Correspondence

Letters to the Editor should not exceed 500 words.

Amphetamine Prescribing

SIR,—Scarcely a day passes without news of another drug haul from a chemist's shop. Thousands of tablets are stolen, and the majority are probably amphetamines.

A glance at the Proprietary Drug Lists will show there are at least twenty-two preparations containing amphetamine alone or in combination with other drugs. Many are virtually identical and some preparations appear both as central nervous system stimulants and sedatives and tranquillizers. The chemist is obliged to keep a fairly large stock in order to meet all requirements. It is not necessary to reiterate the undesirability of these drugs being self-administered nor the consequent dangers of addiction, of toxic effects, and of antisocial behaviour, and yet the illegal use of amphetamines is increasing and constitutes a serious problem, and the stocks available in the chemists are one important source of supply.

How much do we contribute to this problem by continuing to prescribe the drug when its use is unnecessary or outdated? It is my belief that all these preparations could be scrapped with very little loss to the medical armamentarium. For mild depression there is now a very good range of antidepressants, more effective than temporary mood elevation, which is the way amphetamines work. For obesity there is a series of appetite suppressants claimed not to have central stimulating action, and for those patients who demand a tonic a pep talk might be more useful and certainly less potentially harmful than a pep pill (assuming, of course, there is no physical cause for debility).

My experience in general practice convinced me there were far too many women habituated, if not actually addicted, to amphetamines ostensibly for the treatment of obesity. As a psychiatrist I have found it possible to avoid medication with amphetamine, except for abreaction purposes and the occasional rare case of narcolepsy. If we all tried to cut down our use of these drugs as much as possible smaller stocks would be needed, and one source of supply would be reduced.—I am, etc.,

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Mycobacterium tuberculosis in Pasteurized Milk

SIR,—During the war years 1939–45 the high-temperature short-time (H.T.S.T.) process of pasteurization rapidly replaced the holder process, and this new process was officially recognized in 1941 by the issue of provisional regulations.

The minimum time/temperature condition of this new process was at first 15 seconds at 162° F. (72.2° C.), but in 1949 this minimum temperature was lowered to 161° F. (71.7° C.), and this minimum requirement has since remained unchanged. These particular conditions were carefully chosen to give a safety margin in respect of the destruction of *M. tuberculosis*, and a phosphatase test was introduced by the Ministry of Health in 1944 as a statutory check on the heat-treatment process.

However, at the end of the war it was considered desirable to confirm the absence of *M. tuberculosis* in such pasteurized milk. Accordingly samples of high-temperature short-time pasteurized milk from five processing depots have been examined at regular intervals, and this examination continued over a period of 20 years.

In addition to submitting each sample to the phosphatase test as a check on the heat-treatment, all samples were examined for the presence of *M. tuberculosis* by inoculation into guinea-pigs (two animals per sample), except during the last four years, when, instead, a direct bacteriological examination of the milks was made. The results of the tests for the presence of *M. tuberculosis* are shown below and indicate that in only one sample out of the total of 1,759 samples examined was there any definite evidence of the presence of this organism.

Year	No. of Samples Tested	No. Found Positive (i.e. Containing *M. tuberculosis*)
1945	79	0
1946	127	0
1947	148	1
1948	160	1*
1949	153	0
1950	159	0
1951 to 1956	70/year (average)	0
1957 to 1961	50/year (average)	0
1962 to 1965	60/year (average)	0
Total	1,736	2 (*1 using 1 guinea-pig only)

The results of the tests made during the first two years of the survey were reported by E. B. Anderson in the *British Medical Journal*.[1] All samples of pasteurized milk proved negative and all passed the phosphatase test. Confirmation of this satisfactory position appears in a survey published in 1953 by J. G. Davis,[2] and again in a report on the biological testing of milk recently published by the Public Health Laboratory Service.[3]—I am, etc.,

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2 Davis, J. G., *Dairy Ind.*, 1953, **18**, 210.
3 Public Health Laboratory Service, *Mth. Bull. Minist. Hlth Lab. Serv.*, 1965, **24**, 34.

Nitrites and Nitrates in Ischaemic Heart Disease

SIR,—I read your "Today's Drugs" article (11 March, p. 617) with great interest. I feel, however, that I must take issue on two points. You indicate that the "beneficial effects of nitroglycerin in angina of effort are undoubted," but I think this requires qualification in so far as it is only the prophylactic value that is undoubted. I did in fact doubt its therapeutic effect in treating an established attack of angina and found this doubt amply justified by the results of an objective study which showed clearly that nitroglycerin neither shortened the duration of anginal pain after exercise nor, more important still, favourably influenced the ischaemic changes in the electrocardiogram compared with a placebo.[1]

Which brings me to my second point, that I find it difficult to accept your dismissal of the exercise tolerance test as being of little value in assessing a new antianginal drug, and your emphasis on subjective assessment. Apart from the well-established unreliability of anginal subjects in assessing new drugs, which you quite correctly point out, the fact that angina improved with a drug (even when assessed on a controlled double-blind basis) does not indicate that the improvement is due to a reduction of myocardial ischaemia. In fact the reverse may occur, as I found when I evaluated pheniprazine, a monoamine oxidase inhibitor, in angina—the angina certainly improved and exercise tolerance increased, probably as a result of a central interference with pain perception, but ischaemic changes in the electrocardiogram became worse.[2]—I am, etc.,

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REFERENCES

1 Sandler, G., Ilhai, M. A., and Lawson, C. W., *Lancet*, 1963, **1**, 1130.
2 —— *Brit. med. J.*, 1961, **1**, 792.

Urinary Leucocyte Excretion in African Subjects

SIR,—As a recently returned lecturer from the Department of Surgery, Mulago Hospital, Kampala, Uganda, the article by Dr. D. C. Dukes and others (4 March, p. 537) has attracted my interest, and I hope to offer some constructive criticism.

My research work in Uganda was primarily concerned with retention of urine, complicated or caused by post-gonococcal urethral disease, in the African male. In such cases I have found white cell counts from 0 to 10,000