COMMENTS AND RESPONSES

Comment on: Bernstein. Reducing Foot Wounds in Diabetes. *Diabetes Care* 2013;36:e48

We very much appreciate the article by Dr. Bernstein regarding callus debridement and, in fact, agree with him in that calluses and their treatment are not trivial affairs in the patient with diabetes, neuropathy, and peripheral artery disease (1). Just as we have all seen devastating consequences with coumadin therapy or colitis caused by antibiotic dosing, callus debridement in the wrong hands can lead to significant morbidity too. This is increasingly true as our formerly mainly neuropathic patients are now increasingly often neuroischemic (2). We would argue, though, that—just as with coumadin or colitis—the lack of treatment can lead to even more devastation. In terms of calluses, perhaps we should consider the problem, then look at numbers associated with the problem.

Calluses are indeed a normal response to pressure and shear stress incurred during walking. When they become abnormally thick, however, they can impart a massive amount of pressure on the plantar aspect of the foot. Often, this leads to subcallous bleeding and ulceration, which is only uncovered when it is debrided. In fact, leaving the callus over a wound in the absence of painful feedback (or even with it present) allows further damage and progression of ulcer depth to occur. Debriding reduces mean plantar pressure by some

30% (3). Shoes alone could not have this effect, as calluses would take months to disappear. In fact, an Achilles tendon lengthening, from previous work by the group of Armstrong et al. (4), leads to only 28% reduction in pressure.

Let us now have a look at the numbers in our study population: Among the 247 initial lesions that led to inclusion, 26% were footwear related, 13% associated to callosities or insufficient callus care, and another 12% caused by insufficient nail and foot care performed either by the patient, his family, or a professional. The proportions of callosities as the causative event for ulceration were strikingly different among patients with evidence of peripheral arterial disease (4%) and those without (24%). During follow-up, collectively 531 events of ulcer recurrence or new ulceration were observed, resulting in 1-, 3-, and 5-year cumulative incidences of at least one recurring ulcer episode among those under risk (i.e., alive and with at least one leg) of 35, 63, and 77%. These figures for patients receiving standard care are in accordance with those reported from Apelqvist et al. (5). Without provision of preventive measures the majority of patients with diabetes and a history of a foot lesion will exhibit at least one relapse every year. In fact, this kind of approach (seeing a podiatrist along with another member of the diabetes care team) has led to between a 19 and 64% reduction in 6-year amputation rate (6). In this case, it appears that the more you need a team approach, the better it works. We applaud Dr. Bernstein for his care and commitment. We would urge him—just as with any treatment—not to throw the therapeutic baby out with the hyperkeratotic bathwater.

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