh PR (eds). *The Foot in Diabetes*, 3rd Edn. Wiley and Sons Ltd, Chichester, UK, 2000.

Brand P, Yancey P. *The Gift of Pain.* Zondervan Publishing House, Grand Rapids, Michigan, USA, 1997.

Edmonds ME, Blundell MP, Morris ME *et al*. Improved survival of the diabetic foot: the role of a specialised foot clinic. *Q J Med* 1986; **232**: 763–71.

Faglia E, Favales F, Aldeghi A *et al.* Change in major amputation rate in a centre dedicated to diabetic foot care during the 1980s: prognostic determinants for major amputation. *J Diab Comp* 1998; **12**: 96–102.

The International Working Group on the Diabetic Foot. *International Consensus on the Diabetic Foot*. The International Working Group on the Diabetic Foot, Amsterdam, The Netherlands, 1999.

Larsson J, Apelqvist J, Agardh CD *et al*. Decreasing incidence of major amputation in diabetic patients: a consequence of a multidisciplinary foot care team approach? *Diabetic Medicine* 1995; **12**: 770–6.

Payne C. Increased frequency of diabetic foot publications. *Diabetic Medicine* 1997; **14**: 407.

Tovey FI. The manufacture of diabetic footwear. *Diabetic Medicine* 1984; **1**: 69–71.

Warren G, with Nade S. *The Care of Neuropathic Limbs, a Practical Manual.* Parthenon Publishing Group, London, UK, 1999.

1 Introduction

Time is the nurse and breeder of all good.
Hope is a lover's staff; walk hence with that,
And manage it against despairing thoughts.
*(*The Two Gentlemen of Verona*, III, i, William Shakespeare)*

In this book, the fundamental approach to the diabetic foot is built on a simple staging system which has been developed to provide a framework for diagnosis and management.

There are four main themes in this chapter.

- Practical assessment consisting of history, examination and investigations
- Basic classification of the diabetic foot, into the neuropathic and neuroischaemic foot
- Simple staging system, describing six specific stages in the natural history of the diabetic foot (Table 1.1)
- Multidisciplinary management plan for each stage.

This overall approach to the diabetic foot begins with a simple assessment to enable the practitioner to make a basic classification and staging. The practitioner can place the foot into each stage by means of a clinical assessment.

Having then placed the foot in a particular stage, investigations will be needed to assess severity so as to determine treatment. The book contains sufficient easily accessible information to enable the practitioner to make rapid, effective decisions which will detect problems early, organize rapid treatment and prevent deterioration and progression.

Table 1.1 Stages of the diabetic foot

Stage 1	Normal foot
Stage 2	High-risk foot
Stage 3	Ulcerated foot
Stage 4	Infected foot
Stage 5	Necrotic foot
Stage 6	Unsalvageable foot

PRACTICAL ASSESSMENT

This can be divided into three parts:

- History
- Examination
- Investigations.

History

Every attempt should be made to encourage the patient to be open and non-defensive. The history can be divided into the following sections:

- Presenting complaint
- Past foot history
- Diabetic history
- Past medical history
- Family history
- Drug history
- Psychosocial history.

Presenting complaint

Be aware that some patients may be asymptomatic due to neuropathy.

The presenting complaint is usually one or more of the following:

- Skin breakdown
- Swelling
- Colour change
- Pain.

For skin breakdown, swelling and colour change or any other presenting complaints, the following questions may be helpful:

- Where is the problem?
- When did it start?

- How did it start?
- What makes it better?
- What makes it worse?
- How has it been treated?

As regards pain, this may be a specific complaint alone or it may accompany the above problems.

Pain may arise locally or it may be diffuse. Local sources may originate from bone, joint and soft tissue including skin and subcutaneous tissue. Generalized pain in both feet suggests neuropathy. Diffuse pain in a single foot suggests ischaemia. However, pain in the ischaemic foot should not always be blamed on reduced arterial perfusion because it may be caused by infection. In the neuropathic foot, severe infections can still cause pain, particularly throbbing pain. Pain around an ulcer suggests infection or ischaemia. The following questions should be asked about pain:

- When did it start?
- How did it start?
- Was there an injury?
- Where is the pain?
- What is its nature?
- What aggravates the pain?
- What relieves the pain?
- When does it occur?
- Is it related to time of day or activity?
- What treatments have been given so far?

Clinical tips to diagnose pain due to neuropathy and ischaemia are shown in Table 1.2.

Table 1.2 Clinical tips on pain

Pain due to neuropathy
- Burning pain with contact discomfort in both feet and lower legs which may also involve the thighs
- Sharp shooting (lancinating) or lightning pains like electric shocks, lasting a few seconds
- Pain relieved by cold
- Pain worse during periods of rest
- Unilateral burning pain in the leg with muscle wasting suggests a focal neuropathy, commonly a femoral neuropathy

Pain due to ischaemia
- Persistent pain, worse on elevation and relieved by dependency (hanging the leg out of bed)
- Pain in the calf on exercise relieved by rest (claudication). However, claudication is often absent in ischaemia because of concurrent neuropathy and the distal distribution of the arterial disease
- Feet with severe ischaemia may have little pain because of neuropathy

Patients may not complain of pain itself but of other abnormal sensations which would suggest neuropathy.

- Pins and needles (paraesthesiae)
- Unpleasant tingling (dysaesthesiae)
- Tightness (as if a constricting band is around the foot)
- Cold
- Heaviness
- Numbness ('my feet feel as if they don't belong to me').

After discussing the presenting complaint, the rest of the history is devoted to gathering important relevant information about the patient to aid diagnosis and management. This information can be acquired from various sources including direct questioning of the patient, the patient's medical notes and the referral letter.

Past foot history

- Previous ulcers and treatment
- Amputations:
  Major
  Minor
- Peripheral angioplasties
- Peripheral arterial bypasses.

Diabetic history

- Type of diabetes
- Duration of diabetes
- Treatment of diabetes:
  Insulin
  Oral hypoglycaemics.

Complications of diabetes

Retinopathy
- Background
- Proliferative
- Previous laser therapy
- Vitrectomy
- Cataract.

Nephropathy
- Proteinuria
- Severe renal impairment (creatinine > 250 μmol/L, 2.83 mg/dL)
- Renal replacement therapy:
  Continuous ambulatory peritoneal dialysis (CAPD)
  Haemodialysis
  Renal transplant.

Cardiovascular
- Angina
- Heart failure

- Myocardial infarction
- Coronary artery angioplasty
- Coronary artery bypass.

Cerebrovascular
- Transient ischaemic attack
- Stroke.

Past medical history
- Serious illness (e.g. cancer, rheumatoid arthritis, etc.)
- Accidents
- Injuries
- Hospital admissions
- Operations.

Drug history
- Present medication
- Known allergies.

Family history
- Diabetes
- Other serious illness
- Cause of death of near relatives.

Psychosocial history
- Occupation
- Number of cigarettes smoked per day
- Number of units of alcohol per day
- Psychiatric illness
- Home circumstances:
  Type of accommodation
  Lives alone
  Lives with friends or relatives.

Examination

There is a need for sensitivity on the part of the examiner. Many patients will be fearful and anxious at their first visit. If, rarely, they have ischaemia but no neuropathy, or they have a severely infected foot, then they will be afraid that the examination will be painful. Other patients may be embarrassed about their feet, or may have very sensitive and ticklish feet. Before the feet are handled the patient should be reassured that the examination will not be painful and that everything will be explained. The feet should be grasped gently but firmly, and poking, prodding and tickling should be avoided. The toes should be separated gently: if they are pulled apart violently the skin may split.

The examination should be performed systematically. It consists of five parts:
- Inspection
- Palpation
- Neurological assessment
- Footwear assessment
- General examination.

Inspection
The feet should be fully examined in a systematic fashion: first the right and then the left, including dorsum, sole, medial border, lateral border, back of the heel, malleoli and interdigital areas, with a full assessment of the following:
- Skin
- Callus
- Nails
- Swelling
- Deformity
- Limited joint mobility
- Colour
- Necrosis.

Skin
The general features of the skin should be assessed, especially looking for signs of skin breakdown.

In the neuropathic foot, the skin is dry and fissured and prominent dilated veins secondary to autonomic neuropathy may be visible. Hair loss can be a sign of neuropathy as well as ischaemia. Atrophy of the subcutaneous layer with a thin, shiny, wrinkled skin may indicate ischaemia.

The classical sign of skin breakdown is the foot ulcer. Ulcer assessment is described in Chapter 4. Abrasions, bullae and fissures also represent breakdown of the skin. Bullae are often the first sign of skin breakdown in the ischaemic foot. They are also a feature of fungal skin infections (tinea pedis), as is webspace maceration. Dry skin around the heel will form deep fissures unless an emollient is applied regularly (Fig. 1.1).

Look for other skin lesions, on the leg as well as the foot, including:
- Necrobiosis lipoidica diabeticorum
- Shin spots (diabetic dermopathy).

Necrobiosis lipoidica diabeticorum (NLD) is characterized as well-circumscribed red papules that extend radially with waxy atrophic telangiectatic centres (Fig. 1.2a,b). NLD evolves to ulceration in about one-third of cases.

The round or oval macular hyperpigmented lesions of diabetic dermopathy are found in the anterior tibial region.

As well as skin lesions specific to the diabetic foot, it is important to recognize inflammatory skin disease such as psoriasis, eczema and dermatitis, which also occur in

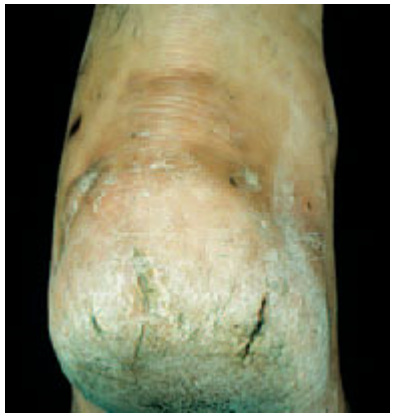


Fig. 1.1 Fissures are a portal of entry for infection and can lead to severe ulceration.

non-diabetic patients but may complicate the diabetic foot and leg.

Corns and callus

These are thickened areas of keratosis which develop at sites of high pressure and friction (Fig. 1.3). Corns are discrete areas, usually not more than 1 cm in diameter, and can extend to a depth of several millimetres. Callus forms diffuse plaques. Neither should be allowed to become excessive as this can be a forerunner of ulceration (usually in the presence of neuropathy). Haemorrhage within callus is an important precursor of ulceration.

Nails

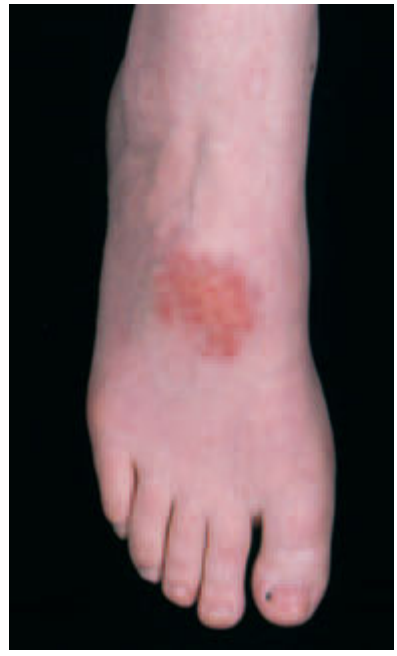
It is important to inspect the nails closely as the nail bed and periungual tissues may become the site of ulceration. The following should be assessed:

- Structure of the nails
- Colour of the nail bed
- Abnormalities under the nail
- Signs of nail infections.

Structure of the nails

Thickened nails are common in the population at large. If the shoes press on the nails they may cause bleeding under the nail. Eventually this may lead to ulceration. Atrophic nails may be present in patients with neuropathy and ischaemia.

Ingrowing toe nail (onychocryptosis) arises when the nail plate is excessively wide and thin, or develops a convex deformity, putting pressure on the tissues at the nail edge. Callus builds up in response to pressure and inflammation. Eventually, usually after incorrect nail cutting or trauma, the nail penetrates the flesh.



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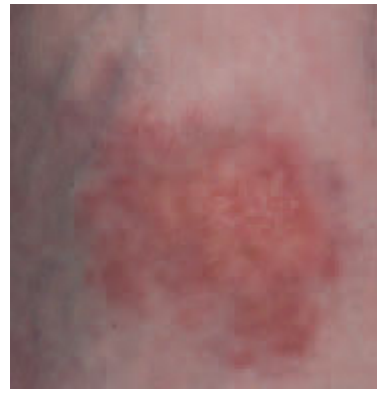


(b)

Fig. 1.2 (a) Necrobiosis lipoidica diabeticorum (NLD) on dorsum of foot and (b) close up of NLD.

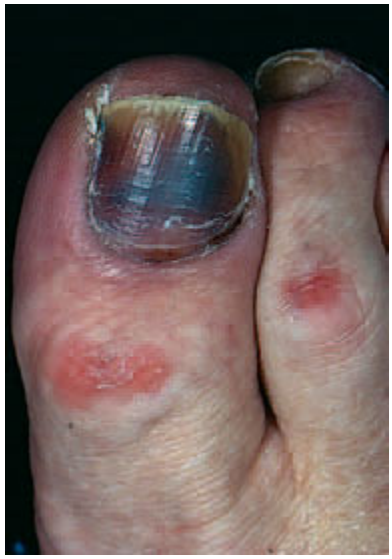
Colour of nail bed

Red, brown or black discolouration of the nails may indicate subungual haematoma. The cause may be acute trauma or chronic trauma such as pressure from ill-fitting shoes (Fig. 1.4a).

In acute ischaemia the nail beds are very pale (Fig. 1.4b).

Fig. 1.3 Corn on the 5th toe.

(a)



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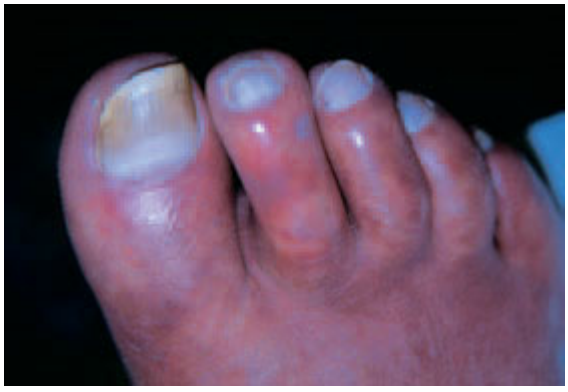


Fig. 1.4 (a) Subungual haematoma and red marks on toes resulting from wearing tight shoes. (b) Acute ischaemia—pale nail beds.

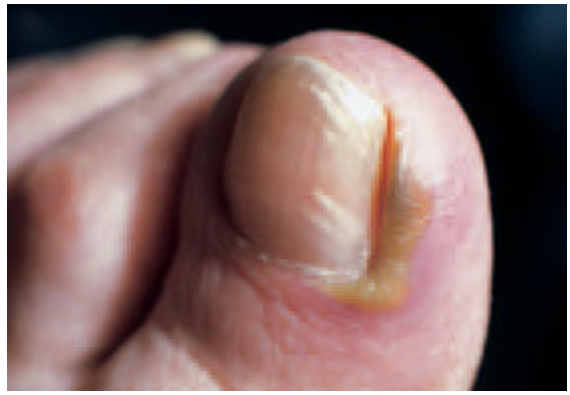


Fig. 1.5 Pressure on the sulcus from a convex nail has resulted in inflammation with secondary infection.

Abnormalities under the nail

Discharge of fluid from beneath or around the nail, and any maceration or softness of the nail plate, may indicate the presence of a subungual ulcer or infection.

Nail infections

Fungal infection of the nail usually invades the nail plate dorsally causing onycholysis. The hallux is the most common nail affected. Infection starts in one corner and over a period of years it spreads to involve the entire toe nail and may affect other nails.

Paronychia is associated with a nail that has a convex nail bed with tendency to incurve in the corners. Repetitive pressure in the insensitive foot can cause repetitive microtrauma in the nail groove, causing the nail to act as a foreign body creating a foreign body inflammatory response with secondary inflammation and localized infection (Fig. 1.5).

Swelling

Swelling of the foot is a major factor predisposing to ulceration, and often exacerbates a tight fit inside poorly fitting shoes. It also impedes healing of established ulcers. Swelling may be bilateral or unilateral. It may involve the foot or be limited to the toes.

Causes of bilateral foot swelling include:

- Cardiac failure
- Renal impairment secondary to diabetic nephropathy
- Chronic venous insufficiency (sometimes unilateral)
- Rarely, neuropathic oedema secondary to diabetic neuropathy, when it is related to increased arterial blood flow and arteriovenous shunting
- Primary lymphoedema

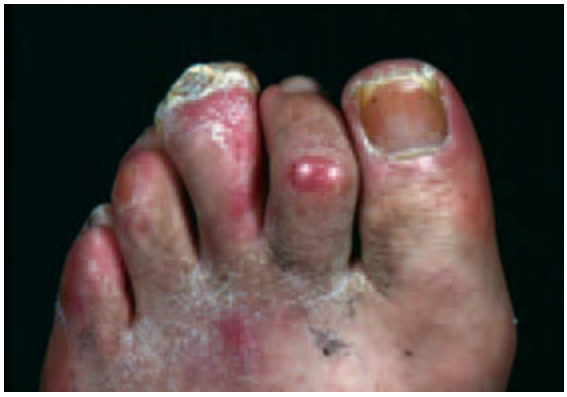


Fig. 1.6 Gout with tophi on second toe.

- Severe ischaemia associated with dependency (often unilateral).

Causes of unilateral foot swelling are usually associated with local pathology in the foot or leg. These include:

- Infection, when it is usually associated with erythema and a break in the skin
- Charcot foot (a unilateral hot, red, swollen foot; sometimes the swelling can extend to the knee)
- Gout, which may also present as a hot, red, swollen foot
- Trauma, fracture, muscle or tendon rupture, often associated with bruising
- Deep vein thrombosis
- Venous insufficiency
- Secondary lymphoedema commonly due to malignancy
- Common peroneal nerve palsy
- Localized collection of blood or pus in the foot, which may present as a fluctuant swelling
- Revascularization of a limb.

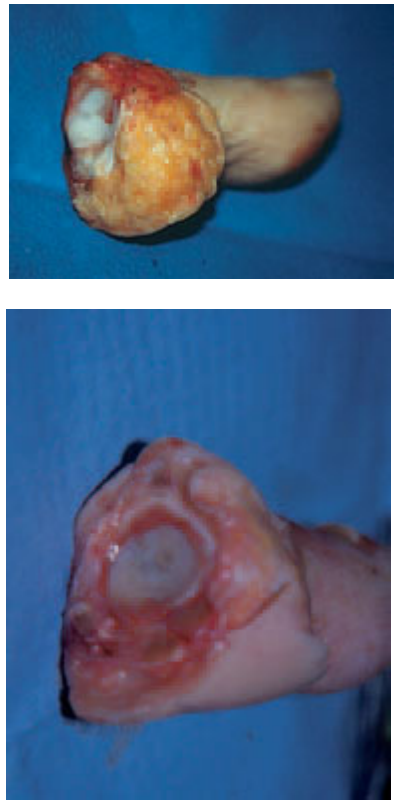
Swelling of the toe can be due to:

- Trauma
- Fracture
- Soft tissue infection
- Osteomyelitis
- Gout (Fig. 1.6)
- Charcot toe.

Deformity

Common deformities include:

- Pes cavus
- Fibrofatty padding depletion (FFPD)
- Hammer toes
- Claw toes
- Hallux valgus
- Charcot foot
- Deformities related to previous trauma and surgery.



(a)

(b)

Fig. 1.7 (a) Amputation specimen from 29-year-old non-diabetic patient showing thick fibrofatty padding. (b) Amputation specimen from 29-year-old diabetic patient with history of neuropathy and ulceration showing great reduction in fibrofatty padding.

Pes cavus

Normally the dorsum of the foot is domed due to the medial longitudinal arch, which extends between the first metatarsal head and the calcaneus. When it is abnormally high, the deformity is called pes cavus and leads to reduction of the area of the foot in contact with the ground during walking. Resulting abnormal distribution of pressure leads to excessive callus formation under the metatarsal heads. This deformity is a sign of a motor neuropathy but may be idiopathic. It is often associated with clawing of lesser toes or a trigger first toe.

Fibrofatty padding depletion (FFPD)

A common complication is reduction of the thickness of the fibrofatty padding over the metatarsal heads (Fig. 1.7a,b).

Normal feet contain cushions of fibrofatty padding over the metatarsal heads which absorb plantar pressures.

In diabetic neuropathy the fibrofatty padding may be pushed forward or depleted by previous ulceration, rendering the plantar metatarsophalangeal area prone to ulceration.

Hammer toe
A hammer toe is a flexible or rigid deformity characterized by buckling of the toe. The toe takes on the configuration of a swan's neck. In people with diabetic neuropathy, hammer toes are commonly caused by weakness of the small intrinsic muscles (interossei and lumbricals) of the foot, which can no longer stabilize the toes on the ground. Muscle imbalance results in the affected toes sitting slightly back and up on the metatarsal head. This deformity results in increased pressure over the metatarsal head, over the prominent interphalangeal joint and at the tip of the toe.

Claw toes
Claw toes are similar to hammer toes, but with more buckling and greater deformity. There is fixed flexion deformity at the interphalangeal joint, associated with callus and ulceration of the apex and dorsal aspect of the interphalangeal joint. Although claw toes may be related to neuropathy, they are often unrelated, especially when the clawing is unilateral and associated with trauma or surgery of the forefoot. Claw toes may rarely result from acute rupture of the plantar fascia.

Hallux valgus
Hallux valgus is a deformity of the first metatarsophalangeal joint with lateral deviation of the hallux and a medial prominence on the margin of the foot. This site is particularly vulnerable in the neuroischaemic foot and frequently breaks down under pressure from a tight shoe.

Charcot foot
Bone and joint damage in the tarsometatarsal joints and mid-tarsal joints leads to two classical deformities: the rockerbottom deformity, in which there is displacement and subluxation of the tarsus downwards, and the medial convexity, which results from displacement of the talonavicular joint or from tarsometatarsal dislocation. Both are often associated with a bony prominence which is very prone to ulceration and healing is notoriously difficult.

When the ankle and subtalar joints are involved, instability of the hindfoot can result.

Deformities related to previous trauma and surgery
Deformities of the hip and fractures of the tibia or fibula lead to shortening of the leg and abnormal gait, which predisposes to foot ulceration. Ray amputations remove the toe together with part of the metatarsal. They are usually very successful but disturb the biomechanics of the foot leading to high pressure under the adjacent metatarsal heads. After amputation of a toe, deformities are often seen in adjoining toes.

Limited joint mobility (including hallux rigidus)
Limited joint mobility can affect the feet as well as the hands. The range of motion is diminished at the subtalar and first metatarsophalangeal joints. Limited joint mobility of the first metatarsophalangeal joint results in loss of dorsiflexion and excessive forces on the plantar surface of the first toe, causing callus formation and ulceration. It is commonly seen in barefooted and sandal-wearing populations.

Colour
It is important to observe the colour of the foot including the toes. Colour changes may be localized or diffuse. Common colour changes are red, blue, white or black.

Causes of the red foot
- Cellulitis
- Critical ischaemia, especially on dependency (dependent rubor)
- Charcot foot
- Gout
- Burn or scald.

Causes of the red toe
- Cellulitis
- Osteomyelitis
- Ischaemia
- Gout
- Chilblains
- Dermatitis/eczema.

Causes of the blue foot
- Cardiac failure
- Chronic pulmonary disease
- Venous insufficency (often with brownish pigmentation —haemosiderosis).

Causes of the blue toe
- Severe infection
- Ischaemia.

The foot may have a pale white appearance in severe ischaemia, especially on elevation. In acute ischaemia, the foot is pale, often with purplish mottling. The cause of black appearances are discussed under necrosis.

Necrosis

Areas of necrosis and gangrene can be identified by the presence of black or brown devitalized tissue. Such tissue may be wet (usually related to infection) or dry.

Causes of the black toe

- Severe chronic ischaemia
- Acute ischaemia
- Emboli
- Bruise
- Blood blister
- Shoe dye
- Application of henna
- Tumour (melanoma).

Palpation

Palpation should take place to assess:

- Pulses
- Temperature of the foot
- Oedema
- Crepitus.

Pulses

The most important manoeuvre to detect ischaemia is the palpation of foot pulses, an examination which is often undervalued.

- The dorsalis pedis pulse is palpated, using the index, middle and ring fingertips together, lateral to the extensor hallucis longus tendon on the dorsum of the foot (Fig. 1.8)
- The posterior tibial pulse is palpated below and behind the medial malleolus (Fig. 1.9).

If either of these foot pulses can be felt then it is highly unlikely that there is significant ischaemia in the foot.

If both pulses are absent, a full examination should include palpation for popliteal and femoral pulses.

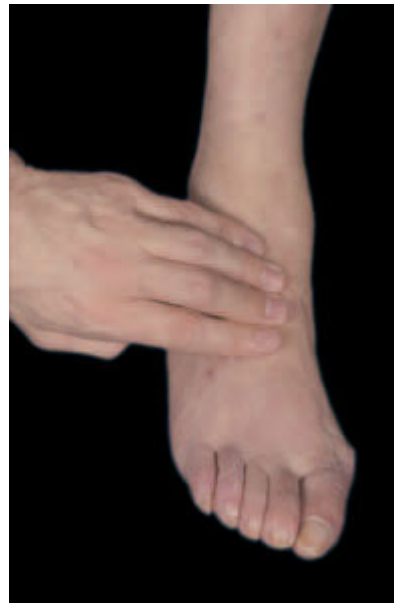


Fig. 1.8 Palpation of the dorsalis pedis pulse.

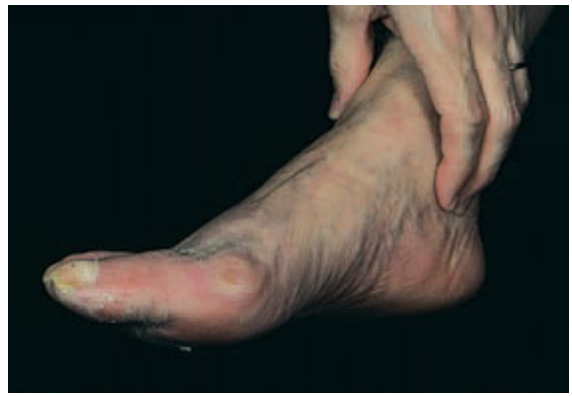


Fig. 1.9 Palpating the posterior tibial pulse.

Temperature of the foot

Skin temperature is compared between both feet with the back of the examining hand. Warm areas or hot spots indicate inflammation which may be due to infection, fracture, Charcot's osteoarthropathy or soft tissue trauma. Unilateral pedal temperature increase, especially in the absence of ulceration, is best presumed to be Charcot's osteoarthropathy.

The temperature gradient is checked by using the back of the hands and gently moving them from the pretibial region of the leg distally over the dorsum of the foot to the toes while keeping in contact with the patient's skin. An asymmetric gradient may indicate either unilateral ischaemia on the colder side or unilateral inflammatory response such as Charcot's osteoarthropathy or infection on the warmer side.

In the neuroischaemic foot, coexisting autonomic neuropathy may keep the foot relatively warm, although an ice-cold foot is indicative of acute ischaemia.

Causes of the hot foot

- Cellulitis
- Charcot foot
- Gout
- Venous insufficiency
- Deep vein thrombosis.

Causes of the cold foot
- Chronic ischaemia
- Acute ischaemia
- Cardiac failure.

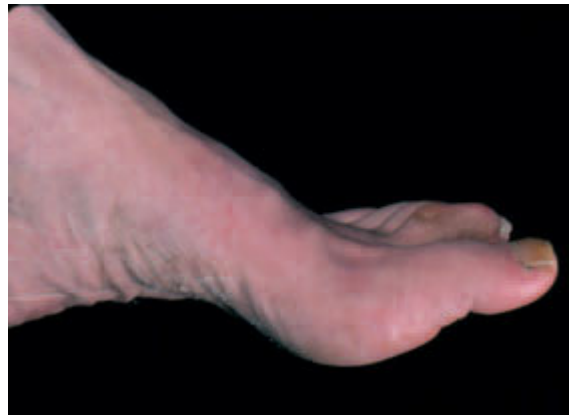
Oedema
Oedema already suspected on inspection can be confirmed by gentle digital pressure applied for a few seconds.

Crepitus
Very occasionally palpation may reveal gas in tissues as a fine crackling sensation.

Neurological assessment
Simple inspection will usually reveal signs of motor and autonomic neuropathy but sensory neuropathy must be detected by a sensory screening test or a simple sensory examination.

Motor neuropathy
The classical sign of a motor neuropathy is a high medial longitudinal arch, leading to prominent metatarsal heads and pressure points over the plantar forefoot (Fig. 1.10a,b).

Complicated assessment of motor power in the foot or leg is not usually necessary, but it is advisable to test dorsiflexion of the foot to detect a foot drop secondary to a common peroneal nerve palsy, which is usually unilateral and will affect the patient's gait. If painful mononeuropathy is suspected from the history, a more detailed neurological examination is indicated to rule out compressive lesions of nerve roots supplying the lower limb—see under Painful neuropathy in Chapter 3.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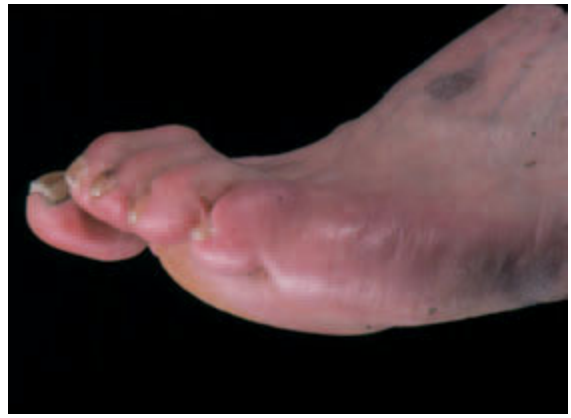
(b)

Fig. 1.10 (a) Neuropathic foot with high medial longitudinal arch. In severe cases, pressure points develop over the apices and dorsal interphalangeal joints of claw toes. (b) Claw toes in neuropathic foot.

Autonomic neuropathy
Signs of an autonomic neuropathy **in**clude a dry skin with fissuring and distended veins over the dorsum of the foot.

The dry skin is secondary to decreased sweating. The sweating loss normally occurs in a stocking distribution, which can extend up to the knee. The distended veins are secondary to arteriovenous shunting associated with autonomic neuropathy (Fig. 1.11).

Sensory neuropathy
An important indication of neuropathy will be a patient who has no pain even when significant foot lesions are present. Painless ulceration is definite evidence of a peripheral neuropathy. It is important to detect patients who have sufficient neuropathy to render them susceptible to foot ulceration. This can be carried out using a monofilament which, when applied perpendicular to the foot, buckles at a given force of 10 g. Ability to feel that level of pressure provides protective sensation against foot ulceration. It is helpful first to demonstrate the technique on the patient's forearm.

The number of sites used varies according to different protocols. Sites examined include the plantar aspects of the first toe, the first, third and fifth metatarsal heads, the plantar surface of the heel and the dorsum of the foot. The filament should not be applied at any site until callus has been removed. If the patient cannot feel the filament at any of the tested areas, then protective pain sensation is lost, indicating susceptibility to foot ulceration (Fig. 1.12). The 10-g monofilament may become overstrained and inaccurate after use on numerous occasions and should be replaced regularly. A recent study has assessed differences in the performance of commercially available 10-g

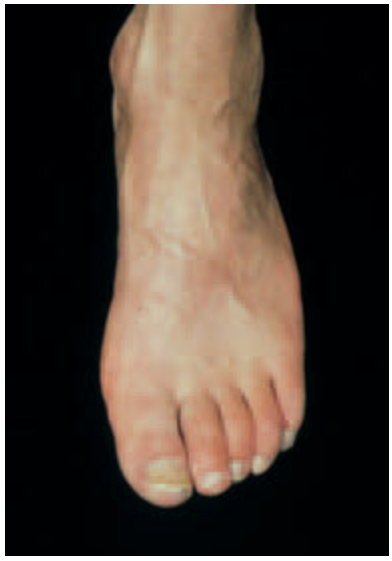

Fig. 1.11 Distended veins secondary to autonomic neuropathy.

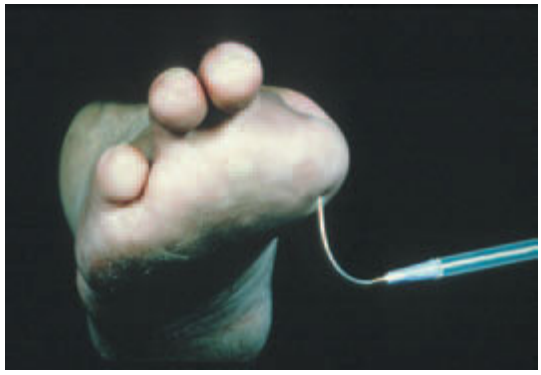

Fig. 1.12 A monofilament is applied perpendicular to the foot and pressed until it buckles at a given force of 10 g.

monofilaments. Monofilaments were tested using a calibrated load cell. Each monofilament was subjected to 10 mechanical bucklings of 10 mm while the load cell detected the maximal buckling force. Longevity testing was performed on a subset of the monofilaments by subjecting them to continuous compression until the buckling force was less than 9 g. Longevity and recovery testing suggest that each monofilament would survive usage on 10 patients before needing a recovery time of 24 h before further usage.

If filaments are not available, then a simple clinical examination detecting sensation to light touch using a cotton wisp and vibration using a 128-Hz tuning fork will suffice. It is best to compare a proximal site with a distal site to confirm a symmetrical stocking-like distribution of the neuropathy, and to avoid the use of 'pin-prick' to detect sensory loss.

Other useful but simple and practical tests for detecting neuropathy, if health-care practitioners have no access to formal equipment, include the Achilles tendon pinch, and the application of vertical pressure onto the nail plate. In practice, any patient who walks on a foot with ulceration or heavy plantar callus without concern has significant neuropathy.

Footwear assessment

It is important to examine both shoes and socks.

Examination of patient's footwear

- Is the shoe long enough?
- Is the toe box broad and deep enough?
- Are the heels low?
- Does the shoe fasten with a lace or strap?

Slip-ons are unsuitable for everyday wear.

- Is the sole thick enough to provide protection from puncture wounds?
- Is the shoe lining worn, with rough areas that may prove irritating and warrant replacement?
- Are there foreign bodies within the shoes?
- Is there excessive wear under hallux suggesting a hallux rigidus?
- Is there wear across whole of tread suggesting pes cavus?

Examination of patient's socks

- Are the socks large enough?
- Are the seams too prominent?
- Is there a tight band at the top?
- Are the socks in good repair—no holes or lumpy darns?
- Are the socks made of absorbent material?
- Are the socks very thick, taking up too much space in the shoe?

General examination

As part of the diabetic foot assessment and indeed the diabetic assessment all patients should have a physical examination including the following systems:

- Cardiovascular
- Respiratory
- Abdomen
- Eyes:
  Visual acuity
  Fundi.

(A patient lacking necessary visual acuity to give himself a

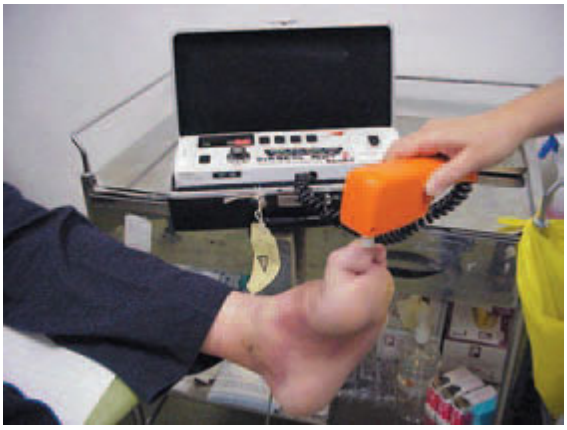

Fig. 1.13 The neurothesiometer.

daily foot examination is a patient at risk, and his family or caregiver should help him.)

Investigations

Investigations include:
- Neurological
- Vascular
- Skin temperature
- Laboratory
- Radiological
- Foot pressures.

Neurological

The degree of neuropathy can be quantified by the use of the biothesiometer or the more recently available neurothesiometer (Fig. 1.13). Both are devices which, when applied to the foot, deliver a vibratory stimulus, which increases as the voltage is raised. The vibration threshold increases with age, and for practical purposes, any patient unable to feel a vibratory stimulus of 25 volts is at risk of ulceration.

A small number of patients have a small-fibre neuropathy with impaired pain and temperature perception but with intact touch and vibration. They are prone to ulceration and thermal traumas but test normally with filaments and biothesiometer, and a clinical assessment of light touch and vibration is normal. As yet, there is no simple inexpensive method of detecting and quantifying small-fibre neuropathy. However, a simple temperature assessment of cold sensation can be made by placing a cold tuning fork on the patient's foot and leg.

Vascular

A small hand-held Doppler can be used to quantify the vascular status.

Used together with a sphygmomanometer, the brachial systolic pressure and ankle systolic pressure can be measured, and the pressure index, which is the ratio of ankle systolic pressure to brachial systolic pressure, can be calculated. In normal subjects, the pressure index is usually > 1, but in the presence of ischaemia is < 1. Thus, absent pulses and a pressure index of < 1 confirms ischaemia. Conversely, the presence of pulses and a pressure index of > 1 rules out ischaemia and this has important implications for management, namely that macrovascular disease is not an important factor and further vascular investigations are not necessary.

Many diabetic patients have medial arterial calcification, giving an artificially elevated systolic pressure, even in the presence of ischaemia. It is thus difficult to assess the diabetic foot when the pulses are not palpable, but the pressure index is > 1. There are two explanations.
- The examiner may have missed the pulses, particularly in an oedematous foot, and should go back to palpate the foot after the arteries have been located by Doppler ultrasound
- If the pulses remain impalpable, then ischaemia probably exists in the presence of medial wall calcification. It is then necessary to use other methods to assess flow in the arteries of the foot, such as examining the pattern of the Doppler arterial waveform or measuring transcutaneous oxygen tension or toe systolic pressures. Furthermore, absence of foot pulses would be an indication to investigate popliteal and femoral arteries.

Skin temperature

It is helpful to follow-up the clinical assessment of skin temperature with the use of a digital skin thermometer. An infrared thermometer is ideal and skin temperatures are compared between similar areas on each foot (Fig. 1.14). This is particularly helpful in the management of the Charcot foot.

Laboratory

Laboratory investigations are determined by clinical findings, but the following investigations are useful as a baseline in most patients:
- Full blood count (to detect anaemia or polycythaemia)
- Serum electrolytes, urea and creatinine (to assess baseline renal function)
- Serum bilirubin, alkaline phosphatase, gamma glutamyl transferase, aspartate transaminase (to assess baseline liver function)