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.,

D. M. D. LAMBERT

Keighley, Yorks

- 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.,

D. J. F. ROWE

King's College Hospital Dental School,
 London S.E.5

- 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.

treatment of wounds. Dr. Richardson has, however, fallen into the error of regarding débridement as carried out by Larrey with the procedure as now practised. Thus he describes (p. 13) Larrey's practice in gunshot wounds as "... excising all dead and dying tissue and removing all foreign bodies from a wound—a procedure known as débridement." A reference to Larrey's *Clinique Chirurgicale Exercée Particulièrement dans les Camps et les Hôpitaux Militaires depuis 1792 jusqu'à 1829* reveals that this statement, inaccurately describes his practice. His primary treatment for wounds not requiring amputation was *pansement*, a technique of careful bandaging which kept the dressings in place until they spontaneously separated many days, and sometimes weeks, later, and an anticipation of Trueta's practice in the Spanish Civil War.

Larrey's primary use of the verb *débrider* is with the meaning of "to incise." Thus in volume 1 page 54 he writes: "*Si les plaies d'armes à feu sont accompagnées d'hémorragie, il faut nécessairement les débrider pour mettre les artères à découvert et en faire la ligature. . .*" Later he uses it as an extension of its literal sense of "to unbridle" (Fr. *bride* bridle) to refer to the division of actual or potential constricting bands. Thus for deeper wounds which extend to "*les parties charnues et aponévrotiques*" one must divide "*des brides propres à produire l'étranglement*" (p. 55). He goes on to say that wounds in regions where there are neither muscles nor aponeuroses "*ne doivent pas être débridées*" except to ligate arteries. It is true that he says (p. 57) that in deep wounds with fragmentation of bone one should remove separated fragments "*dont on ne peut espérer la soudure avec le reste de l'os*," but this is clearly because they are useless in the healing process.

Larrey did establish the importance of one aspect of débridement, the division of constricting aponeurotic tissues. The other, the removal of tissue debris serving as a culture medium for micro-organisms, had to await the surgical application of modern bacteriology and pathology.—I am, etc.,

DAVID PATEY

Hythe, Kent

Infections in Asplenic Adults

SIR,—Dr. L. E. Ramsey (27 July, p. 254) writes of the susceptibility of asplenic adults to infection. I must say that in some 40 years' experience I have not found, with one possible exception, an increased susceptibility to infection among asplenic adults. In 1935 I reported¹ a follow-up for an average of eight years of 100 adults in whom the spleen had been removed for rupture (the spleen otherwise being normal) and no increased rate of infectious disease could be found. It has later been suggested that splenectomized persons may be more liable to contract babesiosis of the brain. Of this I have no experience. My original studies in this matter had to do with the incidence of cancer in splenectomized persons, but I could not find any evidence of an association.²—I am, etc.,

E. ASK-UPMARK

Uppsala, Sweden

¹ Ask-Upmark, E., *Svenska Läkaresällskapets Förhandlingar*, 1935, 61, 197.

² Ask-Upmark, E., *Medical Masqueraders*, Stockholm, Scandinavian University Books, 1969.

Amitriptyline Poisoning in Childhood

SIR,—Goel and Shanks¹ have called attention to the increasing incidence in children of poisoning by tricyclic antidepressants. Meadow and Leeson² described the problems in managing poisoning from delayed-release tablets (Debendox). We report here a case of accidental poisoning due to a sustained-release form of amitriptyline.

A 5-year-old boy was admitted to hospital at 2.30 p.m. Three and a half hours before then his mother had noticed his gait to be reeling and had discovered that he had ingested four 50-mg tablets of orphenadrine hydrochloride (Disipal) and an unknown number of 50-mg capsules of Lentizol (amitriptyline), which contains a pellet of the drug in a form which delays liberation of the active constituent. On arrival he was disorientated, agitated, and incoherent. His pupils were fixed and dilated and his tendon jerks very brisk. He was subject to episodes of purposeless screaming and thrashing about of his body, which hindered assessment. His temperature was 37°C, blood pressure 120/80 mm Hg, and pulse rate 120/min (before therapy). He was sweating profusely. There was no cyanosis or other evidence of respiratory depression. Intramuscular paraldehyde 4 ml was given to facilitate gastric lavage. No identifiable capsules or pellets were retrieved. His bladder was catheterized in anticipation of urinary retention. We treated him as we do children poisoned with delayed-release tablets—that is, oral purging and colonic lavage. Magnesium sulphate 5 g was left in the stomach after gastric lavage and was subsequently given orally two-hourly. Enemata of soap and water and magnesium sulphate 65 g were given alternately half-hourly for six hours. Further sedation was required at 11 p.m. and thereafter his clinical condition slowly improved. Continuous cardiac monitoring showed only a persistent sinus tachycardia. Recognizable pellet fragments were recovered in the rectal effluent 14 hours after the start of treatment. The boy had completely recovered 36 hours after admission.

This is the first case we have seen of a child poisoned with this amitriptyline preparation. The manufacturers have had no other reports to date. This may be partly because it is not prescribed as often as shorter-acting formulations. In particular, we have not known of it being given for enuresis. The dose of amitriptyline associated with severe symptoms is said to be 20 mg/kg.¹ This child would have had to have taken only six capsules to receive that dose. His mother did not know the exact number taken but thought that the bottle had been "almost full." Our treatment was aggressive because of previous experience in this unit.² We thought it justified in this case because of the difficulty of treating amitriptyline poisoning once the drug has been absorbed, and it seemed imperative to try to remove as much unabsorbed material from the gut as rapidly as possible.—We are, etc.,

TIMOTHY CHAMBERS
A. D. KINDLEY

Leeds Children's Poisons Centre,
Seacroft Hospital,
Leeds

¹ Goel, K. M., and Shanks, R. A., *British Medical Journal*, 1974, 1, 261.

² Meadow, S. R., and Leeson, G. A., *Archives of Disease in Childhood*, 1974, 49, 310.

Lawn Mower Injuries

SIR,—May we add to the interesting letter from Dr. J. R. Hulme and Mr. A. R. Askew (13 July, p. 113) by relating the following case, which occurred last year. It illustrates a further complication resulting from such injuries.

The patient, a gardener aged 35 years, injured the four medial toes of his right foot

when a rotary mower he was using struck his boot. He was admitted to hospital where debridement of the wounds, appropriate wiring of his toes, and suturing of tissues were carried out. A first dose of tetanus toxoid and courses of cloxacillin and ampicillin were given. After three days he was discharged home and sutures were removed two weeks later when the wounds appeared healthy. At follow-up three weeks later the vascular condition of his second right toe was poor and its amputation was planned in 10 days' time. During this interval his general practitioner intended giving him a second dose of tetanus toxoid, but at the time of his appointment he felt unwell, his neck and jaw were stiff, and he did not attend the surgery. Two days later he was much worse and he then visited his doctor, who diagnosed tetanus and arranged his immediate readmission to hospital. The second dose of tetanus toxoid was not given.

In hospital the purulent and gangrenous toe was amputated and sheep antitetanus serum and penicillin in high dosage were given. Next day severe spasms of tetanus started which rapidly increased in frequency and severity; he was transferred to the intensive therapy unit where tracheostomy was performed, curarization undertaken, and full supportive therapy initiated. He was kept paralysed for 3½ weeks. *Clostridium tetani* was not isolated from wound swabs taken at the time of amputation, but was recovered from a section of the gangrenous toe placed in a previously boiled Robertson's meat broth. He was discharged from hospital six weeks after admission, at which time the second tetanus toxoid dose was given. At no time previously had he been actively immunized against tetanus; he had received one dose of A.T.S. in 1962.

Dr. A. T. Willis made the comment that this case illustrates that a first dose of toxoid given at the time of wounding is not protective.

We thank Dr. P. A. Thorn, consultant physician, for clinical details and Dr. A. T. Willis, director of Luton Public Health Laboratory, for confirming the identity of the organism.

—We are, etc.,

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IAN A. HARPER

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New Cross Hospital,
Wolverhampton

Alliterative Anxiety about Arsenic

SIR,—Pleased at the prospect of palates partaking poison-free porcine pâté (24 August, p. 487) the cosmopolitan clientele of your columns craves clarification of the concentration of cacodylic acid in kosher cuisine.—I am, etc.,

S. K. GOOLAMALI

Newcastle upon Tyne

Pontomedullary Listeriosis in Renal Allograft Recipient

SIR,—We were most interested to read the report by Dr. J. F. Mahony and others (29 June, p. 705) of a case of *Listeria monocytogenes* septicaemia with non-meningitic pontobulbar involvement after renal transplantation. We have seen a similar case.¹ In their case the initial presumptive diagnosis was of fungal meningitis, in ours that of herpes simplex encephalitis—both not un-