Epidemic Keratoconjunctivitis

Sir.—We are interested to read the account by Dr. D. L. Barnard and others (21 April, p. 165) of the outbreak in Bristol of keratoconjunctivitis caused by adenovirus type 8. Similar outbreaks occurred in the Clyde valley in 1956, 1967, and 1971. Because of the importance of this disease as a cause of disability and occasional reduction in visual acuity, an attempt has been made to accumulate data in the Glasgow area during the interepidemic periods to determine whether the virus smouldered inconspicuously among industrial populations, among patients attending ophthalmic clinics or, whether, as in Japan, the reservoir of infection was in children in whom the disease was uncommon.

To try and solve this problem virological surveillance of a sample of patients attending ophthalmic clinics with conjunctivitis and keratoconjunctivitis has been continued in Glasgow since the 1967 epidemic subsided. However, from over 1,000 conjunctival scrapings examined, only five isolations of adenovirus type 8 were found in 1968—they were obtained from members of a typical industrial group—and no further isolations were made until the latest outbreak in 1971. Thus this continued six-year virus surveillance provides no clue as to the whereabouts of adenovirus type 8 during the interepidemic period but is compatible with the virus being periodically imported from other areas or countries—for example, by seamen, transport drivers, or other travellers.

During the 1971 outbreak, in contrast to the findings in Bristol, there was very little family spread in Glasgow. In a survey of 584 family contacts of the 200 patients involved secondary spread occurred in only 14 contacts. It is likely that this was due to the strong propaganda measures (for example, to use separate towels, etc.) which were instituted at the start of the outbreak. It may be added that, though it should have been detectable by our methods, we encountered no case of picornaviral acute haemorrhagic conjunctivitis.—We are, etc.,

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SERUM ALKALINE PHOSPHATASE AND RICKETS

Sir,—As Drs. W. T. Cooke and P. Asquith mention (10 February, p. 324; 5 May, p. 302) the relationship between the serum levels of alkaline phosphatase and vitamin D activity during the adolescent growth spurt is clearly relevant in determining whether the alkaline phosphatase "flare" at puberty is due to "biochemical rickets." Our data in 59 healthy Caucasian schoolboys, whose ages were evenly distributed between 12 and 17 years and who were sampled at the same time of year, are shown in the figure. We were unable to sample girls simultaneously.

There was no significant correlation between plasma alkaline phosphatase and 25-hydroxycholecalciferol (25-HCC) levels (r = -0.02, t = 0.161, P > 0.8). 25-HCC is very stable in human plasma, levels remaining constant when stored even in unseparated plasma at 4°C over periods of 11 days and being unaltered after separation by repeated freezing and thawing. We believe that plasma 25-HCC levels provide the most accurate available index of vitamin D nutritional status in man.

A significant inverse correlation between plasma 25-HCC and alkaline phosphatase is of course to be expected in a vitamin-D-deficient population with a high prevalence of rickets. Caucasian children are not in this category. It would also be not unreasonable to expect an increased rate of utilization of 25-HCC during the puberty growth spurt; this could be associated with fall in 25-HCC levels, within the normal range, occurring at the age of peak height velocity (13 years in boys) in an adequately nourished population; studies are in progress to provide evidence on this question.

Relationship between plasma alkaline phosphatase and 25-HCC levels in 59 healthy schoolboys aged 12-17 years sampled between 27 September and 6 October 1971.

Our present findings indicate by themselves, however, that the high phosphatase levels in this age group are not produced by 25-HCC deficiency and help to confirm, as we have previously stated (14 April, p. 113), that the alkaline phosphatase flare is a normal physiological event at puberty.—We are, etc.,

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Troubles with I.U.C.D.s

Sir,—Probably many of us who have fitted a number of intrauterine contraceptive devices have come across strange cases. In February 1968 I fitted a 24-year-old para-2 with a Lippes loop size C. Fitting was quite uncomplicated and simple. She returned to me in May not having had a period since the middle of April. The uterus was retroverted and though there were no signs of pregnancy she subsequently had a normal delivery. The I.U.C.D. did not appear during labour, and five weeks after delivery she was screened with an opaque sound in the uterus. This showed that the I.U.C.D. was lying separate from and anterior to the uterus. It was decided that the I.U.C.D. must be removed. The suction was carefully explained to the patient and she was told that she would never see her loop again. Six months later she appeared in the surgery and handed me the loop.

It would appear that this device had perforated the uterus, probably leaving the tails in the wall of the uterus, and it had subsequently tracked back and been discharged per vaginam.—I am, etc.,

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PREVENTION OF PULMONARY EMBOLISM

Sir,—Your leading article (7 April, p. 1) seeks a prophylactic method which can be


