suggests that the risk of every clinical manifestation of ischaemic heart disease is increased in hypertension. So it would seem to me that by including a beta-blocker in the treatment of hypertension the benefit to the patient will also include a lesser risk of infarction and not a greater risk.—I am, etc.,

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- ¹ Lambert, D. M. D., *Lancet*, 1972, 1, 793.
² Kannel, W. B., and Dawber, T. R., *British Journal of Hospital Medicine*, 1974, 11, 508.

Chickenpox from Herpes Zoster

SIR,—The report (8 June, p. 568) of two hospital outbreaks of chickenpox originating from herpes zoster was of great interest, particularly as importance is attached to the fact that the majority of staff affected were from overseas and so might have been expected not to have had the same levels of immunity as a corresponding group of native British adults.

Southmead Hospital experienced a similar outbreak in March 1974 in which, in contrast to the above reports, all affected staff members were native to the British Isles. The source was an immunosuppressed patient with Waldenström's macroglobulinaemia and facial herpes zoster. The latter diagnosis was confirmed by electron microscopical examination of the vesicular fluid, and the rash was treated topically with 25% idoxuridine in dimethyl sulphoxide. The staff members affected were the house officer, ward sister, a male night nurse, a nursing auxiliary, and a student nurse. All had been in close contact with the patient shortly after his admission, and had developed chickenpox 14-17 days later. The wife of the night nurse also developed chickenpox 14 days after her husband. No patient was involved. The source patient died of intercurrent *Escherichia coli* septicaemia two days before the first case was diagnosed. Measures introduced when the outbreak became apparent were effective in its control.

This outbreak shows that native British adults may be as much at risk of developing chickenpox as those who spent their childhood overseas. It also illustrates the high infectivity of herpes zoster in the immunosuppressed patient. We thank Dr. S. K. R. Clarke, Public Health Laboratory, Bristol, for the examination of the vesicle fluid by electron microscopy.—We are, etc.,

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Alkaline Phosphatase Levels in Epileptic Subjects

SIR,—In reply to Drs. P. Rødbro and C. Christiansen (22 June, p. 667) I should like to say that neither my methods nor my opinions have changed during the past three years. We have now carried out two separate surveys of the patients at the Chalfont Centre for Epilepsy^{1,2} and some of these results may help to clarify the apparent existing controversy.

In the first survey 48 out of 160 epileptic patients had raised plasma total alkaline phosphatase levels (defined as >10 and >12 King-Armstrong units (K.A.U.)/100 ml in females and males respectively). Of these 48, 18 (37.5%) had raised levels of bone isoenzyme (>5 K.A.U./100 ml). In the second survey 88 out of 310 epileptics had raised total alkaline phosphatase levels; 32 (36.4%) of the 88 had bone isoenzyme levels above 5 K.A.U./100 ml. Of the total number of 50 epileptics with raised bone alkaline phosphatase levels, 23 (46%) were aged between 16 and 20 years compared with only 16% of the total population. As can be seen from fig. 1, bone alkaline phosphatase levels are higher in epileptics aged 16-20 years than in

similarly aged normals, but the epileptic values decrease to normal levels between 21 and 25 years of age.

The histogram (fig. 2) shows that though 5-6% of adult epileptics (perhaps those most at risk of developing clinical osteomalacia) have raised plasma bone alkaline phosphatase levels, 14% of the population have bone alkaline phosphatase levels of less than 0.5 K.A.U./100 ml. Only one normal subject (1.6% of the population) had a similarly low level of bone alkaline phosphatase. The difference between the two groups was significant ($P < 0.005$). Mean plasma levels of bone alkaline phosphatase are thus no higher in adult epileptics than in adult normals.

Having described the syndrome of "anti-convulsant osteomalacia" in some detail,^{3,4} we have no reason now to deny its existence. What we are suggesting is that there may be a second anticonvulsant-drug-mediated bone effect possibly showing up in those epileptics with low bone alkaline phosphatase levels, many of whom also have a degree of hypocalcaemia.—I am, etc.,

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- ¹ Richens, A., and Rowe, D. J. F., *British Medical Journal*, 1970, 4, 73.
² Rowe, D. J. F., 1973, Ph.D. Thesis, University of London.
³ Dent, C. E., et al., *British Medical Journal*, 1970, 4, 69.
⁴ Stamp, T. C. B., et al., *British Medical Journal*, 1972, 4, 9.

Larrey and Débridement

SIR,—The review of Robert G. Richardson's recent book *Larrey: Surgeon to Napoleon's Imperial Guard* (24 August, p. 527) mentions Larrey's use of "débridement" in the

Fig. 1 Fall in level of plasma bone alkaline phosphatase with age in normal and epileptic subjects.

Fig. 2 Plasma bone and liver alkaline phosphatase isoenzyme levels in adult normal and epileptic subjects.