ocular palsy (43%), facial palsy (19%), and lymphadenopathy (79%), which disappeared as the illness passed. Dr. McEvedy and Dr. Beard emphasize the presence of cramps and tingling, which they rightly say may be manifestations of hysterical overbreathing. In fact our patients had cramp but not carpo-pedal spasm and persistent tingling, not generalized, when they were not overbreathing.

Regarding the electrodiagnostic findings, Drs. McEvedy and Beard have selected for mention the absence of signs of lower motor neurone degeneration and the grouping of motor unit potentials on volition. There are of course a number of lesions of lower motor neurones which disturb function and do not produce neurone degeneration. There are a number of ways by which grouping of motor unit potentials can be produced. However, our patients did not appear to do so by a "maximum effort" in which agonist and antagonist counteract each other and by which method Drs. McEvedy and Beard attempted to simulate our electromyograph tracings. In fact sustained activity produced fatigue with breaking up of the groups. Further, we obtained (albeit exceptionally) groups of up to 100 milliseconds in duration which do not seem to occur in trace C of Fig. 2 (3 January, p. 10). Incidentally we have not claimed that any of the electrodiagnostic findings were specifically diagnostic of this disease. Nonetheless it is interesting that a similar abnormal recruitment of motor unit potentials was demonstrated in cases of benign myalgic encephalomyelitis in Durban and Coventry (both unpublished).

This is not to say that we did not recognize psychiatric factors in the symptomatology of this disease. Indeed we think it probable that amongst those studied in the outbreak were some whose manifestations were probably hysterical but are in no doubt that the large majority were affected by an organic illness.—We are, etc.,

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SIR—I have delayed writing about the articles by Dr. C. P. McEvedy and Dr. A. W. Beard (3 January, pp. 7 and 11) until I had an opportunity to consult the colleagues who were concerned with the care of the nurses affected by the outbreak of illness at the Middlesex Hospital in 1962.

The account of this outbreak given by Drs. McEvedy and Beard is substantially inaccurate in a number of ways. There was unmistakable objective evidence of involvement of the central nervous system in many of the cases. If one sets aside paresis, there were extensor plantar responses in five patients, difficulty in swallowing with paresis of the palate in two cases, and ocular paresis in one. Retention of urine requiring either the use of parasympathomimetic drugs or catheterization occurred in five patients. One of the girls (Case 2) had severe though short-lived respiratory paralysis and was nursed for some hours in a Drinker's respirator. This patient still had an extensor plantar response one year after the end of the outbreak. These and the other clinical

and epidemiological findings are recorded in detail in an article which appeared in the *Lancet* in 1954.¹ It is clear that Drs. McEvedy and Beard have not had access to all the information in their review of the evidence.

In their second article Drs. McEvedy and Beard put forward the hypothesis that most of the outbreaks which have been given the name benign myalgic encephalomyelitis were due to mass hysteria. I pointed out in an article² in 1959 that the question of hysteria must enter into the differential diagnosis "in an illness in which there has been a selectivity for young women, no mortality and few positive laboratory findings." For anyone who is interested, the arguments for and against are set out at considerable length in that article. On balance it was felt it was unlikely that mass hysteria was a major factor in the syndrome for the following reasons: (1) In the minority who had undoubted objective physical signs the clinical picture was similar to that which was seen in those who had no objective physical signs. (2) Apprehension about an outbreak of a neurological disease such as poliomyelitis was not a constant factor. For example, at the Royal Free Hospital the apprehension was not about poliomyelitis (as it had been at Los Angeles) but about infectious mononucleosis. In spite of this the course of the disease and type of neurological involvement was similar to those found in Los Angeles. (3) The mental symptoms which are a constant feature of the outbreaks were not typical of hysteria. (4) Muscle pain and tenderness were a striking and distressing feature of most of the outbreaks.

Many years ago Von Economo stressed the ease with which mental symptoms of encephalitis may be confused with those of psychoneurosis. I believe that Drs. McEvedy and Beard would have made a contribution to knowledge if they had undertaken to follow up samples of the Royal Free and Middlesex patients and re-examined them. As it is, apart from noting the fact that one of the Middlesex nurses died subsequently of Addison's disease, which may well be irrelevant, they report no new information about this epidemic.—I am, etc.,

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SIR,—One of the commonest epidemic diseases seen by family doctors is winter vomiting. Usually only of one day duration, it is so benign that less notice is taken of it than the common cold. Sometimes when it affects schools the medical officer of health is informed, as food poisoning may be suspected. Although no virus has been isolated it is undoubtedly viral in origin. When I wrote an article on it¹ I found it much commoner in females, as did Gray² and Bradley.³ In other reported epidemics meningeal symptoms have been present.⁴ The same virus may produce vertigo and collapse, as reported by Haworth *et al.*⁵ and Pollock and Clayton.⁶

When it affects boarding-schools it is put down to "epidemic hysteria," because no physical cause can be found, and the incu-

bation period is so short (sometimes less than one day, according to my research) that all the pupils are affected on the same day. But when you follow an epidemic in the general population, as I have, you can definitely rule out hysteria. Without knowing what has happened at the school, parents have reported children suddenly vomiting in the middle of the night. The vomiting is so sudden and unexpected that I am convinced the virus affects the central nervous system (hence the variations of vertigo, collapse, or meningeal symptoms). Just as the influenza virus may become more virulent, it is reasonable to postulate that this virus can become more virulent, and that is what I believe happened in the Royal Free epidemic. However, I think hysterical symptoms may have overlaid the basic illness.—I am etc.,

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Rickettsial Antibodies in Multiple Sclerosis

SIR,—The well-reasoned paper by Professor E. J. Field and Miss Mavis Chambers (3 January, p.30) cites comments on my alleged "negative findings" by Le Gac and Arquie.¹ I must point out that I have not personally worked on this problem. The relevant work in my department was carried out by Dr. Constance A. C. Ross, was not limited to *R. burneti* but covered a range of rickettsiae, and included microagglutination tests by the method of Giroud, using rickettsial antigens kindly supplied by Dr. P. Giroud and Dr. M. Capponi of the Service des Rickettsioses, Institut Pasteur, Paris.

In a preliminary study complement fixation tests of sera from 40 patients with multiple sclerosis showed no evidence of infection with Q fever or murine typhus; tests for rickettsialpox were also negative, except for two cases with low titres of doubtful significance.² A further series of cases with matched controls was then investigated by Dr. Ross and her colleagues.³ Complement fixation tests again showed no evidence of Q fever in 85 cases and 85 controls except for low antibody titres in one case and two controls. After visiting the Institut Pasteur, Paris, to study the technique, Dr. Ross performed microagglutination tests with the following antigens: *R. burneti*, *R. conori*, *R. mooseri*, and Q-18 (a "neorickettsia"). These tests showed no significant difference between 30 cases and 30 matched controls.³

Thus two independent controlled studies in different areas of Britain have failed to reveal evidence of specific association between multiple sclerosis and raised rickettsial antibodies detectable by the same technique and antigens used in the cases reported by Le Gac. I agree with the suggestion of Professor Field and Miss Chambers that previous residence in areas where rickettsiae

are endemic could explain the finding of rickettsial antibodies in the sera of multiple sclerosis patients studied by Le Gac. It should be noted that rickettsiae are endemic not only in tropical Africa but also in northern Africa and other areas bordering the Mediterranean, including France. The only rickettsia affecting man known to be endemic in Britain is *R. burneti*, and no indication that Q fever is related to multiple sclerosis has been shown by the British investigations involving microagglutination tests in altogether 79 and complement fixation tests in 125 multiple sclerosis cases.

I therefore agree with Professor Field and Miss Chambers and with my local colleagues that there is no acceptable evidence for a rickettsial aetiology of multiple sclerosis, and I see no grounds for diverting time and resources to clinical trial of Le Gac's treatment.—I am, etc.,

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Management of Infants with Cerebral Palsy

SIR,—Dr. B. A. Stoll (29 November, p. 558) asks about district assessment centres for handicapped children and their place in the management of infants with cerebral palsy.

The country's first assessment centre for handicapped children, the Newcomen Clinic at Guy's Hospital, has one of the infant treatment units, as mentioned by Dr. Stoll, in the same building. Both the clinic and the infant treatment unit are available for patients from district and regional levels. As there are few therapists suitably trained for this work in the country at this time, they would be most effectively used at regional level. By teaching the parents management and exercise techniques, one therapist can supervise daily therapy for up to 50 infants, on a basis of monthly appointments. As such infants make some progress, parents do see an apparent response to their efforts. Distance from the centre ceases to be a disadvantage if appointments are intelligently geared to public transport facilities, and the unit provides a kitchen in which to meet the requirements of infant and parent.

As yet, there is little evidence of a specific benefit solely attributable to any of the exercise techniques currently available. It is, therefore, difficult to justify the time and expense involved from the standpoint of therapeutic effect. On the other hand, it would be impossible to provide overall guidance and stimulation without some basic exercise system. The physiologically most acceptable means of learning movement control and gaining sensory experience of all kinds is by voluntary movement. Techniques for promoting voluntary movements exist and, as one might expect, they do not require the effort demanded in passive movement therapy. The use of exercises for infants and young children is not new.

Rejection has not occurred during treat-

ment, perhaps because the parents are doing something to help, albeit in a limited way. They have also had the benefit of early, comprehensive assessment and all the general support inherent in both assessment and treatment sessions.

Let us first complete the organization of regional assessment centres with some provision for infant treatment in them. It is at this level that sufficient expertise may be acquired, research carried out, and teaching facilities provided. The regional centre's experience of local needs will then ensure that district centres are set up in the right place, at the right time, and with properly trained staff.—I am, etc.,

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Coronary Care

SIR,—We would like to comment on a few points made by Dr. J. Dominian and Miss M. Dobson in their recent paper on psychological attitudes of patients admitted to a coronary care unit (27 December, p. 795). In general we are in agreement with their findings and conclusions.

We have found good factual communication with the patient about his illness, combined with emphasis on the feasibility and need for rapid rehabilitation and return to work, to be most important in maintaining confidence and in preventing anxiety. Within a day or two of admission, when the initial high-risk period has passed, each patient is given a copy of a brochure entitled *Explaining Coronary Disease and Coronary Care* which has been prepared by us for this purpose. Our patients have invariably responded favourably to the detailed information on coronary heart disease causation and treatment contained in this brochure. We seldom require to provide sedation or tranquillizers, because we find that keeping the patient accurately informed of his illness and progress is a more successful and rational way of preventing anxiety. We share the experience of the authors that the transfer of the patients from the unit to other wards may give rise to feelings of insecurity and anxiety. We have avoided this problem by having four of our six monitored beds in the open wards of the cardiac department and by returning patients to cardiac beds contiguous to the coronary care beds after they have been demonitoring. It has been our successful policy to provide "open-plan" coronary care in a cardiac department of twenty beds where resuscitation facilities are immediately available to all occupants. Our patients find this arrangement to be reassuring, and, like the authors, we find that only a small minority of our private patients prefer to be moved subsequently to a private room. Apart from the psychological benefits of having coronary care beds and beds for convalescent patients in the same wards, we have been successful in resuscitating the isolated late primary ventricular fibrillations, because patients remain in the coronary care area during their convalescence.

In discussing the long-term psychological attitudes of these patients we would like to make a few points about the length of stay

in hospital. Lown and Sidel¹ have advocated the early discharge of patients after myocardial infarction, and they consider it feasible to discharge the uncomplicated case in less than two weeks after admission. Early discharge may mean failure to resuscitate the occasional patient with delayed primary ventricular fibrillation, but we would put forward two other reasons why patients with recent myocardial infarction should be kept in hospital for a minimum period of three weeks.

Firstly, in the uncomplicated myocardial infarction patients should be fully ambulated by the end of the third week. From the psychological point of view it is of immense benefit that this active physical rehabilitation should take place under the direct supervision of the cardiologist and his medical, nursing, and physiotherapy staff. In particular we have found organized ward physiotherapy sessions to be valuable in the psychological as well as the physical rehabilitation of the patients. Secondly, no patient should be discharged without a full screening of all possible risk factors if an effective programme of secondary prevention is to be instituted. Screening includes studies of blood lipids, glucose tolerance, blood pressure status, smoking and eating habits, weight and exercise experience, family history, personality assessment, and other associated illnesses. An analysis of risk factors requires time and careful organization, and is best left until the patient is recovered from the acute illness. Measurement of serum cholesterol is best delayed for three weeks or more, because of a temporary depressing effect of the heart attack on lipid levels.² The assessment of the patients' blood-pressure status and glucose tolerance is also best done after two to three weeks.

The proposition that the elimination of risk factors will reduce subsequent morbidity and mortality in patients with coronary heart disease may well be a valid one and will appeal to the patient's sense of logic. In our experience this approach has been the single most successful factor in the full psychological recovery of our patients.—We are, etc.,

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Looking at the Liver

SIR,—Dr. K. P. Poulou and colleagues (6 December, p. 585) make an important point in emphasizing that major tumour surgery should be preceded by an evaluation of the patient's hepatic status. To be helpful to the patient this should be achieved without the need for a diagnostic laparotomy; the mortality for this procedure ranges between 12 and 19%.¹ Biochemical tests are notoriously unreliable. Liver histology would help if one could be certain of biopsying a representative area. Blind needle biopsy has been shown to detect metastases in less than 50% of cases with known hepatic deposits.² Needle biopsy with the help of a liver scan may increase the accuracy of the