inguinal glands on both sides were easily palpable but were neither tender nor enlarged. There was no constitutional disturbance.

These rather unpleasant-looking lesions slowly resolved without treatment, and had disappeared six weeks from the date of referral.—I am, etc.,

J. R. E. JAMES.

Cardmarthen.

Cerebral Malaria

SIR,—I am interested in the recent correspondence (27 July, p. 250; and 14 September, p. 680) regarding cerebral malaria.

I spent some nine years in East Pakistan where *P. falciparum* is endemic and was able to treat a fair number of cases during that time. The most striking thing about a case of the disease is the rapidity of its deterioration once coma has set in. Many patients may come to a remote country hospital or dispensary from some distance in extremis. Need of treatment is paramount. Delays while lumbar punctures and drips are prepared may be fatal.

My earlier experience of using intravenous quinine as the sole parenteral medication was frightening. Two cases on successive days died. However, Manson-Bahr in his invaluable *Tropical Diseases* advocates the use of intramuscular adrenaline. From then onwards the standard treatment in our small hospital was as follows: Intravenous quinine dihydrochloride 10 gr. in 10 ml. distilled water given slowly over 10 minutes. Immediately thereafter 1 ml. 1:10,000 adrenaline intramuscularly over five minutes. We treated probably about a dozen cases without a single fatality by this method. It would appear that just as the steroids have supplanted adrenaline in bronchial asthma so they are doing in cerebral malaria.

The rationale of the adrenaline treatment would appear to be threefold. (1) It causes the spleen to contract, expelling parasites into the blood stream and facilitating the action of the quinine; (2) it causes peripheral vasoconstriction, and this aids the resuscitation of the patient who is always collapsed, with poor

peripheral circulation; and (3) it has an effect on the cerebral oedema mainly because of its vasoconstrictive action. Needless to say, other supportive measures such as ice bags and drips are necessary, but my experience would suggest that the consistent use of quinine and adrenaline saves time. The other measures will aid recovery.

Dr. T. Harding need not fear his budget will be stretched. Adrenaline is an essential item of equipment in every general practitioner's bag, let alone in a small hospital, and it is relatively cheap, compared with dexamethasone.—I am, etc.,

I. T. PATRICK.

Bilton,
Rugby.

Chemotherapy of Malaria

SIR,—I have to-day received the *B.M.J.* (13 July, p. 71) and read with interest the leading article on chemotherapy of malaria. It is certainly true that in Tanzania at least there have been no substantiated cases of malaria resistant to 4-aminoquinolines. There have, however, for a long time been many areas where there has been resistance to both pyrimethamine and proguanil, so much so that for many years we have completely given up using these drugs in any form of malaria, either for treatment of an acute attack or for chemoprophylaxis.

I think, therefore, it might have been best if this was explained in the second paragraph of your leading article, from which one would understand that pyrimethamine and proguanil should still be used as chemoprophylaxis. I would also have preferred the use of the word chemoprophylaxis or causal prophylaxis rather than chemosuppression. I feel sure that if proguanil and pyrimethamine continue to be used as prophylactics where resistance to these drugs has developed this will not only cause an increase in the incidence of resistant strains of *P. falciparum* but also considerable morbidity and loss of work. The literature is also clear that there have been a not-negligible number of fatal *P. falciparum* malaria in those who have used the above drugs as chemoprophylactics.

So far as our experience goes, we have continued to use the 4-aminoquinolines in both prophylaxis and treatment of malaria and have run into no trouble as a result of this. The position as regards malaria in Tanzania has been fully documented.—I am, etc.,

JOSEPH TAYLOR.

Mvumi Hospital,
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REFERENCE

- ¹ Clyde, D. F., *Malaria in Tanzania*, 1967. London.

Stress and Bronchitis

SIR,—Dr. A. C. Stenhouse (3 August, p. 287), reporting on acute exacerbations of chronic bronchitis, reports on 22 episodes of "non-specific bronchitis" confined to a few of the patients under study. We would like to point to an emerging factor of significance in the study of upper respiratory infection and other illness that may account for the selection of this and other subgroups,

given equivalent virological data and knowledge of their endocrine state. Though the age of the particular patients in question is not given, the data of Dr. Stenhouse's prior study (19 August 1967, p. 461) would place them above the age range of our study; however, this would appear to make the concept no less valid, given the state of knowledge about geriatrics.

Acute upper respiratory infection has come under closer scrutiny with current virological, epidemiological, and endocrinological study. Observation has shown that the average number of colds per person each year is remarkably constant, but that considerable variation is noted in the number of colds experienced annually by both individuals and families.¹ It has been postulated that genetic factors might govern innate susceptibility or resistance.¹ Host susceptibility, of whatever origin, appears to be a factor in the relative frequency of subclinical and clinical infections.² A mechanism of susceptibility involving endocrine function on a cellular level has been hypothesized.^{3,4} The recent endocrine balance study of Mason *et al.*⁵ showed a number of pre-illness hormonal changes, including an abrupt spike of 17-OHCS, epinephrine, and norepinephrine, and a gradual drop in butanol-extractable iodine. Sick subjects with upper respiratory infections differed from less sick by having a greater number of extreme values. The psychoanalytic literature has presented dynamic formulations that may bear on susceptibility, especially related to conflict situations,⁶ and M. Jackel before the fall 1967 meeting of the American Psychoanalytic Association implicated depression, as had others previously.

The menstrual cycle has well-documented phasic hormonal changes.⁷ As part of an ongoing study at this laboratory it was found that levels of characterological anxiety, or anxiety trait, were associated with physical-emotional-social impairment during the premenstruum. Inquiry as to the incidence of upper respiratory infection in the studied population of 161 female student nurses showed a significantly greater number of acute upper respiratory infections in the "high" trait anxious group than in the "low" anxiety group. This relationship to trait anxiety, as measured by the Taylor Manifest Anxiety Scale, was significant at the 0.01 level. Measures of situational anxiety, or anxiety state (the Scheier Cattell Anxiety Battery), and of depression (the "D" scale of the Minnesota Multiphasic Personality Inventory) were not significant in differentiating the populations with and without colds. There was not a significant correlation between colds and impairment independent of anxiety.

A synthesis of the data from various fields indicates that there should be a discriminating factor in the incidence of upper respiratory infection. The study of premenstrual tension has provided a naturally occurring "stress" that has documented hormonal changes that fit with previously delineated mechanisms of infection. Since all women undergo hormonal changes during the menstrual cycle, the factor of significance, however subtle it may be, is the influence of psychic response to such "stress." Response characteristics may accentuate or modify the pattern of hormonal change in the menstruum, thereby enhancing susceptibility. This accentuation or modification in the individual may be based on a characteristic response to acute stress of defined type. The observation of a significant relationship between trait anxiety and the incidence of upper respiratory infection represents a start toward the better definition of individual difference as a significant factor in the onset of illness. This finding can aid in understanding such findings as those of Dr. Stenhouse, where appar-