REPLY: NONSEPTIC TARSAL DISINTEGRATION IN LEPROSY

Sir.

We have done a study on more than 50 cases of tarsal disintegration (TD). The research work comprised studying various biomechanical and other factors involved in the aetiopathology of this neuropathic entity.

To date we have a record of 28 cases having the nonseptic variety of TD. None of them had a history of ulcers. Out of 28 cases (2 bilateral) the majority were of the borderline–tuberculoid variety. All of them were under regular antileprosy treatment and had negative skin smears. Twenty-two patients had medial pillar involvement, 6 patients had posterior pillar involvement and in 2 patients all pillars were involved.

Among 28 there were 5 cases who underwent tibialis posterior transfer surgery for foot drop. These patients were treated with ordinary MCR footwear with arch support and follow-up showed stable lesion.

The rest of the patients were treated successfully by Fixed Ankle Brace (FAB) Walkers and a period of 2 years of follow-up showed stable results.

Superficial and deep sensory testing could be performed only in 8 patients. All of them had total loss of superficial sensation. There was a loss of joint position sense in all. All of them had higher vibratory threshold values when compared with the other leg.

Superficial sensory testing has direct correlation with the process of disintegration: however, loss of deep sensation does not seem to have predictive value as observed from our experience.

All the cases who had TD had significantly higher vibratory threshold values, but all cases having higher vibratory threshold need not have TD. This was observed in a separate study where in 40% of the patients who had no ulcers and no signs of TD had abnormal vibratory threshold values.

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V N KULKARNI

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Mr Kulkarni has thrown interesting light on this subject. It is particularly valuable that they have demonstrated a correct approach to early prevention of further damage, once the loss of proprioceptors is a fact. If, as the preliminary evidence indicates, early diagnosis of loss of joint position sense is of predictive value, then there really is hope that we may be able to prevent at least many of the late, unfortunate and disastrous results that too often leads to ablative surgery with all its unhappy effects.

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REPLY: NONSEPTIC TARSAL DISINTEGRATION IN LEPROSY

Sir,

Dr J G Andersen has written about 'Nonseptic tarsal bone disintegration in leprosy' and asks about other cases (*Lepr Rev*, 1988, **59:** 187). What he describes is not unusual in the Eastern half of Asia.

In the first patient that he speaks of he states '. . . some new bone formation of a hazy nature' was seen along with the lysis of other bones. This statement provides the clue to management.

A large proportion of tarsal bone disintegrations is nonseptic and the remnants of bone are still in the foot—though virtually invisible because of the degree of osteoporosis. If the foot is completely immobilized in a functional position in a contact walking plaster cast the bones will return. That is, recalcification to occupy the shape as moulded in the cast, will recur and eventually (9–12 months) the bones will be hard enough and sufficiently sclerosed for the patient to walk again without braces or moulded footwear. Healing should be carefully tested clinically, by a trial of walking—starting with a few minutes a couple of times daily and increasing the duration daily, provided there is no heat or swelling (hot spots). The occurrence of hot spots that persist or rapidly recur suggests that the contact cast needs to be replaced for a further 2–3 months.

These patients can be detected early by teaching the patient to look for 'hot spots'. Although there is usually no direct history of trauma, the initial lesion is usually a fracture, which may be very mild and go unnoticed because of no pain. Osteoporosis develops around any fractured bone and the weakened bone is then fragmented as the two edges rub together during use. Biopsy has shown that osteoclastosis and osteoblastosis are normal but continued movement prevents the formation of solid bone. So it is not unusual to find evidence of 'new bone' in radiographs.

Unfortunately, there is no satisfactory way of neurological testing that will prewarn of the possibility of a degeneration occurring. Any patient with a sensory neuropathy may be affected, but some patients have developed marked neuropathy in the presence of apparently normal motor and sensory function. Obviously, deep sensation can be affected without superficial neural deficit.

Prevention lies in teaching daily self-care, which these patients apparently did, together with checking for hot spots. The patient can be taught to check and institute therapy early, if needed, before marked deformity has occurred. He needs to know the significance of a hot spot.

The reconstructed bones will last many years (I have follow-ups of over 20 years now) provided the shape achieved in the plaster is compatible with use. Feet that are grossly deformed can be reshaped surgically and bone healing will occur. It is probably easier to wait 3–6 months in plaster before starting reconstructive surgery, so that the bones have somewhat reconstructed and one can see better what one has to work with. Sometimes the bones are soft enough to cut with a knife!

There is no place for surgical decompression in the swelling phase—the swelling will subside when movement is stopped.

Early diagnosis carries a good prognosis if adequately treated.

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Dr Warren rightly underlines that nonseptic tarsal disintegration is not uncommon. I am in full agreement with the outline of treatment and prevention that she employs. Certainly it is a major tragedy to come across patients with tarsal nonseptic disintegration that require major ablative surgery. This must at all costs remain the very last resort.

Unfortunately the correct treatment requires not only a good understanding and cooperation from the patient, but also social and economic conditions that well may prevent the patient from accepting the correct treatment. Less than perfect peripheral services may often mean that these patients simply come too late for correct treatment.

I am puzzled by her statement that there is no satisfactory way of neurological testing that will prewarn the possibility of a degeneration occurring.

My own experiences indicate that absence of joint and position sense, as distinct from sensory loss, is an eliciting factor, although not all people with loss of proprioceptor sensation do develop nonseptic tarsal disintegration. The remark that surgical decompression has no place in the management of this condition must be underlined. 'Where there is pus, evacuate. Where there is no pus, for goodness sake don't evacuate.'

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REPLY: NONSEPTIC TARSAL DISINTEGRATION IN LEPROSY

Sir,

I want to respond to the letter on this subject by Dr J G Andersen. In reading over Dr Andersen's letter he talks of doing major ablative surgery in patients who have had tarsal disintegration. I'm not clear as to what he means, I think the implication is amputation.

In my experience it is rarely necessary to amputate for tarsal disintegration, even when there is marked destruction of bone. It is usually possible to arthrodese what bone there remains by cutting flat opposing surfaces and bringing them together by the use of a Charnley clamp applied to Steinman pins for opposition and fixation.

In instances where there are inadequate bony surfaces to provide solid fixation with a Charnley clamp a 3 pin procedure can be used. Thus, when there are 3 bones that can be brought into apposition, the opposing surfaces are prepared and fixed with 3 pins. In order to hold the Steinman pins they are encircled at either end with long strips of rubber which are cut from an inner tube of a tyre. The duration of fixation in such cases is necessarily somewhat longer than for a normal extremity in which there is sensation. In nearly every instance a good functioning extremity will result. If protective footwear is supplied this can provide a practical and functional foot.

I recall one patient who had a completely flail ankle joint and approximately 4 inches of shortening of the distal end of the tibia. Even in this extreme situation it was possible to obtain a solid bony fixation and provide adequate function for ambulation after this type of surgery, plus footwear with ankle support.

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R E PFALTZGRAFF

Drs Pfaltzgraff and Warren have responded with an excellent description of the correct treatment of such conditions. With this I am in full agreement. Nor should ablative surgery be necessary. That it unfortunately may become so, is probably to some extent due to problems in the field management of the disease. I wanted to mention that I have consistently found absence of response to vibration test, indicating loss of proprioceptor sensation. In my present situation I am unable to follow up this observation. I therefore ask workers with the potential for such studies to do so. None of us has mentioned that the differential diagnosis of nonseptic tarsal disintegration against deep septic infection or oedema of other aetiology may be difficult. Neither antibiotics nor corticosteroids have any place in the management of this condition.

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J G ANDERSEN

REPLY: NONSPECIFIC TARSAL DISINTEGRATION IN LEPROSY

Sir,

In recent travels in Africa this year, I saw 2 cases, reminiscent of that described by Dr Andersen, hospitalized in 2 different countries. In neither case were the team aware of the need to detect and prevent this distressing complication of leprosy.

In one case, a heavy man who had been on bed rest after an amputation of one leg walked with crutches too much and too soon after immobilization, and in the absence of adequate pain protection pounded the osteoporotic talus of the 'good leg' to fragments. Radiologically the talus was fragmented and the tarsal architecture disintegrated.

The other case (clinically diagnosed as early disintegration) was a woman with a hot swollen ankle, walking too early and too much just after ulcer healing by immobilization, started her on the slippery path towards total disintegration. Hopefully if the precautions given were observed her foot will be saved.

There is no way a sprained ankle can stand uninhibited walking in the absence of pain protection without disintegrating, especially if the tarsal bones are already weakened either by disuse, osteoporosis or infection.

Although the degree of anaesthesia is scientifically interesting and may be predictive, all patients with anaesthetic feet should be aware of the dangers of diminished (if not absent) protection of their ankles by pain, and appropriate deformity prevention measures observed.

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Reference

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