Management of Raynaud’s Phenomenon  
Focus on Newer Treatments  

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Summary  
Current thinking on the general approaches to handling patients with Raynaud’s disease is briefly described, and the principles of management discussed. The various categories of drug treatment available – vasodilators, especially those active on the smallest blood vessels, drugs acting on endothelium and platelets and their products, rheologically active drugs and some whose action it is difficult to classify – are mentioned. By far the most widely tested drugs in this field are the dihydropyridine-like slow calcium channel antagonists, of which nifedipine is probably the best known. Side effects are common and the optimal dosage and drug formulation are yet to be achieved. Serotonin antagonists (naftidrofuryl, ketanserin) look promising, although ketanserin is not generally available yet. Drugs active in the sympathetic control of vascular tone may well be best reserved for the most severe forms of Raynaud’s, especially perhaps those associated with tissue loss in the secondary disease. Older vasodilators, such as glyceryl trinitrate (nitroglycerin) and some of the nicotinic acid derivatives, have not been studied of late but the transdermal applications of glyceryl trinitrate at least sound attractive. Drugs active in the cyclo-oxygenase systems, especially those with prostacyclin-like activity or thromboxane antagonists, are obviously promising; however, their unavailability in oral, sublingual or transdermal forms limits comment on them at present. Non-drug approaches such as biofeedback control of vascular responses may be interesting in a small number of patients, but the advice to ‘keep warm’ (and how to achieve this) is probably the most valuable suggestion that can be given to patients with Raynaud’s disease.
Raynaud's phenomenon, despite being described over a century ago (Raynaud 1888), is still not clearly defined or able to be categorised clinically in such a way as to make the organisation of treatment straightforward. Conventionally, however, a distinction is usually made between primary and secondary Raynaud's (Roath 1989). Most patients with primary Raynaud's exhibit the classical sequence of symptoms – blanching of the extremities on exposure to cold, followed by painful rewarming and recolorisation. There are probably subgroups of the primary form: for example, one variant may be the case where the initial blanching apparently does not occur but swelling and discolouration of digits is prominent. There is also a group of other cold-associated disorders which may be confused with Raynaud's, and a large group of important disorders whose resulting symptom complex may mimic some of its features. Figure 1 gives an approximate breakdown of distribution of various disorders which may be associated with the phenomenon, and table I lists some of the more important causes of secondary Raynaud's. Table II lists some other cold-associated phenomena which should be distinguished from Raynaud's.

By far the largest group of patients to present are likely to be those with primary Raynaud's disease, although if followed for long enough about half of them may develop some underlying disorder of which the Raynaud's is a symptom. Frequently, the presentation is in young women, although new patients with the primary disorder do present at all ages. There appears to be no known racial predisposition, although symptoms obviously tend to be masked when the skin is pigmented. Occasionally, what appears to be primary Raynaud's may be seen in infants and children. The pathophysiology of the primary disease is still poorly understood although, in general terms, it appears to be a quantitatively abnormal response.