BCG immunisation

SIR,—The Department of Health, in circular EL(89)P/93, asked health authorities to postpone their regular BCG programme for schoolchildren this autumn because the British supplier of the vaccine is in the throes of moving to a new site. I wonder if others share my view that this request is unacceptable.

As district vaccination coordinator I hear much stout talk "from on high" about targets and accountability, and I feel that the department must now be seen to meet the standards it lays down for others. No target date for resumption of supply has been offered—is the problem for a week, a month, a year? Or is the hope that the programme will quietly wither away? The department should be held to account for this avoidable disruption to the immunisation programme. I also do not understand why, in a health service increasingly exposed to market forces, the market cannot be allowed to provide a solution. There is more than one reputable manufacturer of BCG vaccine.

We are already threatened with a resurgence of tuberculosis thanks to HIV, increasing poverty, and the possibility of a wave of refugees from Hong Kong and Indo-China. The last thing we need is departmental own goals.

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Antenatal prophylaxis with anti-D immunoglobulin

SIR,—Dr J G Thornton and colleagues[1] confirm that giving anti-D immunoglobulin antenatally at 28 and 34 weeks can contribute to decreasing the number of new sensitisations. The question remains, however, whether a programme of routine antenatal prophylaxis should be introduced generally.

Firstly, this study and the original trial[2] seem to compare routine antenatal prophylaxis with no antenatal prophylaxis. In many parts of the country the current recommendation is to use anti-D immunoglobulin when appropriate during pregnancy—for example, after amniocentesis, antepartum haemorrhage, external cephalic version, etc. Although the conclusion that routine antenatal prophylaxis is effective is valid, the translation of such findings into policy is difficult to justify when current practice differs from the management of their comparison group.

Secondly, about one third of pregnant women do not require protection as they will have Rh negative babies. At delivery these women will have had an unnecessary intervention with a blood product. Furthermore, it is sometimes difficult to distinguish between passively administered antibodies and those resulting from sensitisation. The problem increases in proportion to the number of women given antibodies during pregnancy and inflates the number of pregnancies labelled as high risk. This leads to additional monitoring, which has clinical and financial sequelae.

Thirdly, birth weight and mortality are not long term criteria for assessing the effects of anti-D immunoglobulin. Are there any long term haematological sequelae for either mother or baby 20-30 years later?

Dr Thornton and colleagues do not mention how many babies were affected by and required specific management for Rh haemolytic disease. Such data would help to justify a threefold increase in usage of anti-D immunoglobulin. Further research needs to be conducted on those few women who develop antibodies in their first pregnancy. If these women could be identified prospectively a "high risk only" policy for giving anti-D immunoglobulin antenatally could be developed, negating the need to give it indiscriminately. Even when anti-D immunoglobulin is produced synthetically we need to administer it to as few women as necessary.

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1 Thornton JG, Page C, Foote G, Arthur GR, Tovey LAD, Scott JS. Efficacy and long term effects of antenatal prophylaxis with anti-D immunoglobulin. *Br Med J* 1989;**298**:1671-3. (24 June.)
2 Tovey LAD, Townley A, Stevenson BJ, Taverner J. The Yorkshire antenatal anti-D immunoglobulin trial in primigravidae. *Lancet* 1983;ii:244-6.

Treatment of post-herpetic neuralgia

SIR,—The otherwise excellent leader by Dr Jacqueline Jolleys on the treatment of shingles[1] fails to examine adequately the use of oral steroids in preventing post-herpetic neuralgia. Eaglstein *et al*[2] showed that post-herpetic neuralgia was reduced from 73% to 30% after oral steroids were given. Keczkes and Basheer used oral prednisolone in 20 patients: only three developed post-herpetic neuralgia,[3] but this study was uncontrolled. Elliott also showed a benefit from high dose oral prednisolone.[4] The risk of generalised herpes after oral steroids have been used has been overstated. Merselis *et al* described 17 cases of disseminated herpes zoster in a total of 175 patients who were admitted to hospital with zoster.[5] Of the 17 with dissemination, 11 had serious underlying disorders (mainly haematological). Only two of the patients with no other serious disorder had received corticosteroids (adenocorticotrophic hormone). Four patients in the series died, including these two, neither of whom had any other precipitating factors.

Post-herpetic neuralgia, especially in elderly patients, can be a catastrophic condition. No other treatment has been shown to be other than marginally effective. In otherwise healthy elderly patients a three week course of tapered high dose prednisolone should be strongly considered.

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1 Jolleys J. Treatment of shingles and post-herpetic neuralgia. *Br Med J* 1989;**298**:1537-8. (10 June.)
2 Eaglstein W, Katz R, Brown JA. The effects of early corticosteroid therapy on the skin eruption and pain of herpes zoster. *JAMA* 1970;**211**:1681-3.
3 Keczkes K, Basheer AM. Do corticosteroids prevent post-herpetic neuralgia? *Br J Dermatol* 1980;**10**:551-5.
4 Elliott FA. Treatment of herpes zoster with high doses of prednisone. *Lancet* 1964;ii:610.
5 Merselis JG, Kaye D, Hook EW. Disseminated herpes zoster: a report of 17 cases. *Arch Intern Med* 1964;**113**:679-86.

Gastro-oesophageal disorders in adults with severe mental impairment

SIR,—We read with interest the paper by Drs Joseph Kuruvilla and Peter N Trewby concerning the aetiology and epidemiology of recurrent vomiting in severely mentally handicapped patients who reside in mental hospitals.[1] We would like, however, to report a few observations, although we welcome the interest that the authors have shown in this group of patients.

The study is small and (presumably for ethical considerations) uncontrolled. The authors recorded the results of investigations undertaken in only 56% of their sample. Resident patients in mental hospitals are unrepresentative of mentally handicapped patients in general; bias undoubtedly occurred during the referral process.

Of those patients who were not investigated (17), the authors state that "all responded to treatment with H_2 blockers, metoclopramide, or antacids, which suggested that upper gastrointestinal disease was the cause of their vomiting." We believe this assumption to be unsound for two reasons: firstly, we are not informed of the duration of follow up (vomiting as a symptom of psychiatric disorders can be cyclical); and, secondiy, giving extra medication and showing concern for the symptom, etc, increase the attention given to the patient: attention seeking behaviours are common among mentally handicapped patients who reside in mental hospitals.

We were interested that the authors were unable to account for the vomiting in six of the 22 patients whom they investigated. We suggest that they may be making exaggerated claims in attributing causality to discovered oesophageal disease in patients when only an association can be entertained. It could, for example, be argued that oesophagitis was the result, not the cause, of recurrent vomiting.

Finally, we wish to point out that the term "mental impairment" is not synonymous with mental handicap but is legally defined within the Mental Health Act 1983.

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1 Kuruvilla J, Trewby PN. Gastro-oesophageal disorders in adults with severe mental impairment. *Br Med J* 1989;**299**:95-6. (8 July.)

Sexual behaviour of men

SIR,—Drs D Forman and C Chilvers reported that 1·7% and 2·9% of two groups of 480 white men (with non-completion rates of <17% and <2%) admitted to having engaged in homosexual intercourse.[1] These figures were underestimates, contended a representative of the Lesbian and Gay Medical Association.[2]

In the United States 28 659 non-institutionalised adults were randomly surveyed (with a rejection rate of 11%) to determine their risk factors for AIDS. Respondents were asked whether any of the following (without designating which) was true of them: "hemophilia; native of Haiti, Central or East Africa; you are a man who has had sex with another man at some time since 1977, even once; taken illegal drugs by needle; been the sex partner of any person who would answer 'yes' to any of the above; engaged in prostitution at any time since 1977." Each month between 2% and 3% of respondents answered yes (2-4% of men (median 3%, mean 2·9%) and 1-2% of women (median 2%, mean 1·8%).

Thus if 3% is taken as the best estimate of those "at risk" that represents about 5·3 million adults and about 3·3 million men. Yet if 3% of the 83 million men in the United States had engaged in homosexual acts over the past 10 years this would represent 2·5 million men and if 4% 3·3 million men. Thus a figure of 4% for the proportion of men engaging in homosexual acts would mean that all the men at risk of AIDS had homosexual acts as a risk factor in addition to any other risk factors. If 2·5% is taken as the best estimate of the population at risk of AIDS (about 2·7 million men) then the figure of 4% for those who have engaged in homosexual acts is impossible.

In 1989 the Kinsey Institute and the Family Research Institute (with rejection rates of 57% and 53% respectively) reported estimates of male bisexuality or homosexuality of 6·2% and 5·8%.[3,4] How can these findings be reconciled with the lower estimates of homosexuality by Forman and