CORRESPONDENCE

Correspondents are urged to write briefly so that readers may be offered as wide a selection of letters as possible. So many are being received that the omission of some is inevitable. Letters should be signed personally by all their authors.

Myocardial infarction: home and hospital treatment

SIR,—Dr H G Mather and his colleagues (17 April, p 925) are warmly to be congratulated on a number of points. They have attempted a randomised trial in so difficult a field as the care of acute myocardial ischaemia; they have persisted with it in spite of criticism; their methods have broken new ground in the use of experimental epidemiology; and their results have already had an international impact on attitudes to treatment.

Their total population of 1895 cases is, in fact, divided into four chief groups: 457 (24%) for whom hospital treatment was considered mandatory; 837 (44%) who were considered suitable for randomisation but elected hospital treatment; 151 (8%) who elected home treatment; and the remaining 450 (24%) who were randomised to home and hospital treatment. Their previous report[1] included case fatality rates among the "elective home" and "elective hospital" groups and demonstrated that the immediate outcome of a heart attack among these groups was very similar, regardless of the presence of hypotension, to those for patients allocated randomly to home or hospital treatment. It would be most helpful in the evaluation of the whole experiment if the investigators, through your columns, now published 300-day survival rates for the "elective home," "elective hospital," and "mandatory hospital" groups.

One of the implications of the authors' work is that some patients are best treated in a coronary care unit (CCU) while others will be better off at home. The question that still faces the general practitioner, who is the central figure in all of this, is what to do about any one particular patient. In their present report Dr Mather and his colleagues suggest that "on average, older patients and those without initial hypotension fared rather better under home care." Another factor may be distance from home or from place of attack to hospital. We found,[2] in a study of men admitted to the CCU at Oxford, that the "case incidence" of ventricular fibrillation and case fatality rates were higher for patients with homes in the surrounding towns and countryside than for those living in the city itself. Our results are tantalising for two reasons. Firstly, the numbers involved were rather small, so that, although the differences in rates were large, they could have arisen by chance. Secondly, we could not tell whether patients from outside the city, who had a lower rate of admissions per age-standardised population than the rest, were particularly severe cases to start with or whether the long ambulance journey had done them harm. So it would be helpful, too, if Dr Mather and his group could determine whether their data relating to their randomised and other groups support the idea that there is a relationship between length of journey to hospital and case fatality. They might also be able to provide information about whether there is a relationship between distance from hospital and the allocation of cases to each of the four major groups.

ROY M ACHESON
COLIN SANDERSON

London School of Hygiene and Tropical Medicine, London WC1

[1] Mather, H G, *et al*, *British Medical Journal*, 1971, **3**, 334.
[2] Acheson, R M, and Sanderson, C F B, *British Heart Journal*. In press.

Dextropropoxyphene poisoning

SIR,—We should like to draw the attention of your readers to our recent experience of deaths following the ingestion of analgesic preparations containing dextropropoxyphene. The most commonly prescribed of these appears to be Distalgesic, each tablet of which contains 32·5 mg of dextropropoxyphene hydrochloride and 325 mg of paracetamol. The metabolic disturbances which may be induced by excess paracetamol are well documented, but the more immediate danger of acute respiratory depression due to overdosage with dextropropoxyphene appears to have escaped the attention of most doctors. Many seem to believe that preparations containing dextropropoxyphene are relatively innocuous and may be taken almost with impunity. They fail to appreciate the morphine-like narcosis which may follow overdosage.[1]

During the past three years we have

investigated 30 fatal cases in which we are convinced that dextropropoxyphene played the major part in the death. All these patients had taken Distalgesic tablets, some with suicidal intent and many in association with alcohol. The combined cerebral depressant effect of dextropropoxyphene and alcohol seems particularly hazardous, although all the victims had apparently ingested the analgesic in doses exceeding the therapeutic range.

It appears to us from the absence of published material in Britain that the problem has not yet been fully recognised, although recent information, not at variance with our own experience, is now reaching us from the USA.[2,3] We are at present in the process of collating our detailed results for publication, but feel that the time is opportune to highlight what we feel is an increasingly serious problem.

DEREK J L CARSON

Department of Forensic Medicine,
Institute of Pathology,
Belfast

E DENIS CARSON

Department of Industrial and
Forensic Science,
Belfast

[1] *Martindale: Extra Pharmacopoeia*, 26th edn, ed N W Blacow. London, Pharmaceutical Press, 1972.
[2] McBay, A J, and Hudson, P, *Journal of the American Medical Association*, 1975, **233**, 1257.
[3] Kopjak, L, and Binkle, B S, personal communication.

Snake bite in Britain

SIR,—It is perhaps not widely realised that snakes are quite common in many of the country areas and that the traditional means of identification are not always present.

The only poisonous snake in Britain is the viper or adder. It is found anywhere in the country, perhaps more commonly in the south, in Devon and Cornwall, and in south and west Wales. It can attack livestock as well as humans and it can be a considerable hazard in this respect to farmers. It may be confused with the grass snake, also called the "ring-collared" snake, or, less frequently, with the slow or blind worm. The appearance of the viper is subject to considerable variation. It may be coloured black, brown, dark green, grey, or almost white, with markings on its body varying from deep red to black. The under surface of the body may be black, brown, grey, or blue and the tip of the tail orange, red, or yellow. Its most constant characteristic is the zig-zag stripe running down the centre of the back, which is said to vary in only 2% of vipers. The inverted V-shaped mark on the head is less constant and may be X-shaped or indistinguishable, particularly in the black vipers. The male snakes grow to about 56 cm (22 in) and the females to about 76 cm (30 in).

The grass snake rarely occurs north of latitude 54°—that is, roughly along a line joining Lancaster and York. It is olive green or brown in colour, with transverse black bars on the back and vertical bars on the flanks. There is a yellow or orange collar around the neck, bordered behind with black. The under-surface of the body is black or white and black and it grows to 1-1·2 m (3-4 ft) in length. The grass snake does not bite. When alarmed it emits a foul-smelling liquid at the hind end of its body.

The slow worm or blind worm is not a true snake but a legless lizard. It is light or dark brown in colour and may have longitudinal stripes. It rarely exceeds 38 cm (15 in) in length, does not bite, and differs from snakes in having eyelids and an external ear opening.

I would therefore like to reinforce the suggestion by Drs D J P Barker and K J Foster (26 June, p 1591) that any snake bite should be attributed to an adder. There are, of course, occasional exotic snakes imported into the country, but this is a different problem. I have seen four snake bites in the past four weeks, only one of which was envenomated, but in addition to local swelling of the whole of the leg in this particular case there was generalised allergic reaction, which must not be confused with the general effects of absorbed venom. This particular patient had an allergic diathesis.

With regard to treatment, we have issued advice to the public in this area to the effect that the victim of the bite should be kept at rest, the bitten area kept as cold as possible, and transport arranged to the nearest large hospital. The severity of general symptoms appears to depend on the rapidity of absorption of the venom and these measures are intended to reduce the rate of absorption. In hospital we give prophylactic antihistamines, hydrocortisone, and a supportive intravenous drip. In the past 20 years I have had to use antivenom serum on only two occasions and in both these cases the bite was over the saphenous vein and venom appeared to have been injected intravenously. Now that human antitetanus serum is easily available I have added this to the treatment regimen in patients who are not otherwise protected against tetanus.

R VERNON JONES

Accident and Emergency
Department,
Royal Gwent Hospital,
Newport, Gwent

Amoxycillin, talampicillin, and ampicillin

SIR,—It is stated in the new (1976-78) edition of the *British National Formulary* (p 104) that amoxycillin should replace ampicillin because it is twice as well absorbed after oral administration. Amoxycillin possesses antibacterial activity differing little from that of ampicillin,[1] but has the theoretical advantage of producing higher serum levels. Nevertheless, it has not yet been shown that amoxycillin is superior to the same dose of ampicillin in the control of infective exacerbations of chronic bronchitis, which is probably the most frequent indication for the use of these two antibiotics. Amoxycillin is, moreover, very much more expensive. The basic cost of 100 250-mg amoxycillin (Amoxil) capsules is £8·91, while that of 100 250-mg ampicillin (Amfipen or Vidopen) capsules is only £3·11. I feel that the Editorial Committee of the *British National Formulary* should be asked to explain why they have categorically recommended an expensive new derivative of ampicillin when there is no evidence that ampicillin itself would not be equally effective.

Exactly the same argument applies to another derivative of ampicillin, talampicillin (Talpen), which is at present being vigorously promoted by another division of the same pharmaceutical company which markets Amoxil. It is claimed that talampicillin (which costs £6·50 for 100 250-mg capsules) is less liable to produce diarrhoea than ampicillin,[2] but the statistical evidence for this claim is unconvincing.

The patent rights on ampicillin have now expired and there has been a significant reduction in the price of this drug. Unless evidence can be produced to show that amoxycillin and talampicillin are clinically more effective than ampicillin itself, general practitioners and hospital doctors who prescribe the new preparations must be open to the charge of wasting public money.

IAN W B GRANT

Respiratory Unit,
Northern General Hospital,
Edinburgh

[1] Sutherland, R, Croydon, E A P, and Rolinson, G N, *British Medical Journal*, 1972, **3**, 13.
[2] Leigh, D A, *et al*, *British Medical Journal*, 1976, **1**, 1378.

Coeliac disease and diffuse pulmonary disease

SIR,—I was most interested to read of this case reported by Dr J Cummiskey and his colleagues (5 June, p 1401), in which coeliac disease, diffuse pulmonary shadowing, and avian precipitins occurred together in the absence of "exposure to known organic allergens" (presumably including birds). While the pleural thickening and airways obstruction have not, as far as I am aware, been previously described in this context, I know of three other observations that may be relevant to the suspicion that "the occurrence of coeliac disease, diffuse pulmonary disease, and positive avian antibodies may sometimes be a manifestation of disease separate from or additional to extrinsic allergic alveolitis."

Firstly, Lancaster-Smith *et al*[1] discovered three out of 24 patients with coeliac disease to have evidence of diffuse pulmonary disease (widespread radiographic shadowing and impairment of gas transfer) together with avian precipitins[2] but no known contact with birds.

Secondly, in two of the five patients we recently reported with bird-fancier's lung and jejunal villous atrophy[3] the pulmonary disease relapsed severely approximately 15 years after the initial episode, in the presence of avian precipitins, despite no known exposure to birds (over and above the small amounts we may all be exposed to in the atmosphere).

Finally, a higher than expected incidence of avian precipitins (to whole bird serum) has been observed in the serum from patients with coeliac disease by us[3] and others[2,4] (irrespective of bird exposure), and in three such individuals whom we looked at closely the carbon monoxide gas transfer factor (single breath method) appeared lower than expected by comparison with our own controls.

W T BERRILL

Professorial Medical Unit,
Southampton General Hospital,
Southampton

[1] Lancaster-Smith, M J, Benson, M K, and Strickland, I D, *Lancet*, 1971, **1**, 473.
[2] Lancaster-Smith, M J, 9th International Congress of Gastroenterology, Paris, 1972, Abstracts, p 398.
[3] Berrill, W T, *et al*, *Lancet*, 1975, **2**, 1006.
[4] Morris, J S, *et al*, *Lancet*, 1971, **1**, 754.

SIR,—It must not be forgotten that the coeliac syndrome and diffuse lung disease sometimes occur together in patients with sarcoidosis. At least three patients with sarcoidosis have been shown to have jejunal villous atrophy[1-3] and Kveim tests were positive in five of 17 patients with coeliac disease.[4] Moreover, a woman