the bacteriological findings correlate well with those in that condition. The normal cell count in the CSF is rare in tuberculosis of the central nervous system but may occur.¹⁻³ We think that a positive culture for *M. tuberculosis* might be a more common finding, were one to look for it, and that the possible tuberculous nature of meningitis should not be discarded because of benign presentation only.

ETIENNE M GRANDJEAN
LUC HUMAIR
REMY G H CLOTTU

Service de Médecine,
Hôpital de la Ville,
La Chaux-de-Fonds,
Switzerland

HARALD MODDE

Institut neuchâtelois de Microbiologie,
La Chaux-de-Fonds,
Switzerland

¹ Emond, R T D, and McKendrick, G D W, *Lancet*, 1973, 2, 234.

² Taylor, K B, Smith, H V, and Vollum, R L, *Journal of Neurology, Neurosurgery, and Psychiatry*, 1955, 18, 165.

³ Kocen, R S, and Parsons, M, *Quarterly Journal of Medicine*, 1970, 39, 17.

Premenstrual tension

SIR,—Paraphrasing the opening sentence of the leading article, "Premenstrual tension" (27 January, p 212), I would say that there is nothing pleasant about inaccuracies, especially when they appear in such a well-written, well-documented, up-to-date article.

You cite two studies that "have failed to confirm any rise in prolactin concentrations in patients with premenstrual tension." This statement is incorrect, because Andersch *et al*¹ clearly report: "In patients with PMT prolactin levels were significantly higher premenstrually compared to levels during the follicular phase. In the control group no such increase was observed."

A careful examination of the second article cited² shows a very vague definition, selection, and mental evaluation of their sample group. According to table II in that article, the average severity of mental complaints among the group was 1.01—that is, slight—and they did not mention any control group for the drug-free cycle.

None the less, more than one variable seems to be implicated in the cause of the premenstrual tension syndrome, and, concerning the accumulating data, one may assume that we are dealing with a group of syndromes which may be caused by an imbalance between several factors affecting the central (and possibly CNS peripheral) nervous system.

A better understanding of the aetiology of these syndromes is unlikely before a precise definition is formulated.

URIEL HALBREICH

College of Physicians and Surgeons of
Columbia University,
New York 10032

¹ Andersch, B, *et al*, *Acta Endocrinologica (Copenhagen)*, 1978, 88, Suppl No 216, p 165.

² Andersen, A N, *et al*, *British Journal of Obstetrics and Gynaecology*, 1977, 84, 370.

Treatment of orthopaedic tuberculosis

SIR,—Absence overseas has prevented my commenting earlier on the letters of Sir John Crofton (6 January, p 52) and Mr M Wilkinson (24 February, p 558) on the treatment of orthopaedic tuberculosis.

I was astounded to read of the practice attributed by Sir John to "some orthopaedic surgeons" of giving no more than one month's chemotherapy to patients with tuberculous sinuses. The absence of any letter questioning the frequency of such maltreatment leads me to fear that Sir John has no need to feel "ashamed" of his letter, but rather that he deserves every support for urging, as I have always urged myself, that any surgeon treating any case of tuberculosis should do so in consultation with an expert in chemotherapy, who will almost always be a chest physician. The simple fact is that tuberculosis of bone and joint, though done nothing but harm by inadequate or inappropriate chemotherapy, will be cured by adequate chemotherapy in almost every case. Mr Wilkinson is quite wrong in asserting that this does not apply to disease of the thoracic or thoracolumbar regions of the spine, even if large paravertebral abscesses are present. Reference to the last two reports^{1 2} of the Medical Research Council's working party on this subject makes this abundantly clear.

Radical anterior surgery of the "Hong Kong" type, performed by experienced experts and reinforced by adequate chemotherapy, hastens cure and lessens deformity, thus conferring great benefit on the patient with spinal tuberculosis. The high expectation of eventual cure, however, is not increased even by this operation and certainly not by any lesser surgical measure such as that suggested by Wilkinson. Our studies in Bulawayo and in Hong Kong indicate without question that even a full debridement of the focus confers no worthwhile benefit on the patient receiving adequate chemotherapy.

D LL GRIFFITHS
Chairman,
MRC Working Party on
Spinal Tuberculosis

Eglwysbach,
North Wales LL28 5TY

¹ Medical Research Council, *Journal of Bone and Joint Surgery*, 1976, 58B, 399.

² Medical Research Council, *Journal of Bone and Joint Surgery*, 1978, 60B, 163.

Steroid treatment in giant cell arteritis

SIR,—In the reported discussion (17 March, p 727) on the first case in the clinicopathological conference held at the Royal College of Physicians questions were asked about steroid treatment, the sedimentation rate, and the age of patients affected by giant cell arteritis.

It might be helpful to recall observations made a quarter of a century ago and reported in the *British Medical Journal* (31 December 1955, p 1593). In case 1 of that report, the patient was aged 55 years at the time of onset of her symptoms and was first seen in medical outpatients in July 1954, when she was 56 years of age. The diagnosis of giant cell arteritis was confirmed by biopsy; her erythrocyte sedimentation rate (ESR) was 80 mm in the first hour. Oral cortisone was started, 200 mg being given daily during the first week. Thereafter the daily dose was 150 mg during the second, 100 mg during the third, 50 mg during the fourth, and 25 mg during the fifth week. After the first week of treatment the ESR fell to normal and remained so. A second biopsy performed five weeks after the first showed no inflammatory cellular infiltration.

In case 2 the initial ESR was 36 mm in the first hour. After 11 days' treatment with

ACTH gel by injection 25 mg three times a day a second biopsy specimen was totally free from cellular infiltration and treatment was stopped.

At that time we thought that these were the first cases reported in which serial biopsies had shown such histological changes. Our clinical and histological evidence suggested that cortisone and ACTH reduced the oedema and the inflammatory cellular reactions. This was probably the cause of the relief of symptoms. There was no evidence that the treatment had any effect on the scarring and intimal thickening that had already taken place; in fact in the first case thrombosis occurred despite treatment. It seems probable that it is thrombus that causes the arterial obstruction and symptoms after cortisone has been given, and it might be argued that there is an indication for using anticoagulants during and after cortisone therapy.

ROBERT J HARRISON

Goring-by-Sea,
West Sussex BN12 4LJ

Drugs and breast-feeding

SIR,—The British physician, at least, should not be ignorant about interference of drugs with breast-feeding, as you imply in your leading article (10 March, p 642). He may obtain for a mere 90p Dr Linda Beeley's marvellous booklet *Safer Prescribing*,¹ where, on page 37, most of the interfering drugs you mentioned and more are listed.

KARL H KIMBEL

Medicines Commission of the
German Medical Profession,
5000 Köln 41

¹ Beeley, L, *Safer Prescribing: a Guide to some Problems in the Safer Use of Drugs*. Oxford, Blackwell Scientific, 1976.

Cimetidine in acute upper gastrointestinal bleeding

SIR,—We write to report a trial of cimetidine in the treatment of upper gastrointestinal bleeding, and a negative result similar to that of Mr R G Pickard and others (10 March, p 661).

Patients admitted to the medical wards of a district general hospital with haematemesis or melaena were randomly allocated to a treatment or non-treatment group: the patients were stratified into two age groups, above and below 55 years. To those allotted to the treatment group cimetidine was given intravenously, 200 mg in 200 ml normal saline, over a period of two and a half hours, repeated six-hourly for 48 hours: this was followed by oral treatment, 200 mg three times a day and 400 mg at night, for five days. Except in a small number of cases, upper gastrointestinal endoscopy was performed within 16 hours of admission. Blood was transfused as necessary. Patients were referred for surgery if (a) there was no apparent improvement after transfusion of six units of blood or (b) deterioration continued during transfusion or (c) after an interval during which there was no sign of bleeding fresh blood was aspirated or there was evidence of hypovolaemic shock or rapidly progressive anaemia. The decision to refer a patient for surgery was made by a member of the team who did not know whether the patient was receiving cimetidine, but who had either performed endoscopy himself or was aware of the endoscopic findings.

Fifty-eight patients were allotted to the group treated with cimetidine and 55 to that without; in each group, eight patients were referred for surgery. In the group treated with cimetidine four