

recent reports.<sup>2</sup> It is recommended that potentially lethal infection with *Str. pneumoniae* or *H. influenzae* be seriously considered in any asplenic patient with pyrexia which is not readily explained and, if leucocytosis is found, that treatment with ampicillin be commenced immediately after obtaining blood for culture. Asplenic patients presenting with meningitis or Waterhouse-Friderichsen syndrome should be treated with a regimen including an antibiotic effective against both *Str. pneumoniae* and *H. influenzae*—for example, intravenous ampicillin—until the causal organism has been clearly identified.—I am, etc.,

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- <sup>1</sup> Whitaker, A. N., *Medical Journal of Australia*, 1969, 1, 1213.
- <sup>2</sup> Ramsay, L. E., and Bouskill, K. C., *Journal of the Royal Naval Medical Service*, 1973, 59, 102.
- <sup>3</sup> *Annals of Internal Medicine*, 1972, 77, 143.
- <sup>4</sup> Smith, H., *Medical Annual*, 1973, 91, 278.
- <sup>5</sup> Horan, M., and Colebatch, J. H., *Archives of Disease in Childhood*, 1962, 37, 398.
- <sup>6</sup> Ravry, M., et al., *Annals of Internal Medicine*, 1972, 77, 11.

### Treatment of Genital Herpes

SIR,—Your leading article (1 June, p. 461) describes the local treatment of genital herpes by incorporating a light-sensitive compound into the virus and then irradiating the infected cells. It also underlines the difficulties of evaluating the results of any form of treatment, especially as one of the most important parameters is the subjective index of relief of pain and tenderness in the herpetic lesions. It might have pointed out more emphatically the need to restrict evaluation to the results obtained in cases of primary herpes simplex virus (H.S.V.) infection, as recurrent attacks are usually mild and short-lived.

In genital herpes local applications may be impracticable; the urethra is not uncommonly involved in men and a severe necrotizing cervicitis is present in at least one-third of women with primary vulval herpes. Furthermore, in a smaller percentage of patients there are severe constitutional symptoms, presumably arising from viraemia. A systemic remedy should therefore be our therapeutic goal. Both idoxuridine and cytarabine cause marrow suppression when given systemically and their use would be justified only for life-threatening H.S.V. encephalitis. For the past four years we have used co-trimoxazole in severe cases of primary genital herpes with such good results that we are undertaking a double-blind trial to provide a firm evaluation of this simple, safe, and inexpensive form of treatment.—We are, etc.,

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### Beta-blockade in the Presence of Renal Disease and Hypertension

SIR,—We were most interested in the results of investigations by Drs. F. D. Thompson and A. M. Joekes (8 June, p. 555). They support the widely held impression that beta-blockers do not adversely affect renal

function in most hypertensive patients. The results do not, however, provide evidence to refute our suggestion that beta-blockers may cause deterioration in renal function in some patients with chronic renal failure (27 April, p. 193). It is significant that three consecutive patients in whom there was an association between deterioration in renal function and the introduction of beta-blockers were admitted to a specialist renal unit in the space of six months. Having excluded other likely causes of deterioration in renal function we could only conclude that beta-blockade might be responsible. Recent evidence<sup>1</sup> that the incidence of atherosclerotic heart disease in patients with renal failure is high supports our view that there may be some patients with chronic renal failure who are particularly susceptible to the negative inotropic and chronotropic effects of beta-blockade on the heart.

We await with interest the full results of the work of Drs. Thompson and Joekes and in particular a detailed analysis of the effects of beta-blockade on renal function in those patients whose creatinine clearance is less than 30 ml/min.—We are, etc.,

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- <sup>1</sup> Lindner, A., et al., *New England Journal of Medicine*, 1974, 290, 697.

### Puerperal Rubella Vaccination and Anti-D Immunoglobulin

SIR,—We were interested to read the letter from Mr. B. Alderman and Mr. D. W. Charters (29 June, p. 724).

It has been the practice in Bradford to perform rubella haemagglutination inhibition (H.I.) tests routinely in pregnancy at the first antenatal attendance and to offer rubella immunization with Almevax vaccine in the puerperium to women with titres of 1/16 or less. Some of these patients to whom rubella immunization was offered had been given anti-D immunoglobulin, and in view of suggestions that this might prevent a successful vaccination we have followed up as many patients as possible to see if they had a serological response to the rubella vaccine.

The number of results so far available is very small. Of four women whose antenatal H.I. titres were less than 1/8 the post-vaccination titres were 1/128 in two cases, 1/64 in one, and 1/32 in one. In three of the four cases two additional H.I. tests were done after delivery, before rubella immunization was performed—namely, before and after the administration of anti-D immunoglobulin; all these titres also were less than 1/8. The postvaccination specimens were collected 51–60 days after immunization. Seven women whose prevaccination H.I. titres were 1/8 or 1/16 and who also received anti-D immunoglobulin showed less response and had postvaccination titres which were only 2- or 4-fold higher or remained unchanged.

Though we do not have enough data to permit firm conclusions, it does seem that at least those patients who have no detectable rubella antibodies are likely to develop a satisfactory serological response following rubella vaccination in spite of the prior

administration of anti-D immunoglobulin. An extended investigation to confirm our preliminary findings would be desirable, and we should be interested to know the experience of other workers in this field.

We wish to thank the consultant obstetricians in Bradford for their co-operation in this investigation and Dr. R. J. Rand and Dr. Brij B. Khurana for their assistance.

—We are, etc.,

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### Healing of Leg Ulcers

SIR,—My attention has been drawn to an erroneous reference in my paper "Vertical Leg Drainage of Oedema in Treatment of Leg Ulcers" (15 June, p. 581). The correct reference is Myers, H. B., and Cherry, G. (1971), *American Surgeon*, 37, 167.

It is possible that my paper conveyed the impression that I considered my healing rates equalled those quoted by Myers and Cherry. This was not my intention; their healing rates were much faster, but their ulcers were much bigger than most of mine, and in the similarly large ulcers in my series the rate was much above my average.

It is worth emphasizing that my method is cheaper, especially in the absence of loss of work time for the patient.—I am, etc.,

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### HI Antibodies to Influenza A/Finland/1/74

SIR,—During the epidemic season of 1973–4 influenza outbreaks in Finland were caused mainly by B "intermediate" strains, but small outbreaks and scattered cases due to influenza A also occurred throughout the country. From a follow-up study the rate of influenza A infections among adults was clearly below 10%. The 28 influenza A isolates from 21 patients were found to be antigenically closer to A/Port Chalmers/1/73 than to the previous variants of Hong Kong virus. The strains studied in the World Influenza Centre differed even from the Port Chalmers strain by showing very low, if any, HI titres with anti-A/Hong Kong/1/68 serum. Similar strains were isolated in the Democratic Republic of Germany, Federal Republic of Germany,<sup>1</sup> and Norway.<sup>2</sup>

During the recent influenza epidemics an HI titre of  $\geq 40$  has appeared to confer protection against infection.<sup>3</sup> In pre-epidemic sera collected in Finland in autumn 1973 from 40 conscripts and from 65 pregnant women the percentages of cases with an HI titre of  $\geq 48$  to A/Finland/1/74 were 30 and 27. The corresponding percentages of cases with HI antibodies to A/Hong Kong/8/68 were 65 and 75. In a series of 43 acute phase sera from A/Finland/74 patients the highest titre of the homologous antibodies was 24.

We have analysed the HI antibody responses of influenza A patients during the five H3N2 epidemics occurring in Finland. All patients were conscripts of 18–21 years. The acute sera had been taken not later than in the fourth day from the onset of illness,