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

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1 Wright, C. S. W., Campbell, S., and Beasley, J.-Lancet, 1972, 1, 1278.
8 Craft, I.-Lancet, 1974, 1, 397.

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 indicates only 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.

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

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

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Clinical Diagnosis of Rete'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 chronic and disabling interest in Rete's syndrome I may I develop her idea a little farther? I suggest that what she terms "toxic encephalopathy with fatty visceral changes due to a specific toxin" should constitute a separate entity because it was the original intention of Rete et al., to place the entity of "encephalopathy with fatty degeneration of

BRITISH MEDICAL JOURNAL 4 MAY 1974