and could be traced in the records of the department of gynaecology. Of the 162 patients, 29 had cervical carcinoma stage 0 as diagnosed at conisation of the cervix and five had invasive cervical carcinoma stage I-II. One hundred and seventy-two age-matched controls were selected from the registers of births, marriages, and deaths and followed up in the same way as the patients. Seven cases of carcinoma of the uterine cervix stage 0 were found and one case in an invasive stage. The frequency of carcinoma was thus 20% in the patients compared with 5% in the controls—that is, four times higher in the patients who had previously had infection with *neisseriae*.

The results are in agreement with the general opinion that genital infections predispose to cervical neoplasia. Neither in the *neisseria* group nor in the controls was the frequency of herpesvirus infection known. We feel that estimation of the significance of herpesvirus infection and of the importance of possibly carcinogenic metabolites of bacterial origin requires well-planned long-term studies. Attention must be given also to other micro-organisms known to be of importance in genital infections, such as *Mycoplasma hominis*² and *Chlamydia trachomatis*.³

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² Mårdh, P-A, and Weström, L, *British Journal of Venereal Diseases*, 1970, **46**, 179.
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Epidemic myalgic encephalomyelitis

SIR,—From Dr H G Easton's letter (24 June, p 1696), I can only conclude that he ignored all the evidence at the symposium on myalgic encephalomyelitis at the Royal Society of Medicine that did not fit in with his theories. If he had stayed to the end, when a vote was taken, he would have found himself the only person present still in favour of the "conversion hysteria" theory.

Dr Pampiglione, for example, referred to the electroencephalographic abnormalities—which were quite different from those seen in an acute encephalitis or liver failure with encephalopathy. The abnormalities were also different from those in chronic psychiatric patients. The cerebral cortex appeared to be disturbed by an insidious illness which was worthy of further investigation. Dr Peter Behan said that lumbar puncture performed on different days could give different results. Abnormal lymphocytes had been found by Dr Dillon, and reference was also made to anticomplementary activity in the serum and the ability of lymphocytes to live in vitro for an extended period, as is found with lymphocytes from patients with known viral diseases.

Dr Easton's "vapour and virus" remarks are hardly worth serious attention. Failure to isolate a virus does not rule out the possibility of a viral illness, because viruses that are of low antigenicity could become non-detectable if they were incorporated into the host cell's DNA. I would suggest that before putting pen to paper again he talks to some of the nurses (who have never been adequately followed up)

permanently disabled as a result of the Royal Free Hospital outbreak. He might then discover how much unnecessary anguish the "conversion hysteria" theory had added to their mental and physical suffering. Or perhaps he would prefer to talk to some of the patients affected in the recent Essex outbreak, originally diagnosed as multiple sclerosis. Your leader admirably demonstrated the similarity between the two conditions. Perhaps Dr Easton thinks that multiple sclerosis is also hysterical in origin, since many of the symptoms, like those in myalgic encephalomyelitis, cannot be proved to be of organic aetiology.

Dr Peter Behan said at the symposium that he thought the disease was an immunological reaction triggered off by a virus. It would be helpful if more doctors, instead of pursuing outdated theories, seized the ample opportunities which study of this illness offers for research, a point which you rightly make in your leader.

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Leukostasis in chronic myeloid leukaemia

SIR,—In his recent paper (6 May, p 1169) on leukostasis in acute myeloid leukaemia (AML) Dr A L Harris draws attention to an important, avoidable cause of early death which is also relevant to other types of leukaemia. We have recently seen a similar event in a patient with chronic myeloid leukaemia (CML).

The patient was a 26-year-old man who presented with sudden deterioration of vision in one eye. Examination revealed several deposits with surrounding haemorrhage in both fundi and splenomegaly of 10 cm. Blood and bone marrow examination showed CML; his haemoglobin concentration was 6.8 g/dl, leucocyte count $500 \times 10^9/l$ ($500\,000/mm^3$) with a typical differential count, and platelet count $75 \times 10^9/l$ ($75\,000/mm^3$). Neutrophil alkaline phosphatase activity was absent and the Philadelphia chromosome was present. He was treated initially with busulphan 4 mg daily, but his condition deteriorated, with malaise, fever, pain in the splenic area, and some confusion. It was thought that these features reflected the very high leucocyte count, now $630 \times 10^9/l$ ($630\,000/mm^3$) and he was therefore transferred for leukapheresis (Hemonetics model 30). A blood count immediately before the procedure was as follows: haemoglobin 6.1 g/dl, leucocyte count $770 \times 10^9/l$ ($770\,000/mm^3$), platelets $100 \times 10^9/l$ ($100\,000/mm^3$). The leucocyte count immediately after leukapheresis had fallen to $560 \times 10^9/l$ ($560\,000/mm^3$) and a transfusion of 5 units of blood and 2 units (10 donations) of platelets was given. The following day the leucocyte count had not changed significantly ($550 \times 10^9/l$ ($550\,000/mm^3$)) and a second leukapheresis was carried out. Within 4 h the clinical features of a subarachnoid haemorrhage developed. The leucocyte count at this time was $300 \times 10^9/l$ ($300\,000/mm^3$). The patient's condition deteriorated and he died shortly afterwards. Permission for necropsy was not obtained.

Although no positive proof was obtained the clinical features and course of this patient were sufficiently similar to those described by Dr Harris to make cerebral leukostasis and haemorrhage the likely cause of death. It is evident that transfusion must be approached cautiously in any patient with a very high leucocyte count and preferably postponed until the count has been reduced to a level which does not significantly affect whole-blood viscosity. This can be achieved within a few days in AML but may take several weeks in

CML with traditional treatment. In these circumstances more vigorous chemotherapy or leukapheresis may be needed. Although in this case each leukapheresis produced a fall of over $200 \times 10^9/l$ ($200\,000/mm^3$) the leucocyte count still remained high enough for leukostasis to result from the combined haemodynamic effects of transfusion and the rapid blood volume changes inherent in the leukapheresis technique.

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Treatment of ulcerative colitis

SIR,—We wish to reply to the remarks made by Mr P G Bevan about the problems of colitis patients being treated in fever hospitals (3 June, p 1459). As a group associated with a so-called "fever hospital" we feel that the discipline of infectious diseases has a contribution to add to the subject of inflammatory bowel disease (IBD).

It is not widely appreciated that salmonella infection by itself can produce a severe inflammatory colitis with persistent bloody diarrhoea¹ and even, on occasions, toxic dilatation of the colon,² so it would be true to say that the distinction from IBD on clinical grounds is not possible. In fact the differentiation between IBD and salmonellosis in an acutely ill patient may not be readily solved by sigmoidoscopic or barium x-ray appearances or even by biopsy findings.³ We find that it is only by repeated assessment and repetition of the major investigations over a period of time that the diagnostic puzzle can be resolved, with all its implications for the management of the patient. To make matters worse, there are occasional patients in whom IBD of acute onset and salmonella infection coexist, and in these all the problems are compounded.

We feel that the specific bowel infections should be considered as part of the spectrum of IBD and, as in the case of ulcerative colitis and Crohn's disease, to produce good results it is necessary to have close co-operation at senior level between physicians and surgeons with experience of the problem.

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Spontaneous remission of nephrotic syndrome in amyloidosis

SIR,—I have read with interest the article by Drs J Michael and N F Jones on spontaneous remissions of nephrotic syndrome in secondary renal amyloidosis (17 June, p 1592). Spontaneous remission can also occur in primary amyloidosis. Indeed I have observed