with the minimum of sequelae.—We are, etc.,

IAN CRAFT

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1 Wright, C. S. W., Campbell, S., and Beatley, J., Lancet, 1972, 1, 1278.
7 Craft, I., Lancet, 1974, 1, 1344.

Meningococcal Disease

Sir,—In their interesting account of an outbreak of meningococcal disease in Devon (16 March, p. 50) Dr. D. M. Easton and his colleagues raise several points worthy of discussion.

They used a combination of penicillin, sulphonamides, and chloramphenicol in the initial treatment of most cases and suggest that if the low cerebrospinal fluid cell count in meningococcal meningitis is "indicative of minimal inflammatory response in the meninges penetration of penicillin into the C.S.F. may be poor, so that the inclusion of chloramphenicol which passes well into the C.S.F. might be helpful"

The inclusion of sulphonamides in the treatment of meningococcal meningitis is of less certain help than formerly owing to the presence of resistant meningococci. Of the strains isolated in Scotland and forwarded to this laboratory for testing in 1973, 15% were fully resistant to sulphonamides. What is also of importance when considering prophylaxis is that in addition 51.5% of strains were partially resistant—that is, would not be eradicated from the nasopharynx by sulphonamide therapy. Hence if prophylaxis is to be used sulphonamides cannot be recommended.

This raises the question as to who should be protected and how. Firstly, I cannot agree with Dr. Easton and his colleagues that "family contacts should be screened and should all be given an adequate course of prophylactic treatment" if they imply that prophylaxis should be dependent upon screening. Screening will not pick up all carriers and in any case I would agree with Wenzel et al. 1 that the acquisition rate of meningococci that is important in terms of the dynamics of meningococcal infection, not the carrier rate. In other words, meningococcal disease is, like poliomyelitis, a "failure of immunity" and it is interesting to note that in both these diseases asymptomatic infection leads to the production of antibodies. Hence prophylaxis must be used early if at all and must cover the case contacts, not so much to eradicate carriage but to try to prevent acquisition leading to disease. There is no doubt that during meningococcal epidemics, as has been shown in Africa 2 and in service camps in the U.S.A. 3 but the problem is how this should be carried out in the face of sulphonamide resistance and the knowledge that penicillin will not eradicate meningococci from the nasopharynx of carriers. 4 One approach might be to give penicillin in the hope of preventing disease (and maybe reducing the chance of introducing the disease to a low level to reduce the likelihood of acquisition by the non-carrier). However, there is evidence 5 that penicillin not only will not eradicate carriage but will not prevent the onset of meningitis. The other approach is to give eradication treatment—for instance, using minocycline and rifampicin 6 or rifampicin alone. 7 Until reading the account of Foster et al. 8 I might have conceded that in a family group of meningitis, especially if penicillin by injection, should be adequate, but I now feel that, as in an institutional outbreak where long-term carriage following inadequate prophylaxis could result in the breaking of the chain of infection, if prophylaxis had been ended, eradication treatment should be considered. It would be interesting to hear the views of others on this important topic.

At one time there was a suggestion that family contacts should receive prophylaxis would not have been accepted because outbreaks in family groups were rarely reported, but in recent years outbreaks involving more than one member of a family have been reported 9 and the experience in the Devon outbreak emphasizes that this can happen. Hence the urgency for reappraisal of the problem of prophylaxis is underlined.

Finally, Dr. Easton and his colleagues noted that group B meningococci predominated in their series. Were other groups isolated or were some strains either untypable or untypable—? I am, etc.,

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Misleading Discs

Sir,—The antibiotic discs described by Mr. D. F. J. Brown and Dr. J. B. Selkon (23 March, p. 573) are not the only ones to mislead.

A strain of the Haemophilus genus requires one of both two growth-promoting substances for normal growth on culture. These are haematin, designated the "X-factor", and coenzyme 1, designated "V-factor". Coloured paper discs marked X, XV, and V, impregnated with standard quantities of these substances, are commonly used as a simple screening method for preliminary recognition of haemophilus species. Recently a culture of H. influenzae appeared as only a fine ring of growth around an XV disc, but outside a zone 14 mm in diameter without any growth. It was almost invisible without a hand lens. Another haemophilus grew as a ring around a zone without growth surrounding both the X and V discs. Experimental cultures of pneumococci and streptococci also showed zones of inhibition around the X and XV discs. These discs were in date and stored dry at 4°C. A similar phenomenon was observed some years ago. The paper dye was not to blame since control blanks of the three colours were inactive, and it was assumed that this the X-factor (haematin) solution used must have been inactive. The makers were informed and corrected the matter, but the difficulty now seems to have recurred.—I am, etc.,

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Clinical Diagnosis of Reye's Syndrome

Sir,—I entirely agree with the views expressed in the last paragraph of Dr. Ellen S. Kang's letter (16 March, p. 518). Having a strong and abiding interest in Reye's syndrome 1 may I develop her idea a little farther? I suggest that what she terms "toxic encephalopathy with fatty visceral changes due to a specific toxic agent" should not be confused with "toxic encephalopathy" because it was the original intention of Reye et al. 2 to place the entity of "encephalopathy with fatty degeneration of

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gingiucocci was demonstrated bacteriologically. A petechial rash was noted in nine cases.

Two children died, a girl of 3 years with severe septicaemia and shock and a boy of 6 who had septicaemia and gross cerebral oedema associated with disseminated intravascular coagulopathy and cerebral haemorrhage. Two other children had brief convulsive episodes and a third who had prolonged convulsions went on to develop bilateral subdural effusions and had considerable residual brain damage. One child had definite arthritis of the right elbow joint and another a transient arthralgia affecting the left knee. There was no history of prior contact with meningococcal disease and their homes were scattered throughout the area served by this hospital.—I am, etc.,

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