Making diabetic foot care evidence-based: what is missing?

Part 1: Screening and prevention

Dr Peter Cavanagh

Peter R Cavanagh PhD, DSc (Med) is Academic Director of the Diabetic Foot Care Program at the Cleveland Clinic Foundation, USA

Some thought leaders believe that we are about to leave the era of evidence-based medicine and enter the uncharted waters of genome-based medicine (Guttmacher and Collins, 2003). Although the first paper that I am aware of that discusses the genetics of foot ulcer healing was presented at the recent American Diabetes Association meeting in Orlando, Florida (Zimny et al, 2004), I would propose that, in many respects, care of the diabetic foot never really entered the evidence-based era. A recent high-profile article in *The Lancet* by Jeffcoate and Harding (2003) described diabetic foot care in general as a 'clinical practice . . . based more on opinion than scientific fact' - a sentiment that I heartily endorse.

This column gives me the opportunity to blow off some steam about what I perceive to be the lack of evidence (or sometimes an ignoring of evidence) that guides much of the assessment and treatment in our field. I have divided the terrain into screening, prevention, and treatment. I will address the first two categories in this piece and will discuss evidence-based treatment in the next edition of *The Diabetic Foot*.

Screening

Peripheral neuropathy and vascular disease are the two principal conditions associated with diabetic foot disease, and the most common approaches to assessment of these conditions in a practice setting are monofilament testing and detection of pulses. Experiments by Booth and Young (2000) in the UK and our own experiments in the USA have shown that the monofilament is a highly unreliable test to be used for such a critical task. In a controlled setting, monofilaments from different manufacturers can differ by as much as 15% in the forces they apply, and these forces decrease markedly with repeated use. But typical clinic use is far from controlled, and Bell-Krotoski and Buford (1997) have shown that the force varies depending on the speed with which the monofilament is bent or bounced against the skin.

Similarly, we all believe that the presence or absence of posterior tibial (PT) or dorsalis pedis (DP) pulses are indications of vascular disease. But a study by Magee et al (1992), now more than a decade old, found only 67% agreement between a group of four experts in establishing the presence or absence of the DP pulse and only 53% agreement in the PT – about the same as flipping a coin! Unfortunately, very few people seem to take notice of this evidence, and we all treat the monofilament as if it were as reliable as the caesium clock and the palpation of pulses as some sacred rite.

Prevention

Good primary prevention depends not only on good screening, but also on well-established and validated interventions. The prescription of footwear, as we shall discuss in the second article, is a highly subjective area and the practitioner has very few quantitative rules on which to base his or her product. Until we understand more about how to match a shoe to a particular foot, and then test this targeted prescription in a controlled study, the notion that footwear prevents the first foot ulcer will remain a strongly-held opinion rather than a demonstrated fact.

There are some serious foot complications for which we are completely unable to provide primary prevention, the most important being Charcot's neuroarthropathy. It is remarkable that more than a century after Charcot identified this condition, the aetiology is very poorly understood. Consequently, prevention (and treatment) is really a matter of individual trial and error. Even the latest studies in this area (Petrova et al, 2004) are still adding small pieces to a presently unfathomable puzzle.

The most compelling question in secondary prevention of foot ulcers is: why are our recurrence rates so high? Various studies have placed the rates at between 28% at 12 months (Uccioli et al, 1995) and 100% at 40 months (Chantelau et al, 1990). My own supposition is that these high failure rates arise from a combination of poor medical care (in the form of inappropriate footwear), poor self care (with lack of compliance to prescribed footwear high on the list) and inadequate or absent patient education (for example, Vileikyte et al [2001] reported that patients have many misconceptions about neuropathy). But there are no studies that help us understand which patients will have recurrent ulcers and which will remain ulcer free. We do know that even patients with active ulcers are extremely non-compliant when asked to wear a protective device (Armstrong et al, 2003). Where are the

controlled studies of different approaches to patient education and behaviour change as they relate to diabetic foot care?

There has been a growing trend in the last decade towards prophylactic surgery as a means of secondary prevention. This approach could be characterised as one that modifies the foot rather than accommodates it with footwear. The field of surgery has different standards of evidence compared, for example, with pharmacological intervention, and there have been no studies where patients have been randomised to either surgery or conservative treatment. But some of the evidence from the available surgical studies on diabetic feet suggests that a much more careful look at the complication rates is warranted. Fleischli et al (1997) reported a 68% complication rate in a series of 22 metatarsal osteotomies in which the most common complication was a Charcot fracture 'with rapid destruction of the midfoot'. The question of whether or not the surgery induced the Charcot fractures needs to be urgently explored.

Outlook

My focus on the absence of evidence should not be taken as a blanket indictment of the field. Clearly, much excellent care is delivered, and many limbs are saved by caring clinicians using the best available knowledge. But I hope the issues discussed here and in the next editorial will identify some constructive directions for research that could be conducted to provide the scientific foundation for a more evidence-based future for diabetic foot care. ■

Armstrong DG, Lavery LA, Kimbriel HR, Nixon BP, Boulton AJ (2003) Activity patterns of patients with diabetic foot ulceration: patients with active ulceration may not adhere to a standard pressure off-loading regimen. *Diabetes Care* **26**(9): 2595–97

Bell-Krotoski JA, Buford WL, Jr (1997) The force/time relationship of clinically used sensory testing instruments. *Journal of Hand Therapy* **10**: 297–309

Booth J, Young MJ (2000) Differences in the performance of commercially available 10-g monofilaments. *Diabetes Care* **23**: 984–88

Chantelau E, Kushner T, Spraul M (1990) How effective is cushioned therapeutic footwear in protecting diabetic feet? A clinical study. *Diabetic Medicine* **7**: 355–59

Fleischli JG, Lavery LA, Vela SA, Ashry H, Lavery DC. (1997) Comparison of strategies for reducing pressure at the site of neuropathic ulcers. *Journal of the American Podiatric Medical Association* **87**:466–72

Guttmacher AE, Collins FS (2003) Welcome to the genomic era. *New England Journal of Medicine* **349**: 996–98

Jeffcoate WJ, Harding KG (2003) Diabetic foot ulcers. *Lancet* **361**: 1545–51

Magee TR, Stanley PR, al Mufti R, Simpson L, Campbell WB (1992) Should we palpate foot pulses? *Annals of the Royal College of Surgeons (Engl)* **74**: 166–68

Petrova NL, Foster AVM, Edmonds M (2004) Is there a pre-existing osteopenia that predisposes to Charcot Osteoarthropathy or does osteopenia only develop after the onset? [abstract]. *Diabetes* **53** (Suppl2): A41.

Uccioli L, Faglia E, Monticone G et al (1995) Manufactured shoes in the prevention of diabetic foot ulcers. *Diabetes Care* **18**:1376–77

Vileikyte L, Rubin RR, Leventhal H et al (2001) Determinants of adherence to preventative foot care [abstract]. *Diabetes* **50** (Suppl 2): A17

Zimny S, Durmachkina E, Pfohl M. (2004) Frequency of the Factor XIII Val34Leu-polymorphism in patients with diabetic foot ulcers [abstract]. *Diabetes* **53** (Suppl 2): A264