Correspondence

Letters to the Editor should not exceed 500 words.

Publicity for Ambilhar

SIR,—The announcements in the lay press concerning the drug Ambilhar recently produced by Ciba have aroused a great deal of public interest, which is causing an increasing number of inquiries concerning it to be directed to persons like myself. This produces considerable embarrassment, for the accounts published in the lay press have made to date virtually no reference to the toxicity of the drug and have inferred that it is safe for general use. In this connexion there are three points which need the firmest emphasis. The first is that no paper permitting critical appraisal of the toxicity of the drug in man has yet appeared in a major medical journal.

Secondly, Ambilhar has not been submitted to the Committee on the Safety of Drugs. As there are many patients with schistosomiasis continually arriving in this country, surely it is premature on the part of the manufacturers to publicize the drug

generally in Britain before it has been designated as safe for use?

The third point is that the drug has been found to inhibit spermatogenesis in animals, and the relevance of this to man has not yet been assessed. It may be that fears concerning this or other toxic effects will prove groundless, but the absence of published evidence indicates the difficulties created by the press announcement.

It is to be hoped, Sir, that you will use your very considerable influence in this field to encourage manufacturers in future to submit their products to critical examination through the media of widely available scientific journals before they influence the lay public and create a demand for the drug by announcements in the general press.—I am, etc.,

A. W. WOODRUFF.

Hospital for Tropical Diseases,
London N.W.1.

Compulsion in the Treatment of Alcoholics

SIR,—As a member (until the final draft) of the B.M.A. committee which submitted evidence on offences associated with alcohol and drug-taking to the Royal Commission on Penal Policy, may I dissent from some of the principal recommendations? I had hoped that the committee's report would be published in the journal, but now understand that it may not be.

The committee seeks additional legislation to compel alcoholics to accept treatment in a new range of security hospitals, under lay administration, "to which a person may be committed by the courts for a minimum period, say three to six months." It hopes by these means to avoid the stigma of prison for sick persons and (oddly enough) to avoid the wasteful use of security accommodation, and it seeks "to establish the principle of differentiation between the sick person and the criminal."

There is an obvious problem in the stage army of alcoholics circulating, in self-destructive manner, round courts and prisons, and there is an understandable urge to force a solution through legislation; yet compulsion is a double-edged weapon and expensive in human effort and resources; it should be a final resort when voluntary measures have been tried and failed. Voluntary help in acceptable form has certainly not yet been adequately tackled. Furthermore, the new compulsory powers are for the use of courts and would not help in the field; the workers in a church crypt, or the doctor in the psychiatric emergency clinic, can scarcely say, "Right ho, old man, I will see that you are charged at Bow Street to-morrow and put away for six months"; he wants somewhere

with a bath, food, warmth and welcome, under medical surveillance, and he wants it at 1 a.m. on a Sunday morning. Some will be incapable of accepting this sort of help; if mentally disordered they should go, or be sent, to psychiatric units (admittedly in short supply); if they are not mentally ill but are dangerous to society they should properly go to prison. The Mental Health Act of 1959 provides all the compulsion that any doctor could reasonably require. The argument that addicts cannot be detained solely on account of their addiction doesn't wash at all: nobody wants to detain them on those grounds alone, but because of the manifest damage they do to themselves and society and because of their wider psychopathic characteristics.

The committee says "a punitive approach will not cure the disease . . ."; but the courts, the prisons, and often the prisoners know full well that many prison sentences are in no way punitive, but simply filling a gap which the voluntary social services should bridge.

It is a hopeless and misguided task to try to distinguish between the sick person and the criminal. To lift favoured sectors from prison to hospital tacitly implies that what is left is just wicked and not deserving of medical attention, which is not the case. Once an addict is dried out there is little difference between his further needs and those of most prison recidivists. Since criminals and sick people are manifestly difficult to distinguish, it is logical that prisons and hospitals in their area of overlap should be equally indistinguishable, and this is recognized in the coming joint appointments of consultants and registrars to work in both institutions. To

set up a new and separate system of security hospitals (which would soon acquire a worse stigma than prisons) would not only be totally impracticable but would delay the potentially much more valuable approximation of prisons and hospitals, a move which stands to benefit all prisoners rather than just addicts.

Finally, why does the B.M.A. produce these smooth reports from which all differences of opinion have been suppressed? Royal Commissions want to hear heated arguments—they can make the final judgments for themselves.—I am, etc.,

Maudsley Hospital,
London S.E.5.

P. D. SCOTT.

Methyldopa in Hypertension

SIR,—Dr. P. Johnson and his colleagues in their important paper on the treatment of hypertension with methyldopa (15 January, p. 133) list among the side-effects 22 cases of temporary drowsiness and 19 cases of persistent listlessness out of 114 cases. In your leader in the same issue (p. 119) drowsiness, it is stated, usually lasts only a few days. These statements I think fall short of the mark, as can easily be found when treatment with methyldopa is discontinued for any reason: the patients always tell you that they feel much more alert.

I have rarely found a patient engaged in one of the professions or in an occupation involving mental alertness who could tolerate treatment of methyldopa for any length of time. In addition, since a very alert colleague, who had taken methyldopa for experimental purposes, told me that he had nearly been involved in a car accident on two occasions through slowness in applying the brakes, I have made it a practice to avoid treatment with methyldopa of patients engaged in driving all kinds of public vehicles or who had to drive for a living. The matter is of such public importance that it should be investigated in a department of experimental psychology.—I am, etc.,

Whittington Hospital,
Highgate Hill,
London N.19.

E. MONTUSCHI.

Antibiotic Vaginitis

SIR,—For some years I have been perturbed at the number of cases of antibiotic vaginitis referred to me. They fall into two groups:

(1) Those which develop as a side-effect after justifiable antibiotic therapy for some condition other than gynaecological. These must be accepted, though there is great need to know which antibiotics are most prone to cause this side-effect.

(2) Cases treated—mostly by general practitioners but also to some extent by other consultants in other specialties—as a result of sending a vaginal swab to the laboratory "for organisms and sensitivity test." These may be treated by local pessaries or systemic antibiotics. This is not only unjustifiable but

positively harmful (except in a small minority of cases), and should be condemned.

I have seen several such cases. The report usually consists of a scant or moderate growth of *Escherichia coli*, non-haemolytic streptococci, *Staphylococcus albus*, etc.—well known to be normal vaginal inhabitants. I have had one of the worst cases whose report was “abundant growth of Döderlein’s bacilli”—treated by tetracycline.

These cases are sometimes complicated by a secondary monilia infection—a well-known side-effect and usually, but by no means always, responding to appropriate therapy. The abacterial vaginitis is the real problem. In my experience no therapy except time is effective, and there may be much misery and discomfort lasting perhaps three, or even six, months before it clears up.

I asked the Committee on Safety of Drugs to undertake investigations into this problem, which I have not seen previously referred to in the literature, but they are unable to do this. They suggest that a letter such as this should be published.—I am, etc.,

Harrogate. C. RUTHERFORD MORISON.

Goodpasture Syndrome

SIR,—I was very interested in the article on idiopathic pulmonary haemosiderosis and the Goodpasture syndrome by Dr. Janet L. Elder and her colleagues (13 November, p. 1152). They clearly stated that it is still uncertain whether the Goodpasture syndrome is simply a variant of idiopathic pulmonary haemosiderosis or represents a distinct disease entity. Another possibility is that it may be an early stage of polyarteritis nodosa.

In my lecture delivered on 8 October 1965 at the Koranyi Society’s Congress in Budapest I suggested that the Goodpasture syndrome is a misnomer, and that it comprised pulmonary haemorrhage and fatal glomerulonephritis in young men. Moreover, though there are exceptions,¹ it is seldom very difficult to differentiate it from polyarteritis nodosa. I think that “mixed” cases also occur. The Goodpasture syndrome is simply a variant of idiopathic pulmonary haemosiderosis—a view also held by Rusby and Wilson.² It was erroneously stated in the article that pulmonary haemosiderosis and glomerulonephritis does not occur in childhood. There are some nine cases, but the prognosis is better than in adults. Ten years ago I³ put forward the hypothesis that idiopathic pulmonary haemosiderosis—which I called “immuno-allergic pulmonary haemorrhage”—is an immuno-allergic disease.³

I treated it as a monosystemic illness, but in the case described by Zollinger and Hegglin⁴ there were many autoimmune signs—including “pulmonary haemosiderosis,” Schönlein-Henoch purpura, and “obscure” anaemia. There is reason to believe that glomerulonephritis also belongs to this category.

The prognosis of immuno-allergic pulmonary haemorrhage is not so grave as has until recently been regarded. With treatment the patients with immuno-allergic pulmonary haemorrhage live longer as an average of three years from the date of diagnosis. Steroid treatment alone cannot now be regarded as either adequate or safe. Review of the follow-up results in seven cases of immuno-allergic pulmonary haemorrhage shows 5–13 years of survival since splenectomy, but of 17 splenectomized patients with immuno-

allergic pulmonary haemorrhage three died suddenly, some months after surgery.⁵

We have recently tried azathioprine (Imuran) in a child with immuno-allergic pulmonary haemorrhage who had not improved on steroids for two years. The patient has now shown much improvement in the last two years. Before treatment with azathioprine he was bedridden. Now he is working, though the sputum shows iron-containing macrophages when the drug is not being taken. He has remained free of symptoms and signs of the disease, and no side-effects were seen. At present we have in the ward a boy aged 14 years with the clinical diagnosis of immuno-allergic pulmonary haemorrhage who underwent splenectomy nine years ago. Since no pulmonary x-ray changes were visible anaemia was absent, vital capacity, forced respiratory volume, and ventilation-equivalent were all normal—but siderophages were found in the sputum—lung biopsy was undertaken. Sections showed haemosiderosis with haemosiderin-laden macrophages. We have now started treating him with azathioprine.

Thus, in summary, I suggest that the best treatment for patients with immuno-allergic pulmonary haemorrhage is azathioprine, combined when necessary with steroids.—I am, etc.,

Department II of Paediatrics, B. STEINER.
Postgraduate Medical School,
Budapest, Hungary.

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- 2 Rusby, N. L., and Wilson, C., *Quart. J. Med.*, 1960, **29**, 501.
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Tropical Diseases in Britain

SIR,—The letter by Professors G. Macdonald and A. W. Woodruff and Dr. B. B. Waddy (6 November, p. 1122) raises several interesting points.

There is no doubt that patients who have been to the tropics carry a risk of developing a complication of a tropical disease such as trypanosomiasis or schistosomiasis. However, in my opinion a distinction should be made between true tropical diseases and illnesses such as amoebiasis—the latter being more common in the tropics but occurring with a fair incidence in all parts of the world.

The two patients whom Macdonald, Woodruff, and Waddy cite with amoebic liver abscesses had been to Portugal and Spain, which after all are not tropical countries. Hundreds of thousands of tourists every year visit the southern countries of Europe, where amoebiasis is endemic, and it is always possible for them to develop an acute form of this disease, such as dysentery or liver abscess. Amoebiasis in chronic forms, on the other hand, occurs not only in countries with temperate climates but also in countries with colder climates—for example, in Scotland, in people who have never left the British Isles,¹ and the majority of papers published on amoebiasis are reports of cases where the diagnosis has been missed.

May I suggest that it is time to realize that amoebiasis is a disease with a universal incidence? If this were generally accepted, then doctors would be cognizant with its clinical pictures and would therefore consider the possibility of amoebiasis in everyday

practice. This would be a much more practical attitude than to refer to the institutes of tropical medicine undiagnosed patients who have been to countries with a warm climate. In these days of affluent living it will soon be difficult to find any patient who has not travelled south.—I am, etc.,

Evangelismos Medical Centre, TH. DOXIADIS.
Athens.

REFERENCE

- 1 McAllister, T. A., *Brit. med. J.*, 1962, **1**, 362.

Hypertension and Cerebral Infarction

SIR,—In defining patterns of cerebral damage Dr. J. Prineas and Dr. J. Marshall (1 January, p. 14) divide a series of patients by the criterion of whether the diastolic pressure was found to be above or below 110 mm. Hg “at the time of their admission with the last stroke.”

One may wonder what sort of shape these patients were in. How had their neurological deficits affected them? Were they agitated, resting, asleep, or in coma? There can be no need to emphasize the part played by mental and physical activity in determining blood-pressure. Even among normal subjects readings are likely to vary by at least 30% during a period of 24 hours.

In so far as the figure by itself has any significance it must surely be the level of pressure under standard conditions before the onset of some cerebrovascular lesion that determines whether, or in what degree, a patient should be regarded as hypertensive.—I am, etc.,

Victoria Hospital, I. MCD. G. STEWART.
Blackpool.

Familial Incidence of Cerebral Aneurysms

SIR,—I note that in their paper (15 January, p. 147) Mr. B. G. Chakravorty and Mr. C. A. Gleadhill come to an incorrect conclusion. They suggest that “the finding of such aneurysms in siblings and parent-offspring suggests that the weakness of the vessel wall is transmissible.” They describe these “weaknesses” as consisting of a defect of the media and the elastic lamina. They do of course consist of a defect in the media only, and they exist in over 80% of the cerebral arterial forks of every single person and also a large number of animals.¹ It appears unlikely that such a common denominator could account for familial cerebral aneurysms, unless particularly large or fragile defects were inherited.

Five of the six patients in their paper had a diastolic blood-pressure of 90 mm. or more. This is suggestive, though not conclusive, evidence that there was some degree of hypertension, and it is most likely that this was the inherited factor, as with most of the cases in the literature.

I have found that hypertension does enlarge cerebral arterial medial defects and results in disruption of the elastica covering them, and these findings will be published shortly.—I am, etc.,

St. George’s Hospital, M. R. CROMPTON.
London S.W.1.

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- 1 Glynn, L. E., *J. Path. Bact.*, 1940, **51**, 213.