my case in a general rather than specialist medical journal as I feel that this unpleasant disease should be regularly drawn to the attention of a wide medical audience.

I did not intend to suggest that the elevated ESR should be the basis of diagnosis. One hundred and seventy-nine arthropathies have been formally identified.³ In 54 the ESR is usually raised, and in 24 it is usually normal.⁴ The ESR clearly cannot discriminate between arthropathies. However, gonococcal arthritis is a disease for which laboratory confirmation can be unhelpful. In Barlow's series the gonorrhoea complement fixation test was positive in 5 out of 16 cases, skin culture in 4 out of 7, joint culture in 3 out of 16, blood culture in 2 out of 9, and anogenital bacteriology in 21 out of 24. Clearly, the clinician needs all the help he can obtain; the ESR may be a useful guide to the nature of obscure arthritis.

I believe Barlow to be the first author to have stated that the ESR is usually raised. I wished to determine whether this parameter could be used to determine whether systemic dissemination of the gonococcus from its site of inoculation had been controlled by antibiotics. One case is clearly not enough to establish a point with important therapeutic implications but the thesis is at least tenable.

Dr Seifert is, of course, correct to emphasise the importance of the rectal sample. It was not relevant in a male heterosexual case and short reports are limited to 600 words. However, in a condition in which about 80% of patients are female rectal specimens are essential. Bhattacharya and Jephcott⁵ showed that 45% of women with gonorrhoea had anorectal involvement, and in about 6% of their patients with gonorrhoea the diagnosis would not have been made if anorectal material had not been examined.

Oropharyngeal gonorrhoea is increasing in incidence and it is no longer sufficient to rely on excluding gonorrhoea by anogenital investigation alone.⁷ Apart from a case of stomatitis described in 1931 all proved cases of oropharyngeal gonorrhoea have been reported in the last decade. Metzger⁸ has described a case of gonococcal arthritis complicating gonococcal pharyngitis. One sampling of the pharynx is not enough. Bro-Jorgensen and Jensen⁹ found the sensitivity of a single pharyngeal culture on selective medium, compared to two or more sequential cultures at varied intervals, to be 78% (only 86 of 110 cases were identified on the first culture).

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¹ Barlow, D, in *Advanced Medicine* 13. Tunbridge Wells, Pitman Medical, 1977.

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⁷ *British Medical Journal*, 1974, **2**, 239.

⁸ Metzger, A, *Annals of Internal Medicine*, 1970, **73**, 267.

⁹ Bro-Jorgensen, A, and Jensen, T, *British Journal of Venereal Diseases*, 1973, **49**, 491.

"Trench foot" caused by the cold

SIR,—The letter from Drs I C Fraser and Judith A Loftus (10 February, p 414) seems to describe not a case of "trench" or "immersion" foot but of frostbite. It is of interest because it emphasises the fact that frostbite of the periphery can occur in conditions of moderate

cold while fairly strenuous exercise is being undertaken if the skin's circulation is interrupted by tight clothing or footwear such as "stretch nylon socks and tightly laced training shoes." Of course, the flow of air over the feet, which would occur during cycling, makes the development of cold injury more likely at a given air temperature, because of the "wind chill" effect.

The somewhat less common condition of "immersion foot" differs from frostbite in a number of ways. Ungley *et al*¹ provide a good description and suggest the name "peripheral vasoneuropathy after chilling" for this form of cold injury. This has the virtue of emphasising that it is exposure to moderate cold for many hours or days which is necessary for the condition to develop and that damage may occur with no more moisture than condensed sweat inside rubber boots. When first examined, the typical case has cold, swollen, and blanched feet which feel heavy and numb. There is a sensation of "walking on cotton wool." This stage is rapidly succeeded by one of hyperaemia, in which the feet are hot and red. This may persist for days or weeks. Swelling and pain may be severe, and blisters may form, leading even to gangrene. This feature makes differentiation from frostbite difficult. In "immersion foot," however, apart from gangrene, the main changes are in the nerves and muscles. The vasomotor paralysis, anhydrosis, analgesia, and paraesthesia may last for long periods or may be permanent. Extensive muscle atrophy may occur, and this appears to be the result of cold ischaemia and not of nerve injury.

A 36-year-old man whom I saw recently had developed typical "immersion foot" after being exposed for 26 hours to peri-zero air temperatures, strong wind, and intermittent light precipitation while taking part in "adventure training." He lay, for the whole of this time, in a shallow depression, devoid of cover except for some bracken. He wore light boots, one pair of socks, a sweater, a hat, and a heavy cotton jacket and trousers. He had no gloves. He drank nothing, and ate only one packet of sandwiches. He soon became extremely cold and shivered violently. After three hours, his feet became numb from the ankles down. When he finally got up to walk a mile or so to shelter, he said, "It felt as though I had frogman's flippers on my feet." About an hour after leaving the depression he took off his boots in a warm room. The skin of his feet looked "very pink." Sensation was lost entirely for the next three days and returned slowly and partially over the next five. For the next three months there were paraesthesiae and inappropriate thermal sensations, but the loss of sensation gradually contracted to a small area of "stocking distribution" around the toes and heads of the metatarsals. The condition was then static for 10 weeks. On examination at this time there was impairment of touch and pain sensation in the area described. Joint sense was also impaired in several of the toes. Heat and cold were sensed correctly but more intensely than normal. The motor power of the feet and hands was normal, except that the lateral three toes of the left foot could not be moved. The left ankle joint was less brisk than the right; the plantar reflexes were flexor. The dorsalis pedis and posterior tibial arteries were strongly pulsatile—perhaps more so than normal. The blanching time was normal, as were the reactions to dependency and elevation. The toe skin temperatures, in a warm room, were normal at between 24.2 and 25.9 C. The vasomotor reaction of the feet to heat was normal, but while ice cold swabs caused rather intense and prolonged blanching on the right side, on the left there was only slight blanching accompanied by a further decrement in touch sensation.

Except for the absence of pain during the hyperaemic stage, the above history was

typical. The signs were also entirely consistent with the diagnosis, and their severity indicated a grade B to C case.² Thus a gradual return of full sensation, as nerve regrowth occurred, was to be expected, although the process might well take many months.

Lastly, the interesting rash described in the patient of Fraser and Loftus may well be a "cold allergic response."³

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¹ Ungley, C C, et al, *British Journal of Surgery*, 1946, **33**, 17.

² Webster, D R, et al, *Journal of Bone and Joint Surgery*, 1942, **24**, 785.

³ Horton, B T, and Brown, G E, *American Journal of Medical Science*, 1929, **178**, 191.

Who heads x-ray departments?

SIR,—Now that the correspondence about my suggestion that the head of an x-ray department need not be a consultant radiologist (27 January, p 271) has finished may I be permitted to make a few final remarks?

The abusive tone was again noteworthy and suggests to me some insecurity on the part of the correspondents. I did not suggest in my letter of (2 September, p 706) that clinicians should run x-ray departments but merely that due to shortage of consultant radiologists this might be necessary in some instances. It is also not possible to draw conclusions as to the acceptance of my views merely on the replies that get into the *BMJ*. My private correspondence suggests that quite a lot of radiologists and clinicians agree with me.

It is not a stupid suggestion that someone other than a radiologist (perhaps not even a radiographer) might run an x-ray department. We all accept lay administrators and I quite agree that clinical decisions should be taken only by those clinically trained. If radiologists were able to give more time to managing patients and less time to administration we should certainly cut down our waiting lists considerably.

The determination of your correspondents to maintain the status quo is particularly unfortunate at the present time, when it is clear to all sensible doctors that constructive dialogue with other health service workers is badly needed, and a satisfactory career structure for non-medical personnel is one thing we should certainly be discussing.

The intemperate tone of the replies to my letter was noted with interest by union members here and I have no doubt that this was the case elsewhere.

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* * * This correspondence is now closed.—ED, *BMJ*.

Drug compliance and the elderly patient

SIR,—Dr G H Curtis Jenkins (13 January, p 124) raises important points regarding the difficulty many elderly patients have in taking their medicines. Pharmacists have been aware of this problem for some time; however, recognising problems is often easier than finding solutions.

It is known that child-proof containers may be difficult for adults to open, and pharmacists have discretion not to use these containers when dispensing prescriptions if they are aware of

the problems. This discretion should not be exercised unnecessarily, as elderly patients often have visiting grandchildren who could be at risk. A demonstration to patients may be sufficient.

The amount of information required on a label means that unless the container is to be unacceptably large the type size must be small. The use of special label typewriters is preferable to poor handwriting, but is not the complete answer. Supplementary labelling or separate instruction cards may be used, but are not without hazard. Here again the pharmacist needs to be made aware of the problem when a prescription is presented to arrive at the best compromise for each patient. Streamlining a patient's drug regimen and hence reducing the number of containers or doses, or both, may help, and pharmacists can often advise on this too. Many elderly patients do not collect their own prescriptions, so the opportunity for the pharmacist to demonstrate the use of child-proof containers and reinforce directions unfortunately does not arise.

These problems can best be tackled by individual doctors, pharmacists, and community nurses working together on behalf of individual patients. With knowledge of the patient's difficulties and home circumstances, some solution, albeit perhaps a compromise, can often be found. Developments such as the establishment of general practice pharmacies in health centres and perhaps in the future registration of patients with pharmacists could encourage this co-operation, but are by no means essential. With good will from everyone involved, local initiative can, and does, produce considerable improvement.

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Stopping lithium prophylaxis

SIR,—Lithium therapy, like many other forms of chemotherapy, is associated with side and toxic effects and before commencing treatment the possible advantages will need to be weighed against these and the discussion leading to the treatment decision shared with the patient. The timing of the likely cessation of the treatment should be formulated during the process of the decision to institute lithium therapy.

Once an equilibrium has been achieved between lithium and other electrolytes, cellular networks and transmitter units will have been pervaded by lithium. It would not be surprising for the sudden withdrawal of lithium to be followed by an acute confusional state if the cessation of treatment occurred in a setting of electrolyte equilibrium. This could be the explanation of the events described by Dr D G Wilkinson (27 January, p 235).

In general terms, once the recurrent affective illness is held by lithium therapy an attempt should be made to withdraw the medication over a period of six months in those patients who have been on prophylactic therapy for more than a year. In coming to the decision to terminate lithium therapy the following factors can be relevant:

(a) The underlying neurophysiological stratum and an assessment of how near is the cessation of its instability (adolescence, the menopause, and the involution). (b) The length of the periodic cycle of the recurrent affective illness, withdrawal being attempted

when the symptoms have been in remission for twice the duration of the cycle when this is over twelve months or four times the length of the cycle where the latter is less than twelve months. (c) The degree of resolution of any personal problems or conflict—if these persist the withdrawal of therapy is more likely to be unsuccessful. (d) If electrolyte levels fluctuate more than plus or minus 0.2 mmol/l electrolyte equilibrium does not exist, and if there is evidence to suggest that the patient is not complying with the prescription this may be a clear indication to challenge the patient and stop medication if the response is not satisfactory. On the other hand, if steady levels of lithium have been achieved over a period of two to three months then a stage of equilibrium has been reached and it is my experience that a more favourable therapeutic response is likely.

On withdrawal the risks of relapse appear to be higher than the appearance of the withdrawal syndrome described by Dr Wilkinson.

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Fats and atheroma

SIR,—Sir John McMichael argues that dietary intervention is a futile and even hazardous approach to the prevention of atherosclerotic heart disease (AHD). His report (20 January, p 173) is a catalogue of the opinions of the uninformed supported by anecdotal data from inadequately studied populations.

The relationship between the concentrations and component ratios of plasma lipoproteins and the incidence of AHD is well documented.^{1,2} At a cellular level there is evidence that arterial uptake of low-density lipoprotein (LDL) cholesterol is proportional to its plasma concentration³ and that high-density lipoprotein (HDL) reduces binding of LDL to both high- and low-affinity cellular receptors.⁴ Similarly, clinical studies of individuals with precocious manifestation of AHD⁵ as well as individuals with extended protection from AHD⁶ support the conclusion that decreased risk is conferred by both high levels of HDL and decreased concentrations of LDL.

The effect of dietary fatty acids on the plasma lipoprotein spectrum has been the subject of extensive and systematic investigation. That cis-polyunsaturated fatty acids (PUFA) reduce concentrations of plasma total and LDL cholesterol is undisputed. Although an extreme increase in the ratio of dietary PUFA to saturated fatty acid (SFA) may have less desirable effects on the HDL component,⁷ preliminary results suggest that more moderate fatty acid modification (PUFA:SFA, 1:0) may be associated with absolute increases in plasma HDL⁸ and certainly with an increase in the HDL:LDL ratio.⁹

The idiopathic effect of certain vegetable oils on myocardial metabolism, as mentioned by Sir John, is hardly central to the argument. No one is recommending the ingestion of high erucic acid—rapeseed, arachis, or hydrogenated oils—for preventing AHD. Plasma cholesterol-lowering agents including PUFA have been associated with an increased incidence of cholelithiasis.¹⁰ An increase in biliary cholesterol may or may not be an inevitable concomitant of centripetal cholesterol flux. In any case, the morbid consequences of cholesterol gall-stone disease cannot be equated with AHD. Of perhaps greater concern is the possible effect on the risk of colonic carcinoma of any dietary regimen that increases faecal bile acid concentration.¹¹ If the latter is indeed a potentiating factor in lower intestinal carcinogenesis a concomitant increase in dietary fibre might be an appropriate recommendation.¹²

Unequivocal proof that the soundly based and widely accepted recommendations for dietary change will in fact reduce the overall incidence of AHD and its mortality is perhaps an impossible goal. The nature of the disease process is such that the inconsistent results of early trials of secondary prevention can hardly be unexpected.^{13,14} The three major primary prevention trials have, however, all shown a reduction in the signs of AHD not attributable to imperfections in design.^{15–17} A greater effect might be expected with a more rigorous dietary protocol. Sir John's reference to the inadequate outcome of the WHO clofibrate trial¹⁸ is hardly relevant since this drug has a weak therapeutic effect in simple hypercholesterolemia.

Two important "natural" experiments lend support to the dietary fat-AHD hypothesis. These include the reduction in the incidence of AHD that bears a definite temporal relation to the dietary changes and associated decrease in mean serum cholesterol concentration in the United States.¹⁹ Similarly, Joossens *et al*²⁰ have documented a gradual increase in the dietary PUFA:SFA ratio in the north of Belgium. This has been accompanied by a decreased serum cholesterol concentration and reduced mortality from AHD not observed in the south of the country, where similar dietary change has not occurred.

Preventing AHD is a major medical and economic concern. A multiple approach is undoubtedly indicated. Although refinements in our understanding of the relationship of diet to lipoprotein metabolism and atherogenesis can be expected, present information provides sufficient basis for immediate and positive dietary advice.

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¹ Miller, N E, *et al*, *Lancet*, 1977, **1**, 965.

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⁵ Lewis, B, *Journal of the Royal Society of Medicine*, 1978, **71**, 809.

⁶ Glueck, C K J, *et al*, *Atherosclerosis*, 1977, **27**, 387.

⁷ Shepherd, J, *et al*, *Journal of Clinical Investigation*, 1978, **61**, 1582.

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¹¹ Hill, M U, *et al*, *Lancet*, 1975, **1**, 535.

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¹⁸ Committee of Principal Investigators, *British Heart Journal*, 1978, **40**, 1069.

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Leucopenia in association with mianserin treatment

SIR,—We wish to report a case of leucopenia following treatment with mianserin hydrochloride. This has not been previously reported in the literature. It was noted in a 73-year-old woman, a chronic depressive, who had been treated with amitriptyline for two and a half years with no good response. She was previously physically healthy.

The patient was started on mianserin hydrochloride 30 mg daily, increasing to 60 mg daily after 10 days. Over the next two days she developed infected, ulcerative lesions on the left side of her face and neck. All drugs were stopped. Blood samples at that time showed a white blood count