and they use their nebulizers every hour or so . . . they become tolerant very quickly. . . . My standard practice is to regard this excessive use of the nebulizer as evidence of deteriorating asthma and therefore to increase the corticosteroid maintenance dose as soon as the number of inhalations exceeds 8-10 in 24 hours."

It would therefore be interesting to compare the increase in asthma mortality observed in some countries with the frequency and practice of using corticosteroids in asthmatic patients, since the reluctance on the part of many doctors to use steroids is well known and one-half of the asthmatics who died in Great Britain (1961-6) were found *not* to have been treated with steroids in their final illness.⁸ In support of the possibility that a decreased use of steroids was related to the increase in mortality rate is the finding of Gandevia⁹ that steroid prescriptions were decreased in one Australian state where the asthma mortality doubled and were two to three times greater in another state where the asthma mortality increased by only 50%. After corticosteroids were introduced into England and Wales in 1952 the number of deaths due to asthma decreased progressively, from 1,879 in 1952 to 1,507 in 1957.¹⁰ Thus one wonders whether or not the use of steroids decreased in England and Wales from 1959 to 1966, when deaths due to asthma increased from 1,214 to 2,040?

Dollery stated¹¹ about the situation in Britain that "there was roughly a doubling of hospital admissions from asthma . . . after the warnings . . . because physicians . . . were recognizing that deteriorating asthma was a more serious condition than they had realized." These sentiments have also been expressed by Gandevia¹² and Harris.⁷—I am, etc.,

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Alcoholism Research

SIR,—Government research policy in regard to alcoholism was brought up in the last paragraph of your leading article (22 September, p. 602) reviewing recent studies of outcome. At a time when the Department of Health and Social Security is devoting more attention to alcoholism than ever previously, I find your remarks a little unkind. Why should not a systematic follow-up of the research reviewed in your editorial find financial support from the D.H.S.S.? I guess that it would, as would "a systematic follow-through of . . . any other research" be able to attract support from the D.H.S.S. or the Medical Research Council. There is

a dearth of proposals for research in the field rather than a lack of support.

Government research policy seems to be no more "haphazard" with regard to alcoholism than could be said of any other field where many different avenues need pursuing, from enzyme studies in rats to social studies in Camberwell. Co-ordination between such disparate approaches must necessarily remain loose without deserving to be called "little integrated" and "haphazard." The D.H.S.S. is certainly very concerned with developing systems for monitoring alcoholism and is now jointly with the M.R.C. looking at potential research over the whole field. The addiction research unit at the Maudsley Hospital and the Institute of Psychiatry is largely funded by these two bodies and we continue to hope for knowledge from this source. It would be a pity, therefore, if you were to give readers the impression that the appropriate Government bodies were unconcerned with the problem of alcoholism research.—I am, etc.,

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Loud Knocking on the Back Door

SIR,—I was somewhat taken aback to read in the *B.M.J.* (6 October, p. liii) a large and expensive advertisement in the personal columns headed "Consultant Surgeon." In this the advertiser openly solicited a medical school place for his son, whose "A" level grades were below the usual entry requirements of most medical schools.

That a reputable journal such as the *B.M.J.* accepted and published this advertisement, the nature of which must cause offence to many people, surprised me. Indeed, my colleagues and I found the wording of this piece so outrageous that we seriously doubted its authenticity.

I do not pretend that entrance to medical schools via the "back door" does not exist, but never before have I seen it exhibited so blatantly. As the *B.M.J.* is regularly scanned by the general press, I should have thought that advertisements of this kind were inviting public criticism of both this journal and the medical profession as a whole.—I am, etc.,

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Intermediate Coronary Syndrome

SIR,—I read with interest your leading article on this subject (22 September, p. 601). Referring to the use of anticoagulants in this condition it is stated: "The rationale of using these drugs is now doubtful and conflicts with modern observations on the low incidence of coronary thrombosis in myocardial infarction."

I have no argument with the first part of this statement but I must disagree with the last part. It is not my experience, or that of my colleagues, to find a low incidence of coronary thrombosis in fatal cases of myocardial ischaemia; in fact, the opposite is true. In 1970 Professor G. L. Montgomery and I published a paper¹ on a two-year study of the cardiac lesions in fatal cases of myocardial ischaemia from the coronary care

unit of the Edinburgh Royal Infirmary. Of 100 cases examined, coronary artery occlusions were found in 88, with thrombosis forming at least part of the occluding lesion in 71. Examination of a large number of similar cases from many sources over the subsequent years indicates no significant change in these figures.—I am, etc.,

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Delayed Attacks of Malaria in Visitors to the Tropics

SIR,—I have a high regard for Professor L. J. Bruce-Chwatt, but I am puzzled by some of his comments (22 September, p. 637) on the article by Dr. Paul Horstmann (25 August, p. 440). In particular, I do not understand his references to insufficient dosage or his reasons for saying that the suppressive dose given to Dr. Horstmann's subjects "was manifestly insufficient." The suppressive dose, 300 mg of chloroquine base once weekly, was adequate to prevent any manifestation of malaria in 90 persons so long as it was being taken, and it seems to have been continued for about four weeks after the last probable exposure to infection. It is true that six of the 90 persons then developed attacks of vivax malaria after latent periods ranging from 7½ to 10 months, but surely this is, in Professor Bruce-Chwatt's own words, merely "a salutary reminder of the well-known fact that chemoprophylaxis with chloroquine must not be mistaken for causal prophylaxis (especially when it comes to *Plasmodium vivax*)."

My own point of view on this subject can be stated briefly.

(1) The suppression of malaria with chloroquine alone is incapable of eradicating all *P. vivax* infections, whatever the suppressive dose used. Provided that the parasites are not drug-resistant, the continuation of dosage for four weeks after the last exposure to infection should eliminate the risk of an attack of falciparum malaria, but it will not prevent the possibility of an attack of vivax malaria.

(2) If an attack of vivax malaria does occur, the time interval between its onset and the cessation of suppressive dosage will be determined largely by the characteristics of the infecting strain of *P. vivax*. The strains typified by the New Guinea Chesson strain have a pattern of short latent intervals, and with one of these the attack is likely to occur within a few weeks; with Korean and other—mainly temperature zone—strains long latent periods are common, and the attack may not occur for 6 to 14 months, though many will occur at about 10 months. Between these two extremes there are other strains mainly in tropical areas, with latency patterns that are irregular and less well defined.

(3) In practice, therefore, vivax malaria must be considered as a possible cause of fever in anyone who has visited a malarious area during the previous 14 months or so, especially if chloroquine or another 4-aminoquinoline was used as the sole suppressive drug.

(4) Though not so relevant to the present discussion, it is worth noting that a radical