LETTERS

The absence of reactive arthritis after *Shigella sonnei* infection

To the Editor:

In 1966, Noer (1) reported the incidence of Reiter's syndrome in 9 of 602 sailors who had shigellosis. The shigellosis had followed a stay in a port where dysentery was endemic. Five of these patients with Reiter's syndrome were restudied by Calin and Fries 10 years later, and an association with HLA-B27 antigen was documented (2). Their report made us aware that Reiter's syndrome does not always have a benign prognosis.

We recently had the opportunity to do a prospective evaluation of 250 Army reservists who had developed *Shigella sonnei* dysentery in September 1980 and to record how many had developed signs or symptoms that suggested Reiter's syndrome. The identification of the responsible organism, *S sonnei*, was done by the Arkansas State Health Department and our hospital laboratory. Both reported the same organism. All patients were treated supportively with antibiotics, and all recovered in 3–4 days.

An easily answered questionnaire was mailed to all of the patients who had documented dysentery. We tried to ascertain whether any joint, eye, back, urethral, or skin problems had subsequently developed. To those who gave a positive response a second questionnaire was sent, in which the responders were asked to give details, including specific complaints, duration, nature of the malady, and present health. We also ascertained whether any of these complaints had been present before the diarrheal episode.

One hundred twenty-seven people (approximately 50%) returned the initial questionnaire. Of these, 6 indicated that they had had some musculoskeletal complaints. Two had had peripheral joint complaints, 3 low back pain, and 1 both joint and back symptoms.

We mailed the second questionnaire to these 6 subjects. All replied that their complaints had cleared within a few days after the dysenteric episode and that they had suffered no recurrences. An exception was 1 person whose personal physician answered; he assured us that the patient was suggestible and that these symptoms had only developed after receiving the second inquiry.

Basing our predictions on Noer's experience, we had expected 3–4 persons to develop Reiter's syndrome, but none did. This was similar to the outcome reported by Good (3). Further review of the literature revealed 3 other pertinent reports in the past few years. Kaslow et al (4) studied 4,205 people exposed to *Shigella sonnei*; 1,907 had been infected, but no cases of Reiter's syndrome were found in this population. Simon et al (5) studied people with both *S sonnei* and *S flexneri* infections. They found no cases of Reiter's in 99 patients with *S sonnei*, but 6 of 410 patients with *S flexneri* had Reiter's syndrome. Morse et al (6), reporting on the frequency of HLA-B27 and Reiter's syndrome in a Navajo population, noted that diarrhea was present in 39% (7 of 18) of their patients. They also pointed out that *S flexneri* is endemic in the area in which their patients lived.

The failure to demonstrate an association between *Shigella sonnei* dysentery and the development of Reiter's syndrome could represent a sampling error. Our response rate of 50% is the same as that experienced by Calin (2), when an attempt was made to locate Noer's original patients. The Army Reserve would not give us the names or addresses of the persons involved in the epidemic, but mailed the questionnaires themselves. Unfortunately, we therefore had no way to followup on the nonresponders. Our questions, however, were slanted to suggest an association between the dysentery and subsequent problems; we feel that symptomatic persons would have been willing to answer the questionnaire in order to qualify for service connection and disability.

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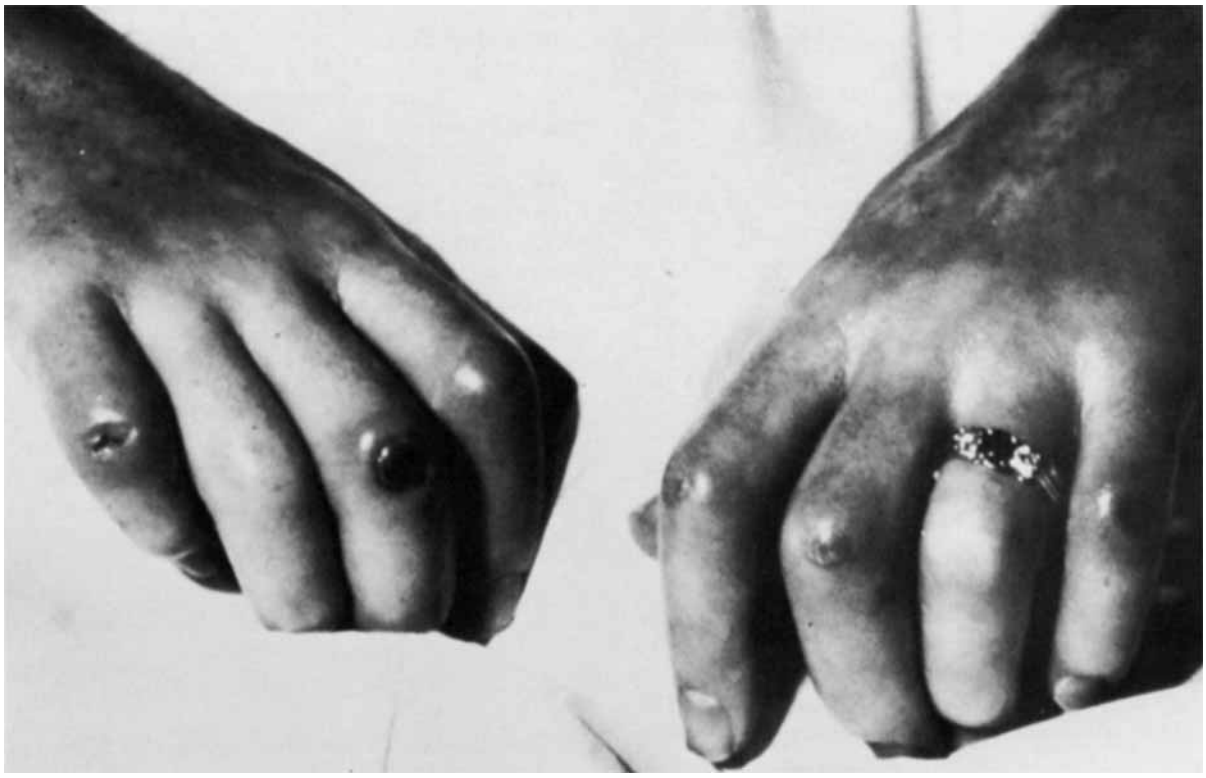
Nifedipine in digital ulceration in scleroderma

To the Editor:

There have been several encouraging reports of the usefulness of calcium channel blocking agents in the treatment of idiopathic Raynaud's disease, as well as Raynaud's phenomenon associated with progressive systemic sclerosis and systemic lupus erythematosus (1,2). I wish to report the apparent effectiveness of the calcium channel blocking agent nifedipine in the healing of refractory digital and elbow ulcerations in a patient with progressive systemic sclerosis and severe Raynaud's syndrome.

The patient is a 38-year-old woman who first developed Raynaud's phenomenon in 1979. This soon became associated with a brawny edema of the hands, which rapidly progressed to severe acrosclerosis and generalized sclero-



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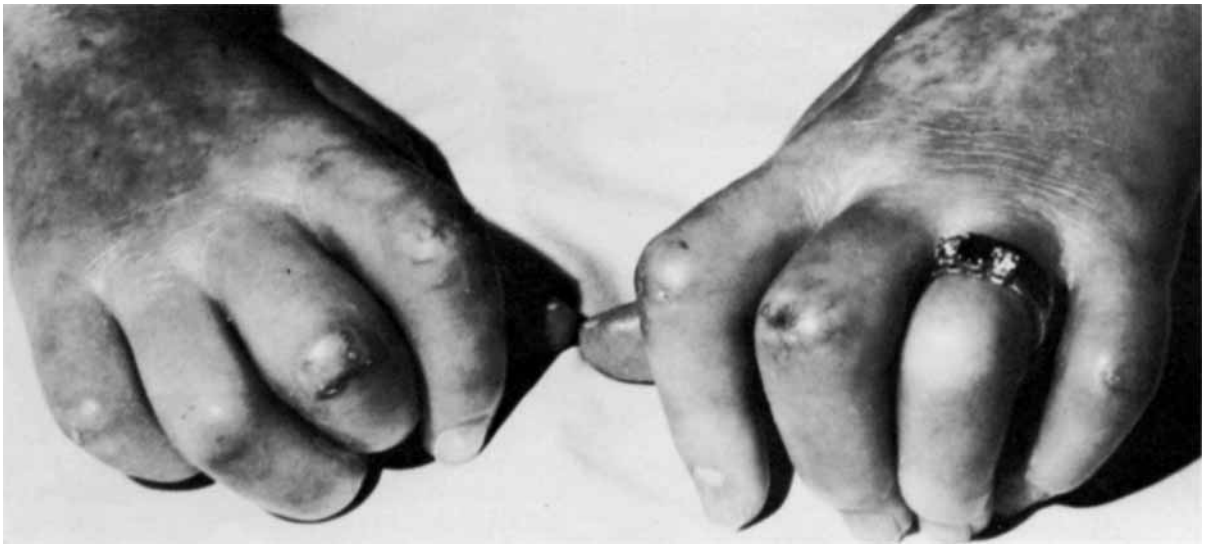

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Figure 1. Improvement in ischemic ulcers during nifedipine therapy. **A**, Active ulcers over the third and fifth proximal interphalangeal joints on the right hand and the fifth proximal interphalangeal joint on the left hand. **B**, Healing of these lesions 12 weeks after the start of nifedipine therapy. Note the revascularization over the first proximal interphalangeal joint on the right hand and third proximal interphalangeal joint on the left hand.