The ABCS of foot care in diabetes: A is for anaesthesia

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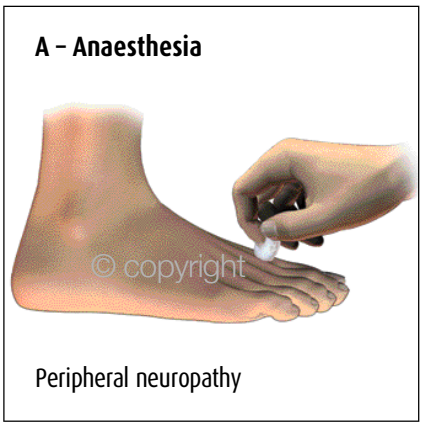
This second article on risk factors for foot problems in people with diabetes discusses the various aspects of peripheral neuropathy and provides guidelines for patients on caring for feet with nerve damage.



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The ABCS of foot care refer to the major risk factors for foot problems in people with diabetes – A, anaesthesia (i.e. peripheral neuropathy); B, blood supply (i.e. peripheral vascular disease); C, care (i.e. routine preventive foot care); and S, structure (i.e. abnormal foot structure). This article reviews the first of these, peripheral neuropathy, and includes a patient handout on nerve damage. The previous article in this series, published in the November 2008 issue of *Medicine Today*, discussed the assessment of the podiatric ABCS, and future articles will discuss the other three risk factors.[1] A patient handout on foot care for people with diabetes, 'Your foot report', will conclude the series.

The foot factor traffic lights

A person with diabetes and normal sensation and circulation in their feet and normal foot structure needs the same foot care and footwear as a person without diabetes. But if the person has one or more of the risk factors for foot problems, more intense foot care and monitoring and special footwear will probably be necessary. This will reduce the likelihood of a problem developing and enable detection of problems that do occur so intervention can be prompt.

The podiatric ABCS can be thought of as a series of traffic lights: if all four are green then there is little risk of foot problems, but the risk increases steadily as more of the lights turn yellow and then red. Table 1 summarises the principle.[1] When there has been sufficient long-term damage to the foot to break the skin and form an ulcer, tissues are at risk of infection. The occurrence of a foot ulcer signals a very high risk of further ulcers and should trigger a review of all the ABCS and the patient's understanding and application of appropriate protection, detection and response plans. Referral to a specialist foot clinic should be considered, if one is available.

Peripheral neuropathy

Several clinical syndromes are associated with peripheral neuropathy in patients with diabetes, and patients often have multiple or overlapping syndromes. Sensory loss (anaesthesia) associated with diabetic peripheral neuropathy becomes progressively more common with increasing duration of diabetes (Figure 1).[2] Early sensory nerve damage is often associated

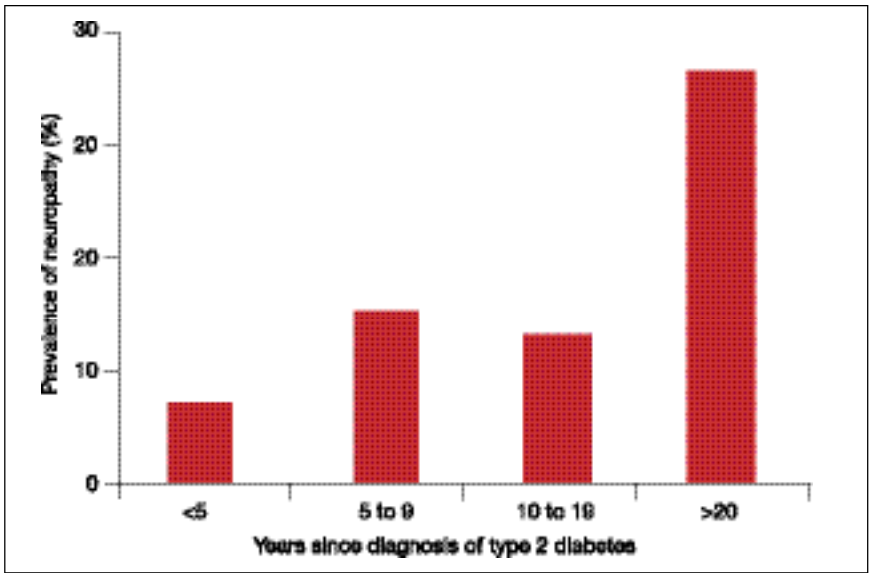

Figure 1. Prevalence of diabetic neuropathy.[2]

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Table 1. Foot factor traffic lights and patient risk assessment

Foot factor traffic lights

Foot factor assessment	Red lights – 'Danger'	Amber lights – 'Caution'	Green lights – 'Healthy'
Anaesthesia – Pinprick, light touch – Reflexes	 No stimuli felt No reflexes	 Reduced stimuli Reduced reflexes	 All stimuli felt Normal reflexes
Blood supply – Pulse palpation	 No pulses	 Reduced pulses	 Normal pulses
Care – Questioning – Observation	 –* Skin breakdown	 Foot care, footwear could be better Threatened skin breakdown	 Appropriate foot care, footwear Normal skin
Structure – Observation	 Weight-bearing ulcer	 Callus or corn	 No skin lesions

Patient risk assessment

Traffic lights	Risk assessment	Recommended action
One or more 'red lights'	High risk	Refer promptly to a podiatrist
One or more 'amber lights'	Moderate risk	Regular podiatry care and assessment
All 'green lights'	Low risk	General foot care advice

* Inadequate foot care in the absence of any other red or amber traffic lights is not a major risk factor for severe foot problems.

with abnormal sensation (dysaesthesia) as well as anaesthesia. As the neuropathy progresses, anaesthesia dominates and two other syndromes – motor neuropathy and autonomic neuropathy – become clinically significant. Motor neuropathy with skeletal muscle atrophy and weakness is associated with secondary foot deformities. Autonomic neuropathy has a wide range of manifestations but most often causes dry skin and peripheral oedema because of damage to the sympathetic nerve fibres that supply the sweat glands and arterioles in the skin.

The patterns of progression of diabetic peripheral neuropathy and of symptoms are very variable. Generally, however, neuropathy progresses, although the four components (dysaesthesia, anaesthesia, motor neuropathy and autonomic neuropathy) may not progress at the same rate.

This article reviews the four aspects of diabetic peripheral neuropathy and discusses their implications for general practice. The assessment of peripheral neuropathy was discussed in detail in the previous article in this series.[1] Briefly, sensation is checked by light touch (with cottonwool or, ideally, a 10 g monofilament) and abnormalities are confirmed by checking for sensation to pinprick and checking reflexes.

Abnormal sensation

'At night armies of ants march up and down my legs. Sometimes the ants decide to burn instead and my legs feel like they are on fire.'

Annie, who has had type 2 diabetes for five years.

The presence of abnormal sensations is the most distressing symptom of neuropathy for patients and may limit their activities during the day and their sleep during the night. The sensations include burning, prickling pain, tingling, electric shock-like feelings, aching, tightness and hypersensitivity to touch. As the neuropathy progresses, the sensations also include numbness, and loss of balance and painless injuries may occur.

The disturbed sensations are caused by early damage to sensory (or afferent) nerves, with the healing process causing nerve fibres (axons) from one sensory receptor to connect to fibres that previously carried signals from a different sensory receptor. Usually this starts in the small unmyelinated or lightly myelinated fibres of the afferent nerves in the distal lower limb, which are particularly susceptible to damage because of their long length. The nerve damage results from prolonged hyperglycaemia leading to accumulations of metabolites in neurons and the nerve fibres ultimately loosing the ability to conduct electrical impulses. The severity of the abnormal sensations is, therefore, generally related to the degree of hyperglycaemia. Improving glycaemic control may, therefore, improve symptoms.

Abnormal sensation may not be limited to the lower limbs. As the nerve

damage progresses, the upper limbs may become affected (the so-called 'glove and stocking' distribution, which describes the lesser involvement of the upper limbs compared with the lower limbs).

Sometimes there is the 'double whammy' of pressure and diabetes-related damage for the nerves that negotiate various 'tunnels' – for example, the median nerve at the wrist, the ulnar nerve at the elbow and possibly the peroneal nerve at the knee. For these nerves, a single cause of damage may not cause problems but combination does.

Nondiabetes-related causes of neuropathy are listed in Table 2. The double whammy of diabetes-related and nondiabetes-related causes of neuropathy can be associated with any or all of the clinical syndromes occurring with diabetic peripheral neuropathy.

In the early stages of dysaesthesia, discomfort from painful neuropathy can be an occasional nuisance when a benign stimulus (such as putting on a sock) results in a disturbing sensation or pain. In the later stages, the pain often becomes more generalised and more severe, typically occurring at night and keeping patients awake.

Patients may find ways to relieve the discomfort by trial and error. Sleeping with the feet outside the covers is often reported, and is effective because the feet become cold and numb and the covers no longer stimulate the skin. A range of complementary medicines is often tried, especially vitamin preparations. However, in the absence of a specific deficiency, vitamin supplements have not been shown to be effective in clinical trials.

Fortunately some interventions do help at least some people (Table 3). They seem to work in various ways, including by:

- reducing the perception of abnormal sensation – e.g. tricyclic antidepressants and transcutaneous electrical nerve stimulation (TENS)
- reducing receptor responses to stimuli – e.g. capsaicin (Zostrix HP), which depletes substance P, a neurotransmitter and neuromodulator in pain perception
- putting a physical barrier between the skin and the environment – e.g. a film dressing such as Opsite Spray, or silicone stockings.

Combining interventions is much more effective that any single intervention alone.

Various other prescription medications are promoted as being effective, such as aldose reductase inhibitors, but evidence for their effectiveness in peripheral diabetic neuropathy is lacking.[3] Apart from potential side effects, financial costs for the patients may be considerable if the medications are not subsidised by the PBS for painful diabetic neuropathy.

Table 2. Nondiabetes-related causes of peripheral neuropathy

- Nutrition, ethanol, vitamin deficiency (thiamin, vitamin B_{12} and folic acid, especially with the 'tea and toast' diet of many older people)
- Medical problems – especially entrapment neuropathies (e.g. carpal tunnel syndrome); rarely myeloma, hypothyroidism, paraneoplastic disorders
- Medications (e.g. sulfasalazine, perhexiline, isoniazid)

Note: Radicular leg pain caused by lumbar spine/disc disease has a different distribution: unilateral and in the nerve root distribution. Pain may occur at the appropriate level of the back.

Table 3. Intervention options for painful neuropathy

Local measures

- Film dressing (Opsite Spray) – wrapping the foot reduces sensory stimulation and dysaesthesia
- Capsaicin (Zostrix HP) 0.075% cream three to four times per day – depletes skin substance P, a neuropeptide involved in pain perception
- Transcutaneous electrical nerve stimulation (TENS) – may reduce central appreciation of the abnormal nerve impulses

Systemic measures

- Antidepressants – low-dose tricyclics such as amitriptyline (Endep), 25 to 150 mg at night, are traditional; selective serotonin re-uptake inhibitors may also be effective
- Nerve stabilisers – antiepileptics can be effective, particularly gabapentin (e.g. Neurontin) 900 to 3600 mg per day; in severe cases, a combination of gabapentin and up to 20 mg per day of oxycodone (Endone, OxyContin, OxyNorm) should be trialled; pregabalin (Lyrica) may also be used

Loss of sensation

'I feel like I walk on cotton wool. Sometimes my feet are like slabs of wood and just slap down on the ground.'

Annie, six years later.

The sensations of walking on cotton wool or with feet that slap down on the ground like pieces of wood may be disturbing for patients but are usually much less distressing than abnormal painful sensations. Patients may prefer loss of sensation but they should be aware that the change from dysaesthesia to anaesthesia marks progression of nerve damage, and not improvement.

Anaesthesia is the major contributor to the foot problems that rank second only to cardiovascular disease in terms of direct health care costs in patients with diabetes.[4] The 'high profile' diabetic complications of nephropathy and retinopathy cost less than foot problems (largely ulcers) and much less than cardiovascular disease (including peripheral vascular disease).

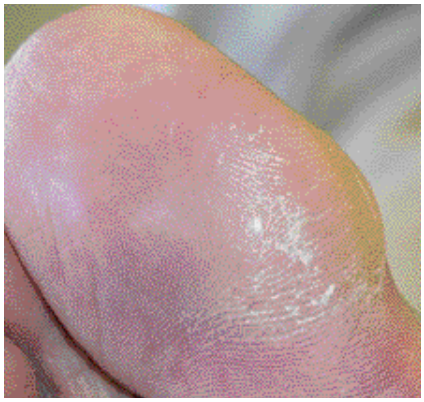

Figure 2. Callus can be a sign of excess pressure that has not been noticed through the sensation of pain by a person with peripheral neuropathy.

Loss of sensation means that the risk of foot problems has increased considerably. The patient may have lost the discomfort but is now unable to detect damage to the foot and has therefore lost the normal protection against injury. Once a patient has lost sensation, he or she and his or her carers must establish systems to protect the feet, detect foot problems early, and respond promptly and appropriately. Patients should be advised about:

- protection – appropriate foot care and footwear
- detection – a daily routine of checking footwear and foot health
- response – clear indications for action and contacting health professionals.

Redness of the skin is an early signal that extra foot protection is needed, and thickened skin (callus) or thickened nails are later signs of excess pressure that does not cause the pain that would have prompted preventive action to reduce the risk of ulceration in a person with normal sensation (Figure 2).

The patient handout on page 67 provides guidance for patients who have sensory loss. It may be that a carer or visiting health professional is the appropriate person to be informed if the patient is not physically and/or mentally able to understand the requirements of adequate foot care, provide the necessary care or respond to early problems.

All too often preventable precipitants cause problems that are not detected and progress to disasters. For example, educating a patient and/or his or her carer may prevent a drawing pin piercing the sole of a slipper and entering the foot, causing considerable tissue damage and facilitating an infection that may spread to the deeper tissues and cause life-threatening sepsis requiring a below-knee (or, even worse, above-knee) amputation. This scenario is illustrated by the story of Amanda in the box on this page. It should be remembered that most nontraumatic lower extremity amputations are caused by diabetes, and that most are associated with sensory loss and are potentially preventable.[2]

Motor neuropathy

'My feet have become so ugly, my toes are like claws and the tops are red and callused from rubbing on my shoes.'

Annie, a few years later.

The motor (or efferent) nerves supplying skeletal muscle are made up of large, heavily myelinated nerve fibres and are affected later in the progression of diabetic peripheral neuropathy than the smaller non- or less-myelinated fibres for sensations and autonomic function. However, the gamma motor fibres (also known as gamma efferent fibres), which regulate the stretch reflex responses of muscles, are small, lightly myelinated fibres that are affected quite early in the process of neuropathy.

Once pain sensation has been lost, it is likely that motor neuropathy will be present. Loss of peripheral tendon reflexes is an early sign, with atrophy and weakness occurring much later. In advanced neuropathy, the toe flexors become dominant and fix the toes in flexion, forming clawed toes (Figure 3).

As motor neuropathy progresses, patients may notice weakness. This may be particularly apparent in the hand, where a weak handgrip can make opening jars or bottles difficult. Changes in foot structure can cause changes such as thickening of the skin and nails of the feet from pressure. The clawed toes and loss of foot arches redistribute the load on the foot, concentrating the load on the metatarsophalangeal area and the tips of the toes. The dorsal surface of the toes may also rub on the underside of the shoe upper. Callus is likely to form over the raised metatarsal heads, and dorsal toe ulceration may result.

Fortunately, clinically significant consequences of motor neuropathy occur late in the course of peripheral diabetic neuropathy. Unfortunately though, when they do occur they can be associated with considerable discomfort and functional

Prevention of diabetic complications is better than treatment

The story of Amanda's left foot

Amanda was diagnosed with type 2 diabetes about 10 years ago and now has no sensation in her feet but as yet not extensive peripheral vascular disease.

Monday: Shopping for new shoes.

Tuesday: Guided tour of the botanical gardens wearing new shoes; blister on left big toe noticed that evening.

Wednesday: Left foot redness and discharge noted, with discovery of a new 'sore' on the sole of the left foot.

Thursday and Friday: Worsening of swelling and redness, development of fever, GP review and referral to hospital.

Friday: Left below-knee amputation.

Most nontraumatic lower extremity amputations in patients with diabetes are associated with sensory loss.[2] In this case, although the patient had normal circulation, severe infection in the lower limb required below-knee amputation because there was extensive tissue sepsis/necrosis.

disability. Podiatrists and occupational therapists can offer useful advice to patients, including appropriate footwear and orthotics and various aids to improve functional capacity. The more abnormal the foot structure, the more important specialised assessment and individualised footwear and orthotics become.

Autonomic neuropathy

'I use special soap and moisturiser and still my skin is dry and cracked and itches like mad. I just can't stop scratching, and of course that makes things worse.'

Annie, in response to your suggestions about foot care.

The postganglionic efferent fibres of autonomic nerves are small and unmyelinated. Like the sensory nerve fibres and the gamma motor fibres, these autonomic nerve fibres are also affected early in peripheral diabetic neuropathy. They are affected in the lower limb before the upper limb, and distally before proximally.

As peripheral neuropathy progresses, autonomic control of the sweat and sebaceous glands is lost and the skin becomes dry and less flexible. Drying of the skin of the feet may be hastened in patients who wear open shoes or thongs.

Dry and cracked skin often becomes uncomfortable and itchy. Scratching, however, only aggravates the damage and itchiness. Patients may find that wetting the skin is the only thing that relieves their discomfort. But this too aggravates the problem, accelerating the drying process. The 'itch/scratch' and 'dry/wet' cycles can be very difficult to break. Breaking the cycles early not only provides symptomatic relief but also reduces the risk of fungal or bacterial infections occurring once the skin is damaged. The principles of caring for dry skin are listed in Table 4 and also summarised in the patient handout on page 67.

The loss of the smooth muscle nerve supply rarely causes obvious problems. The erection of hairs under the control of the sympathetic nervous system acting on the erector pili muscles is gone, but this is not particularly a cause of concern. The dilation of the arterioles within the foot caused by loss of control of the arteriolar smooth muscle may, however, produce postural hypotension or reduce tissue perfusion if another contributor (such as hypotensive medication or arterial disease) is present. Arteriolar dilation also increases capillary and venular pressure. This increases plasma filtration into the interstitial space and may cause oedema. Venules and veins also dilate, sometimes spectacularly – as shown in Figure 3.

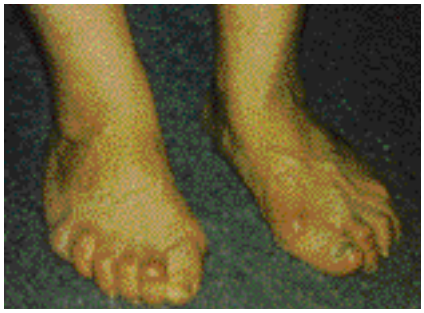

Figure 3. Clawed toes are a sign of advanced motor neuropathy. The toe flexors have become dominant and fixed the toes in flexion. Note the dilated veins caused by autonomic neuropathy.

Patients with type 2 diabetes often already have reasons for peripheral oedema, such as overweight/obesity, incompetent veins, cardiac and/or renal dysfunction and medications such as peripheral calcium-channel blockers. Although it is tempting to manage this fluid imbalance with a loop diuretic, this is not the best option. A diuretic dose in the morning will provide diuresis, but fluid will accumulate later in the day. Further diuretic doses may also have gratifying effects but are still unlikely to control the problem.

As local pressure effects are causing the problem, the use of support stockings is a logical and effective measure for oedema. The stockings should be worn all day and, for best effect, should be put on before getting out of bed in the morning. Patients should be encouraged to shower in the evening as having a morning shower before putting on the stockings allows fluid to accumulate in the unsupported tissues and reduces the effectiveness of the stockings. The help of a carer or a device such as the Ezy-As compression stocking applicator may be needed to get the stockings on.[5] Fortunately, medium strength knee-high support stockings are usually

Table 4. Caring for dry skin

Washing

- Limit frequency
- Use minimal amounts of tepid (not hot) water
- Only soap the areas that need cleaning, e.g. groin and axillae every two to three days, hair weekly
- Moisturise the skin after bathing – a helper may be needed to get to inaccessible areas such as the back

Moisturisers

- Use moisturisers frequently
- Choose the thicker preparations that are still easily spread (thicker preparations have lower water and higher fat content)
- Consider the more expensive but more effective moisturisers that contain urea and/or organic acids if the skin on the heels is cracked. Urea-containing moisturisers specifically indicated for the feet include DermaDrate Cracked Heel Treatment, Eulactol Heel Balm and FootSmart

Clothes and footwear

- Wear clothes made of materials containing non-irritant fibres (e.g. cotton and cotton/polyester mixes); avoid totally synthetic materials as they are too hot and trap moisture
- Wear closed shoes made of leather or canvas (i.e. not synthetic materials), and socks made with at least some natural fibre (e.g. cotton or wool)

adequate, and are not too difficult to put on; full length or firmer stockings can be very difficult.

Patients who are concerned about the stockings being uncomfortable or hot should be reassured that the reduced leg swelling resulting from correctly used stockings increases leg comfort and that not much heat is lost through the lower limbs anyway. The additional protective benefits of stockings should be stressed to patients. Stockings protect the skin of the legs against knocks that might otherwise break the skin and lead to sores that may be slow to heal and might leak oedema fluid, further impairing healing. (Before effective diuretics became available one treatment for severe peripheral oedema was to insert tubes [Southey tubes] into the tissues of the lower limb to drain the oedema fluid.)

Advanced peripheral neuropathy

As noted earlier, diabetic peripheral neuropathy is associated with progression from the small, unmyelinated or lightly myelinated pain and temperature afferent fibres, gamma motor fibres and autonomic nerve fibres being damaged to the larger, more myelinated afferent fibres and the large, heavily myelinated efferent fibres becoming damaged. Once the sensations of touch and pressure are affected (ideally assessed by a monofilament), effects on muscle function as well as loss of proprioception should be expected.

The distribution of the neuropathy also progresses, moving proximally up the lower limbs and then the upper limbs. As a rough guide, neuropathy will start to affect the fingers when neuropathic signs are detected in the mid-calf of the leg, and earlier if the double whammy of pressure and diabetes-related damage has occurred at the wrist or elbow. As the neuropathy spreads to affect other sensory and motor modalities and other parts of the body, a wide range of clinical symptoms, signs and problems can occur, including the neuroarthropathies. These however, apart from a brief discussion of Charcot's neuroarthropathy, are beyond the scope of this article.

It is important to remember that all neuropathy occurring in a person with diabetes may not be caused by the diabetes. Neuropathy in the absence of other microvascular complications or rapid progression should prompt review of other potential contributors (Table 2). Referral of the patient for a second opinion and advice on further management should be considered.

In the final stages of neuropathy, the so-called 'Charcot foot' develops, in which the function of the foot in absorbing and transmitting load is lost. The whole foot and most or all of the lower leg becomes insensate, the foot arches collapse and the toes claw. Gait is disturbed by the loss of normal foot structure, the loss of protective reflexes, the loss of position sensation and co-ordination and the loss of motor function. The patient walks with a slow, high-stepping, foot-slapping gait that further destroys foot structure and causes recurrent lesions in areas of high load. Bisphosphonates may slow the progressive bone destruction and prevent clinical fractures.

A Charcot foot is an extreme risk for future problems, particularly undetected plantar ulcers and fractures – both of which can present as a painless but acutely swollen red foot. Referral to a specialist foot clinic will give the patient access to the necessary protective footwear, orthotics and total contact casting, and to ongoing specialised monitoring and management. Early consideration of the possibility of the occurrence of neuroarthropathy and early referral give the best outcomes.

Conclusion

Four syndromes associated with peripheral neuropathy pose special problems for patients with diabetes and the health professionals caring for them. These syndromes and the problems are:

- abnormal sensation
  - coping with the discomfort
  - being aware that a loss of the discomfort is not a good sign
- loss of sensation
  - developing strategies to avoid excess pressure, systems to monitor foot health and action plans for potential problems
- motor neuropathy
  - dealing with secondary deformities, clawing of toes, flattened feet
  - developing strategies for activities of daily living affected by loss of strength
- autonomic neuropathy
  - maintaining skin health
  - dealing with peripheral oedema.

It should be remembered that all the conditions that can be caused by diabetes may not actually be caused by diabetes. Therefore, other causes should be considered before attributing neuropathy to diabetes. MT

References

1. Phillips P, Evans A. The ABCS of foot care in diabetes: assessing the risk factors. Medicine Today 2008; 9(11): 57-61.
2. Tapp RJ, Shaw JE, de Courten MP, et al; AusDiab Study Group. Foot complications in Type 2 diabetes: an Australian population-based study. Diabet Med 2003; 20: 105-113.
3. Chalk C, Benstead TJ, Moore F. Aldose reductase inhibitors for the treatment of diabetic polyneuropathy. Cochrane Database Syst Rev 2007; Issue 4: CD004572.
4. Hogan P, Dall T, Nikolov P; American Diabetes Association. Economic costs of diabetes in the US in 2002. Diabetes Care 2003; 26: 917-932.
5. Ezy As Compression Stocking/Garment Applicator. Website: http://www.ezyasabc.com (accessed November 2008).

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