intense loneliness, having never been without a dog before. I am now hopeful she may now change her mind, at least to the extent of a Siamese cat, and that this may eventually permit corticosteroids to be tailed-off without relapse of her arteritis in one of its more unpleasant guises. This happened to a patient from the same practice only a few months back who stopped taking her prednisone, prescribed for her temporal arteritis, because the doctor emigrated. Her cardiac infarction, at first thought unremarkable, was followed by pericarditis and mental confusion, both of which, like her previous temporal arteritis, responded at once to prednisone.

It seems likely that further studies on deprivation by death or other means of close relatives or pets will show significant results, not only in mortality in the aged from cardiac infarction but also in morbidity and mortality from the other protean manifestations of giant cell arteritis.—I am, etc.,

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REFERENCE

1 Paulley, J. W., and Hughes, J. P., *British Medical Journal*, 1960, **2**, 1562.

Testing the Pill

SIR,—Your leading article (8 March, p. 592) confuses the issue of assessing the separate roles of oestrogens and progestogens in the contraceptive pill by calling norethisterone a progestogen, and thus implying that it is solely progestogenic. This is not so. Studies of its metabolism[1] and its clinical effect[2] clearly demonstrate some oestrogenic activity. It is most important in assessing these drugs to appreciate the exact biological as well as chemical effect of each component before assigning any side-effects to one or the other.—I am, etc.,

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REFERENCES

1 Brown, J. B., and Blair, H. A. F., *Proceedings of Royal Society of Medicine*, 1960, **53**, 433.
2 Appleby, B., *Lancet*, 1962, **1**, 407.

*** It is true that norethisterone is not purely progestogenic. The purpose of the leading article was to point out that the actions of progestogens and oestrogens can overlap, and to assign side-effects to one or other group of compounds on the basis of rather crude and ill-understood liver function tests is unwise. Only experimentation in animals can clarify the issue in a reasonable period of time.—ED., *B.M.J.*

Indomethacin Therapy

SIR,—In reply to the letter of Dr. J. H. Swallow (22 March, p. 783), we have been careful to avoid stating that gastric ulceration is caused by indomethacin. We felt that there was likely to be a causal relationship on the basis of the rapid healing which followed withdrawal of the drug in seven patients who had no gastric symptoms, either before or since taking indomethacin (21 December, p. 734).

In three patients who developed gastric ulcers while taking oral indomethacin, symptoms continued, and healing failed to occur when suppositories were substituted. In these cases healing occurred rapidly with relief of symptoms when suppositories were withdrawn, suggesting that they were at least perpetuating the process. We agree that no firm conclusions can be drawn from only three cases of such an unpredictable disease.—We are, etc.,

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Hyperbaric Oxygen in Carbon Monoxide Poisoning

SIR,—The controversy on this subject in your columns seems to be losing perspective. In our original paper[1] we stated that "the treatment of choice for patients with *severe* CO poisoning is to expose them to hyperbaric oxygen." In your leading article following this paper you amended our suggestion by saying that "where this is available, exposure to hyperbaric oxygen at 2–2½ atmospheres pressure is the treatment of choice."[2] Quite reasonably, Matthew and Proudfoot[3 4] object to this counsel on the grounds that the prognosis for CO poisoning is excellent with mannitol infusions and conventional oxygen therapy, without resorting to the more complex hyperbaric chambers.

Unfortunately the situation is not so simple, since, as Dr. J. G. B. Thurston has pointed out (15 February, p. 446), the importance of hyperbaric oxygen is not so much in reducing the already low mortality rate, but in reducing the very significant chronic neurological and other sequelae. Permanent brain damage is rare,[5] but profound disability due to visual, dysphasic, dyspraxic, and complex agnosic defects may last for months or years[1] before a functionally useful degree of recovery has taken place. In deciding the optimal treatment in the acute stage of the illness, these facts should not be neglected. I have previously pointed out[6] that the main problem is the restricted availability of pressure chambers; and the difficulty in deciding where to send the patient must also be affected by the distance involved and the related time delay in instituting treatment. The level of carboxy-haemoglobin did not seem to be a reliable index in predicting the severity of sequelae in our patients,[1] and therefore this estimation on admission to hospital is of little help in deciding the need for hyperbaric oxygen.

I would agree with Dr. Thurston that patients should be transported to the listed centres where hyperbaric oxygen chambers are available, but would add the riders that this is contraindicated if:

(1) The distance to be covered in the ambulance unduly delays the inception of treatment. In this event conventional oxygen and carbon dioxide with mannitol infusion at the nearest hospital is indicated.

(2) The duration of exposure is short, the patient is young, and there is no impairment of consciousness or neurological deficit when the victim of poisoning is first seen. In this sort of case the risks of brain damage are remote.

These suggestions are of necessity generalizations, and, as I have previously stated,[6] the severe case, with profound impairment of conscious level, or with signs of acute multifocal cerebral cortical ischaemia,[1] should be given the benefit of hyperbaric oxygen at 2–2½ atmospheres for approximately two hours.—I am, etc.,

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REFERENCES

1 Garland, H., and Pearce, J., *Quarterly Journal of Medicine*, 1967, **36**, 445.
2 *British Medical Journal*, 1968, **1**, 398.
3 Matthew, H. J. S., and Proudfoot, A. T., *British Medical Journal*, 1968, **1**, 638.
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5 Shillito, F. H., Drinker, C. K., and Shaugnessy, T. J., *Journal of the American Medical Association*, 1936, **106**, 669.
6 Pearce, J., *British Medical Journal*, 1968, **1**, 767.

Urinary Tract Infections

SIR,—The two papers reporting on the value of the trimethoprim-sulphamethoxazole combination in the treatment of urinary tract infections (1 March, p. 541 and p. 545) help to establish this compound as a chemotherapeutic agent of major importance. This will no doubt increase the number of requests for bacterial sensitivities, and in this respect I would like to draw attention to a possible source of error in the performance of such tests which could be overlooked.

The action of trimethoprim can be blocked by the presence of antagonists in much the same way as para-aminobenzoic acid antagonizes the action of sulphonamides. The composition of a medium used for testing sensitivity to the trimethoprim-sulphamethoxazole combination is therefore critical in that it must be free from both sulphonamides and trimethoprim antagonists, otherwise organisms which are sensitive to these drugs may appear to be resistant. Oxoid diagnostic sensitivity test (D.S.T.) agar, which is probably widely used, contains small amounts of trimethoprim antagonists, although it is free from sulphonamide antagonists. In order to make this medium suitable for testing sensitivity to trimethoprim it is necessary to neutralize these trimethoprim antagonists, and this can be done by the addition of 0·25% lysed blood.[1]—I am, etc.,

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REFERENCE

1 Darrel, J. H., Garrod, L. P., and Waterworth, P. M., *Journal of Clinical Pathology*, 1968, **21**, 202.

Diverticulosis of the Colon and Diet

SIR,—Wide regional variations are known to exist in the incidence of diverticula of the colon. Thus, although common in many parts of Europe and North America, the condition is rare among the negroes of Africa and among the native populations of India and of the Far East. In the U.S.A., however, the incidence of this condition among the whites and negroes is approximately the same. This phenomenon has been correlated with the type of diet ingested; colonic diverticula appear to occur in areas where a low roughage diet is eaten in contrast to their rarity when a bulky vegetable diet is used.[1]

Some support for this dietary aetiology was given by two reports on the development of