Cytomegalovirus Infection with Lymphadenopathy

SIR,—Infection with cytomegalovirus is not uncommon in neonates and young children. Hitherto, the infection in adults has been reported principally as an infectious mononucleosis-like disease with a negative heterophil agglutination test, though exudative pharyngitis and lymph node involvement are usually absent.¹⁻³

We recently saw a 31-year-old Chinese woman in whom the clinical features of disease were compatible with infectious mononucleosis. On admission the patient had a temperature of 100° F. (38° C.); there was faint jaundice and a maculopapular rash over the trunk, back, limbs, and face. Bilateral tender cervical, axillary, and inguinal lymph nodes were present. The liver was enlarged 2 cm. and very tender. Investigations at this stage revealed: Hb 12.9 g./100 ml., W.B.C. 11,000/cu.mm.; atypical monocytes 28%; E.S.R. 21 mm./hour, Paul-Bunnell negative, serum bilirubin 2 mg./100 ml., serum alkaline phosphatase 26 K.A. units, S.G.O.T. 100 S.F. units.

Within the next few days the patient's condition worsened. Her temperature rose to 104° F. (40° C.), skin lesions became more severe, the spleen became palpable, but the jaundice disappeared. The haemoglobin fell to 8.6 g./100 ml., neutrophils showed a marked shift to the left, eosinophils 30%, atypical monocytes 10%, reticulocytes 6%, E.S.R. 122 mm./hour, serial Paul-Bunnell negative. As the picture was no longer typical of infectious mononucleosis, an autoimmune or allergic process was considered. Investigations along this line proved negative. Six weeks after admission the patient was clinically asymptomatic with no treatment. The titre of complement-fixing antibody for cytomegalovirus was 1/32 on admission rising to 1/128 four weeks later.

This patient is of particular interest in that hepatitis, haemolytic anaemia, and exanthema together with generalized lymphadenopathy were observed. The development of antibodies to cytomegalovirus is good but not incontrovertible evidence that the virus caused her disease.

We are indebted to the laboratory, Edgware General Hospital, for the result of diagnostic tests.

—We are, etc.,

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Acid-base Changes in Haemodialysis

SIR,—Dr. A. G. Morgan and his colleagues (28 March, p. 779) pose once again the problem of respiratory alkalosis consistently observed at the end of a haemodialysis.

This finding is generally considered to

depend on the persistence of acidosis of the cerebrospinal fluid caused by the slow passage of the bicarbonate ions through the blood-brain barrier. Yet this hypothesis does not explain the changes in acid-base equilibrium in all cases. Some of Dr. Morgan's patients began the dialysis with a normal or high blood pH. Under these conditions it is difficult to accept the existence of a C.S.F. acidosis, because the interdialytic period would be sufficient to establish an equilibrium between blood and C.S.F.

This led us to look for an alternative explanation of hypocapnic alkalosis caused by haemodialysis. The diffusion of CO₂ through the dialysing membranes is one possibility. In our experience, a large difference in arterial-venous PCO₂ is present during haemodialysis. This phenomenon is particularly evident when using the Kolff dialyser, probably owing to the more vigorous swirl of the dialysate fluid. On the other hand, the behaviour of PCO₂ in the dialysate fluid proves this interpretation directly.

In fact, at the dialyser input, the PCO₂ of the dialysate fluid resembles that of the atmosphere (0.2 mm. Hg), whereas at the output it is much higher, particularly in the Kolff dialyser (figure).

On this basis we think the diffusion of PCO₂ has an important function in determining the changes of acid-based equilibrium during haemodialysis and at the end of it.—We are, etc.,

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Cardiac Catheterization

SIR,—The reply given to Mr. S. A. Copeland and Mr. N. H. Brooks (30 May, p. 542) by Dr. D. E. Jewitt and others to criticism of their work (28 March, p. 795) on ethical grounds is far from satisfactory. A series of 15 patients with acute cardiac infarction were submitted to simultaneous catheterization of the aorta and the pulmonary artery, not for their immediate benefit but in order to measure the haemodynamic effects of pentazocine.

It was found right from the beginning of the experiment that the effect of the drug was to cause a considerable rise of the pressures in both the aorta and the pulmonary artery, neither of which could be anything

but harmful, especially in the five patients who initially had pulmonary oedema, which is itself associated with high pulmonary artery pressure. The authors themselves state that raising the aortic pressure "provides an after-load stress to the heart." But having found these harmful consequences in three patients why persist and repeat the experiment on another twelve?

I have objected¹ to published accounts from Hammersmith, the National Heart, Edinburgh, and Park Royal Hospitals of series of experiments done in intensive coronary care units.²⁻⁷ These experiments involved such activities as leaving cardiac catheters in situ for nine days in patients with acute cardiac infarction, including in patients aged 87.

I do not consider that combined right and left sided cardiac catheterization, with their combined risks which are not inconsiderable, can be regarded as a routine investigation on any patient with acute coronary thrombosis. Such investigations are purely experimental and consent to them can be regarded as valid only if the patients have been informed that what is to be done to them is not routine procedure and not in their own immediate interests, and they are fully informed of hazards involved in the procedure. No doubt I will be informed that consent was obtained. But all experimental physicians should study most carefully the warnings given by Sir Roger Ormrod, who is both a High Court judge and a doctor, and his views as to what constitutes valid consent.⁸

Such experimental work on patients with acute cardiac infarcts raises not only grave ethical and possibly legal problems but also questions the value of intensive coronary care units. Such units, extremely profligate of medical and nursing manpower and of finance, have not proved their clinical value. I have often talked to many doctors who have worked in such units, and all have informed me that they are not convinced that they have resulted in any significant lowering of mortality. The ultimate outcome of such patients is usually determined in the few hours before actual admission to such units. Possibly a case can be made out for routine monitoring of blood pressure and E.C.G. in such patients, but the routine measuring of various pressures in heart chambers and great vessels cannot be justified on clinical grounds.

I am willing to accept that the experiments of Dr. Jewitt and his colleagues were approved by the ethical committee of the Royal Postgraduate Hospital, but this merely indicates that their understanding of the ethical standards of our profession differs from mine. Is it not time that the M.R.C. and other grant-giving bodies more carefully investigated the ethics of experiments done by doctors receiving grants from them?—I am, etc.,

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