perforation. Any ulcers visible from the serosal surface but unperforated should be oversewn.

Gastroenteritis resulting from the various serotypes of S enteritidis is becoming increasingly common and may be extremely severe. The presentation varies but usually includes abdominal cramps, pain, and diarrhoea that may be bloody. This picture may be confused with that of ulcerative colitis. Diagnosis may sometimes be difficult: salmonella enteritis and ulcerative colitis are both common and their sigmoidoscopic appearances are similar. If the colitis is treated with corticosteroids then simultaneous systemic antibiotic cover is essential.

The micro-organisms invade focal areas of the small and large bowel, and occasionally the colitis progresses to perforation. This complication has been reported after infection with many different species, including S. typhimurium,10 and S. newport.3 We recently encountered perforation and faecal peritonitis in a patient infected in the recent outbreak of S. kegougou. A high index of suspicion is necessary during an outbreak of salmonella enteritis; if perforation of the colon does occur prompt colectomy is essential for a favourable outcome.

Extraintestinal focal lesions with salmonella are often associated with other chronic disease. The focal lesions may manifest themselves long after an episode of enteritis or the original bowel infection may have been silent. S. enteritidis has a predilection for blood vessels. Possibly, intravascular salmonellas localise at sites of atheromatous ulceration and may cause thrombosis of major vessels or may colonise vascular grafts.10 In addition they may damage the venous system, causing septic thrombophlebitis, iliac vein thrombosis, or pulmonary embolism. These focal lesions have a tendency to chronicity and may mimic tuberculosis, particularly in osteomyelitis of a vertebral or paravertebral abscess. Circulating S. typhi is trapped by the liver and excreted in the bile. Acute cholecystitis as a complication of typhoid has become rare since the advent of antibiotics, but it may develop in a normal gall bladder and may proceed to perforation and biliary peritonitis.

Finally, the general surgeon may be asking for help in the management of patients who are chronic typhoid carriers. The typhoid carrier passes organisms in the faeces, presumably derived from a focus of infection in the gall bladder or biliary tract. Chronic typhoid cholecystitis is symptomless. The most notorious carrier was Mary Mallon, who was the source of an outbreak affecting 1300 people in New York in 1903. Although prolonged administration of ampicillin in high dosage has been successful in eliminating carriage of S. typhi even in patients with gall stones and non-functioning gall bladders, cholecystectomy is regarded as the most effective way of permanently curing the carriers—provided there is no associated infection in the biliary or urinary tract.

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Raynaud’s syndrome
Thyroxamine, iloprost, and ACE inhibitors are among the effective treatments now available

Raynaud’s phenomenon was described over 100 years ago, but we still do not understand the pathogenesis of the normally prolonged (sometimes painful) episodic peripheral vasospasm in response to cold or emotional stimuli. The controversy has yet to be settled between the hyperactivity of the sympathetic nervous system described by Raynaud and Lewis’s local fault.

The clinical classification into primary and secondary Raynaud’s syndrome and phenomenon is also unsatisfactory. Patients who have Raynaud’s phenomenon but are otherwise fit may commonly be shown to have one or more autoantibodies present in low titre. Some of these patients will later develop one of the connective tissue diseases.14

So what do we know? Patients with Raynaud’s syndrome may be shown to have increased sensitivity or density of the peripheral α adrenoceptors, or both, in both the vessel walls and the platelet membranes.15 Other factors implicated in the disease are abnormal platelet adhesiveness,1 increased whole blood and plasma viscosity,16 reduced red cell deformability,17 reduced activity of the fibrinolytic system,18 an imbalance in the cyclo-oxygenase products of arachidonic acid—thromboxane (TXA2) and prostacyclin (PG1)—and hypersensitivity of serotonin (5-HT2) receptors.14 Whether any or all of these are crucial in the pathogenesis or are simply epiphenomena remains to be determined. Attempts to correct these abnormalities have been reflected in numerous therapeutic approaches to the disease. Despite the high proportion of women affected the contribution of oestrogen and other female sex hormones remains vague and poorly defined.19 A possible exception may be those patients whose symptoms begin at or near the menopause, in whom hormone replacement treatment may be beneficial.19

In the clinical setting primary Raynaud’s syndrome is common.19 The average general practitioner may expect to see one or two new cases a year. Fortunately it is also usually benign and compatible with an entirely normal lifespan (unpublished data). The prognosis of secondary Raynaud’s syndrome is that of the underlying disease and depends on the severity with which target organs are affected.19

As the common stimulus is cold, either local or general, advice not to smoke and to keep warm remains the cornerstone of management. Smoking a cigarette may produce a fall in temperature of 2°C or 3°C in the fingertips of normal people in a comfortable ambient temperature. Patients may be helped to keep warm with appliances ranging from chemically activated handwarmers to electrically heated gloves, foot warmers, and hand and ear muffs. Further information concerning these may be obtained from the Raynaud’s Association (112 Crewe Road, Alsagar, Cheshire ST7 2JA; tel 0270 872 776).

About one third of patients also have attacks in response to

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emotional stress, though the pattern of response in primary and secondary disease may be different. Advice concerning avoidance of such stress may also be given. Patients in this category may respond to behavioural therapy or may benefit from conditioning (placing the affected hands and feet for a time in warm surroundings during general body cooling)—treatment requiring considerable time and motivation. More simply, placing the hands in warm water for five minutes twice daily is said to increase resting peripheral perfusion and reduce the reactivity to cold.

The ineffectiveness of cervical sympathectomy and plasmapheresis has condemned these measures to interesting historical footnotes. The attacks may be so frequent, or so painful, or last so long that drug treatment may be offered in an attempt to enhance peripheral perfusion and reduce the response to cold, especially during the winter. Despite the several drugs said to antagonise or correct one or more of the pathogenic factors outlined above few have been assessed in well controlled trials over many weeks rather than a few days.

Studies of this kind have been reported with some calcium channel antagonists, mainly nifedipine22-24; the a adrenoceptor antagonist, thymoxamine25; the fibrinolytic activity enhancing agent, stanazolol—‘which should be used only in advanced cases when other methods have failed’26; and for prostaglandin E127-28 and prostacyclin (prostaglandin I2)29 and its stable carbacyclin analogue, iloprost.30,31,32

The prostanoids are available only for intravenous infusion, limiting their use to hospitals, though inpatient management is not usually necessary.

How do these drugs compare? Nifedipine reduces the severity and frequency of attacks in most patients and often produces obvious peripheral skin vasodilatation in healthy and hypertensive people, but in our experience it does not enhance peripheral perfusion in the affected hands and feet of patients with secondary Raynaud’s syndrome.33,34 Clearly its benefits must be due to other mechanisms that enhance tissue oxidation.35 Side effects at conventional doses occur in about one third of patients: they are those side effects expected with generalised peripheral vasodilatation—headache, flushing, dizziness, and, more rarely, peripheral oedema. Potentially more serious side effects such as hepatitis or the nephrotic syndrome are rare.36 Pain in the eyes and blurred vision may be due to increased ocular blood flow.37 The incidence of side effects may be reduced by starting with a small dose—say, 5 mg twice or three times daily—and increasing incrementally until an effective dose is achieved that is free of side effects.

Thymoxamine acts by competitive antagonism at a adrenoceptors and is known to block vasoconstriction in some vascular beds—for example, the skin—while maintaining overall vascular resistance.38,39 The explanation for this selective effect may be the unique 10:1 ratio of α1 to α2 adrenoceptor activity.39 Three randomised control studies have shown that in patients with Raynaud’s syndrome (mostly primary) thymoxamine not only gives subjective relief of symptoms but also abolishes the prolonged vasoconstriction after a cold stimulus as shown by measurement of blood velocity and skin temperatures by Doppler ultrasonography (P M Dewland et al, first international congress of clinical pharmacology and therapeutics, London 1980).40,41 At doses of 40 and 80 mg four times a day thymoxamine had no effect on blood pressure and side effects were rare. In view of the low incidence of side effects thymoxamine is at present the drug of choice in patients with primary Raynaud’s syndrome who continue to get attacks of Raynaud’s phenomenon despite giving up smoking and protecting themselves from cold.

The prostanoids and their more stable analogues have two well documented activities: they reduce platelet adhesiveness and cause generalised vasodilatation. These are acute effects and stop shortly after treatment is stopped. As circulatory (headache and dizziness) and gut (vomiting and diarrhoea) side effects are frequent and may be severe their use has been confined to patients with severe secondary Raynaud’s syndrome with much reduced resting perfusion complicated by digital ulceration and to patients with frank gangrene who are undergoing digital surgery in the hope of enhancing postoperative wound healing. In patients with severe secondary syndrome with reduced perfusion and digital ulceration, regimens have varied with the prostanoid used. The stable carbacyclin analogue iloprost has been given for six to eight hours on three consecutive days at a rate starting with 0.5 ng/kg/min and increasing incrementally at 15 minute intervals by 0.5 ng to a maximum of 2 ng/kg/min. Side effects may preclude this high dosage, but it has been shown to increase digital blood flow above the baseline for many weeks. Digital lesions have healed quickly and there has been a corresponding maintained clinical improvement.34,35,36,37 No explanation for this prolonged beneficial effect has emerged—for it cannot be attributed to the acute effects, reduced platelet stickiness and vasodilatation, which disappear almost as soon as the infusion is stopped.

Though the optimal dosage of prostanoids has not been determined, patients with severe symptoms who are refractory to oral treatment may be treated with repeated infusions. Prostanoids have been available for 10 years but only one transdermal preparation has become available. This is effective in both adults41,42 and children,43 but unaccountably it was withdrawn by the manufacturers. Other transdermal and oral preparations are awaited with interest.

In primary and less severe secondary Raynaud’s syndrome other therapeutic approaches may be possible. Clearly the effect of inhibition of the angiotensin converting enzyme requires investigation in patients without renal impairment. Several new angiotensin converting enzyme inhibitors have become available and others are being developed. Already, however, captopril has been shown to be beneficial in patients with primary Raynaud’s syndrome at a dosage that did not lower blood pressure. Nor at this dose were there any other undesirable side effects—such as neutropenia or a reduction in renal function—as had been observed previously in some patients when high doses were used.44 Another drug said to be effective but requiring further investigation is the serotonin (5-HT2) receptor antagonist, ketanserin (which is not yet available for general use in Britain),45 with which improvement in digital perfusion may be modest.46 The beneficial effect of transdermal glyceryl trinitrate may be difficult to determine because of the large placebo effect associated with substances applied to the affected part by the patient.47,48

Fish oil dietary supplements—rich in the long chain ω-3 polyunsaturated fatty acids, eicosapentaenoic acid and docosahexaenoic acid, have several actions that might benefit patients with vascular disease.49 Not the least of these may be the redirection of prostanoid formation to prostaglandin I2, which retains its vasodilatory and platelet anti-aggregatory effects, and to thromboxane A2, which is biologically inert.50 Fish oil supplements have recently been claimed to improve cold tolerance and delay the onset of vasospasm in patients with primary but not secondary Raynaud’s syndrome, a finding that requires confirmation.51 Evening primrose oil, available over the counter, also contains eicosapentaenoic acid.

The recent advances made in our knowledge of general and local52 control mechanisms in the peripheral vasculature have raised hopes for increased understanding of episodic peripheral vasospasm, in particular the response to cold.
stimuli in health and disease. Meanwhile, we should aim at providing patients with as clear an understanding of their disease as possible and using the drugs available with circumspection, remembering that treatment is likely to be lifelong. Decisions should be made with, rather than for, the patient, making it possible to share disappointment at failure and pleasure when treatment is effective.

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Care for the infirm elderly

A widening gap between the poor and the better off

The 1990s seem likely to be a worrying decade for the elderly in Britain—all of whom are uneasily aware that at some time they may need residential care. The publication of the white paper Caring for People on 14 November 1989 put an end to the speculation about whether and how the government would respond to the Griffiths report on community care. It also completed another part of the jigsaw of radical reorganisation of health and social services provision that began with Promoting Better Health and Working for Patients. The latest white paper has provoked less controversy than its predecessors, but it falls short of the Griffiths report’s recommendations, and it spells out new arrangements for financing and providing services for elderly people who need close care analysis.

The white paper cites the 70-fold increase in public subsidy of residential and nursing home care over the past 10 years as evidence of the government’s investment in community care. But it also acknowledges the criticism by the Audit Commission and Griffiths that existing social security policies have created “perverse incentives” towards institutional care. In the new proposals a unified budget covering the costs of “social care” will be introduced and managed by local authorities. It will be available for either domiciliary or residential care according to individual need. People in residential care will be entitled to claim income support or housing benefit on a means tested basis, as they would in their own homes, but they will be required to pay most of that income to the local authority that is funding their care. Social services departments will be given the responsibility for assessing the care needs of dependent people and for planning and purchasing packages of care. They will be expected to collaborate closely with health and other agencies, to set up a system of quality control, and to reduce the part they play as direct providers. The government expects local authorities to make “maximum use” of independent sector