Beta-adrenergic Blocking Agents in Patients with Renal Failure

SIR,—Dr.s Priscilla Kincaid-Smith and A. S. P. Hua are disturbed (24 August, p. 520) by our suggestion that beta-adrenergic blocking agents should not be used in patients with moderately severe renal failure (27 April, p. 193). They refer to our previous publication, in which we showed that temporary deterioration in renal function in such patients may occur with the introduction of any hypotensive drug. Their observations do not appear to be based on experience with one patient with malignant hypertension who was given 6 different drugs, and is not relevant to the cases described by us. We refer Drs. Kincaid-Smith and Hua to our paper, where they will find that the maximum change in diastolic blood pressure in any of the three cases was 10 mm Hg during the period that renal function deteriorated. Their comments do not shed any light on the mechanism of the events we described.

Drs. Kincaid-Smith and Hua will no doubt agree that renal function should be monitored during the course of therapy with hydralazine in overland travellers at risk of renal failure. Our experience leads us to suggest that this may be especially important when beta-blocking drugs are prescribed and needs to be emphasized now that these agents are being more generally promoted in general practice.—We are, etc.,

David J. Warren
C. P. Swainson
N. Wright
Medical Renal Unit,
Department of Medicine,
Royal Infirmary,
Edinburgh

Malabsorption in Overland Travellers to India

SIR,—Dr. S. Margaret Farquharson's interesting observations (24 August, p. 519) on the frequency of episodic diarrhoea and the use of sulphonamides, tetracyclines, and related agents in overland travellers are in general similar to our own (10 August, p. 380). A characteristic of treatment in our subjects was the repeated use of very short courses of therapy, which varied from one to a maximum of seven days on antibiotics. A number of patients did take metronidazole for varying periods, but this did not cure their symptoms.

We agree that amoebiasis should be excluded and that particular care is needed. The technique described by Alen and Ridley1 at the Hospital for Tropical Diseases ensures a high diagnostic rate by concentrating the amoeba in a faecal extract by 10% formalin and ether. None of our patients had the cystic forms of Entamoeba in their stools: these may, of course, be present in asymptomatic subjects who had had amoebiasis in the past. The vegetative forms of amoebae are usually associated with colonic and rectal invasion with symptomatic disease but no fat or xylete malabsorption. Coexisting amoebiasis was not considered a problem in our subjects since they had a normal mucosa on sigmoidoscopy as well as repeatedly negative examinations of the stool.

We are more concerned with the diagnostic problem of giardiasis than amoebiasis and now include microscopic examination of a smear from a jejunal biopsy2 as well as screening of jejunal aspirate and stool specimens before excluding giardiasis. We do not believe a clinical response to metronidazole can be used to infer either giardia or entamoeba infestation since metronidazole also has antibacterial properties.3 Furthermore, we are finding a considerable number of patients with giardiasis who prove resistant to metronidazole treatment.

Dr. Farquharson's suggestion that metronidazole may be a non-specific agent of benefit in the treatment of the cases of malabsorption has been considered, but our preliminary information does not support this suggestion.

We thank Dr. Farquharson for re-emphasizing the importance of achieving a complete diagnosis in this type of patient, who is often reluctant to be investigated fully.—We are, etc.,

A. Tomkins
w. p. t. James
Clinical Nutrition and Metabolism Unit,
Hospital for Tropical Diseases,
London N.W.1


Wartime Penicillin

SIR,—I feel one statement in the Personal View of an “angry young man” (10 August, p. 407) requires a rebuttal. Dr. Terry Hamlin in an interesting article among other things says he is reminded of the “wartime doctor forced to watch children dying of pneumonia while his penicillin couch and them to cure the troops’ gonorrhoea.” As this statement has been repeated in other places I might be allowed to put the true facts on record.

When it was decided that all the available penicillin should be allotted to the army it was handed over to a small research team. This was a combined R.A.M.C. and M.R.C. project and in this team I remember well the specific and rigid instructions that I received. There were three conditions in which penicillin was not allowed, and one of these was gonorrhoea, and this rule was firmly adhered to. Later, of course, when penicillin became available from American sources I am not in a position to say how it was used.

One of the reasons it was not used for venereal disease was the obvious one of it being in short supply and the other more scientific one was that a full evaluation of it had already been carried out by Florey in Oxford. I know of no child dying of pneumonia when penicillin could be given to troops to cure their gonorrhoea. I feel it is necessary to state this as the article in question has been given rather unfortunate publicity.—I am, etc.,

Ian Fraser
Belfast

Tapeworms and Isolation

SIR,—The article on an isolation system for general hospitals (6 April, p. 41) lists tapeworm among the infecting agents which do not require any isolation. This is true for Taenia saginata (beef tapeworm), but the only tapeworm of human importance endemic in Britain. Cysticercosis, including cerebral cysticercosis, results from the ingestion of ova liberated from segments of Taenia solium (pork tapeworm). Segments escape from the anus and may lie in bedclothing, so it would be well for nurses to exercise a degree of isolation similar to that for amoebic dysentery, when cysts convey the infection.—I am, etc.,

Frederick J. Wright
Moshi, Tanzania

Hypertension and Myocardial Infarction

SIR,—I was most interested to read Dr. J. McD. G. Stewart's letter (27 July, p. 251) in reply to your leading article (5 January, p. 1). I have recently concluded a three-year study of 363 patients in general practice, all suffering ischaemic heart disease, which concerns principally the infarction rate and death rate in these patients in relation to their treatment. The results confirm my own observations1 of the beneficial effects of beta-block treatment.

I have also analysed the same group of patients relating history of raised blood pressure to infarction rate and death rate from infarction. Taking the arbitrary borderline blood pressure of 140/90, those with pressures above that level suffer nearly 50% more infarctions, 50%, more fatal infarctions, and more than double the number of sudden deaths than those in the group with blood pressures below this level. It is interesting to record that the rates for both these groups—above or below 140/90—on beta-block treatment are virtually the same but only half that of the low blood pressure group not on beta-blockers. Sudden deaths in this group vary little from those in the low blood pressure group not on beta-block treatment.

So if the two groups with blood pressures below 140/90 are compared, we can say that those not on beta-block treatment have an infarction rate nearly four times that of those on beta-block treatment, more than four times the fatal attack rate, and more than three times the number of sudden deaths. If those with previous infarctions are excluded the differences are even more marked.

My conclusions so far, then are that there are significant aetiological relationships there is a definite increased risk of infarction and death in those with raised blood pressures compared with a similar group with “normal” blood pressures. Second, there is a very significant beneficial effect on the infarction rate when beta-block drugs are used in treatment. As Dr. Stewart says, the beneficial effects of the treatment of hypertension have been proved—first from fact to hypothesis to fact— and not from hypothesis to fact.

Evidence from the Framingham survey2

1 British Medical Journal: first published as 10.1136/bmj.3.5932.685 on 14 September 1974. Downloaded from http://www.bmj.com on 13 September 2023 by guest. Protected by copyright.