was highly significant ($P < 0.001$). Esophageal peristalsis remained unchanged after administration of each drug.

Diltiazem does not show a tendency to lower LES pressure, which is the major drawback with nifedipine. Therefore, diltiazem is to be preferred over nifedipine in the treatment of Raynaud's phenomenon in patients with PSS.

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Successful treatment of Raynaud's phenomenon with pentoxifylline

To the Editor:

Raynaud's phenomenon (RP) is a common feature of many rheumatic diseases. Although the pathophysiology of RP remains unclear, many theories have been proposed: local digital vascular abnormalities, an "overreactive" sympathetic nervous system, desensitization of alpha receptors, circulating vasoconstrictor substances, abnormal synthesis or release of prostacycline or prostaglandins, and abnormalities of blood viscosity or plasma proteins (1). RP may be primary or it may be secondary to other diseases, particularly rheumatic diseases.

Red blood cell rigidity may be important to blood viscosity (hence small vessel blood flow) and may amplify the so-called inversion phenomenon, a complex alteration of cellular elements of blood flow through the circulation, which effectively decreases viscosity with the reduction in vessel diameter (2). Increased blood cell rigidity has been found in renal failure, in hemolysis, and in some cases of Raynaud's phenomenon (3).

We have observed a systemic lupus erythematosus (SLE) patient with RP that was characterized by 12–15 attacks per day, digital ulceration, and accompanying dysesthesias. While this patient was inadequately responsive to conservative methods of controlling RP, she had a marked and sustained clinical response to pentoxifylline.

The patient, a 37-year-old woman, had SLE of 5 years duration, which was characterized by antinuclear antibody (ANA) positivity (titer 1:320, speckled pattern), arthralgias, arthritis, pleurisy, fever, and subacute cutaneous lesions, without evidence of renal involvement. She presented with a 1-year history of 12–15 episodes of RP per day, and frequent small, painful digital ulcerations. There were no detectable levels of anti–native DNA (by *Crithidia* assay) or cryoglobulin. Anti-Ro, anti-La, and circulating immune complexes were not sought. Drug therapy at the time of presentation included piroxicam and hydroxychloroquine. Despite avoidance of precipitating stimuli and trials of calcium channel blockers (nifedipine and verapamil), her daily painful attacks persisted. She was empirically begun on a regimen of oral pentoxifylline, 400 mg, 3 times a day. Within the first 2 weeks, the number of episodes of RP decreased to 2 per day. Within 1 month, episodes diminished to 1–2 per week, and her digital ulcerations healed. She experienced no adverse effects from the pentoxifylline and has continued treatment with it. She has experienced no further rash or digital ulcerations, and her disease has otherwise remained under adequate clinical control.

Treatment of RP is often unsuccessful. Avoidance of the cold, smoking, and emotional stress, coupled with biofeedback or hypnosis, may be of variable effectiveness. Plasmapheresis is of questionable value except in those patients who have clearly documented hyperviscosity. Digital sympathectomy is clinically effective, but requires operative intervention with attendant risks. Chemotherapeutic intervention with oral or intraarterial vasodilating agents has been variably helpful. Side effects such as hypotension, light-headedness, and/or diarrhea, which indicate intolerance to calcium channel blockers, are common.

There is a subset of patients with RP who have increased red cell rigidity and may respond to pentoxifylline, a newly approved medication for the treatment of nonoperative ischemic claudication. Pentoxifylline is a methylxanthine with vasoactive properties. It changes rheologic properties of blood secondary to an increase in the flexibility of red cells. In addition, pentoxifylline may induce vasodilation by inhibiting cyclic adenosine monophosphate phosphodiesterase and relaxing vascular smooth muscles (4). A decrease in platelet hyperactivity due to pentoxifylline has also been described (5). The therapeutic profile of pentoxifylline is remarkably free of adverse effects (6).

While this report describes only 1 patient, it presents an interesting approach to the treatment of RP. Pentoxifylline could possibly be used with or without calcium channel blockers in a larger clinical trial in the treatment of RP. We

have subsequently treated a 29-year-old woman with systemic sclerosis of 3 years duration, which was characterized by ANA (titer 1:320, discretely speckled pattern), telangiectasias, esophageal motility disorder, sclerodactyly, RP, and digital ulcerations. Her RP has been controlled and her ulcerations have healed with pentoxifylline treatment, despite the Wisconsin winter.

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BOOK REVIEW

Rheumatoid Arthritis. *Second edition. Duncan Gordon, editor. New York, Medical Examination Publishing/Elsevier Scientific Publishing, 1985. 257 pages. Illustrated. Indexed. $29.50.*

This concise, inexpensive, hard-cover book represents an up-to-date treatment of a much-written-about, highly prevalant, rheumatologic problem. Published almost simultaneously with Utsinger's comprehensive textbook, this volume offers a more compact, less expensive coverage of the same disease. As mentioned in Christian's review of the aforementioned text (1), several excellent books on rheumatoid arthritis had been published prior to 1974. Published in that year was the small volume on this subject, which was edited by Harris and utilized chapter editors associated with the Massachusetts General Hospital program (2). The present text uses chapter editors from the excellent faculty at the University of Toronto and thus achieves a coherence not usually found in multi-authored works. This work also updates the excellent management manual written by Hollingsworth (3).

As noted in the Foreword, this volume in Medical Examination Publishing Company's contemporary patient management series presents a state-of-the-art account of the management of rheumatoid arthritis as practiced in the Toronto teaching hospital system. Chapter 1 gives an excellent orientation to the disease, and chapter 12 summarizes the discussion of management, with the intervening chapters presenting practical and lucid discussions of the pertinent material. While one might quibble with individual opinions presented by the authors, such as their recommendation, in chapter 2, of enteric-coated aspirin as the first-choice short-acting drug, this is presented clearly as their own bias. They do mention in chapter 3 that others might choose newer nonsteroidal antiinflammatory drugs, and, by implication, that still others might choose plain aspirin. Differences in the US and Canadian practice of rheumatology are evidenced by the relatively detailed discussion of injectable radioisotopes in chapter 6 and the inclusion in Table 3.1, entitled "Currently Available Nonsteroidal Anti-Inflammatory Drugs," of 7 compounds not currently available for use in the US.

Good coverage of rehabilitation, psychological management, and surgical matters is provided in chapters 7, 8, and 9. With this group's extensive experience in the diagnosis and management of extraarticular rheumatoid arthritis, one might have wished for a more detailed discussion of this in chapter 10, "Complications of Rheumatoid Disease." "Complications of Therapy," the topic of chapter 11, might have been discussed in the chapters on the individual agents rather than in a separate chapter.

Overall, however, the book succeeds admirably in presenting an up-to-date coherent and balanced treatment of the management of rheumatoid arthritis. It would seem to be of more use to the general practitioner, who deals with rheumatoid arthritis patients on a day-to-day basis, than to the rheumatologist or subspecialty-oriented internist, who might desire a more comprehensive treatment of this problem.

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