patent ductus in a 25 week preterm infant); one case of bowel atresia; two infarcts with genetic defects; two infants with genito-urinary defects (including uterus didelphys in a spontaneous abortion); and one hermaphrodite with a cleft palate.

(3) Doses of vitamin A used to produce cleft palate (and other defects) in rodents are usually in the range 100 000-600 000 IU/kg/day. (The particular experiment quoted by Dr Bound[1] used 60 000 IU daily to rats.) This is equivalent to $5\text{-}30 \times 10^6$ IU/day to an adult woman on a weight for weight basis. Pregnavite Forte F provides 4000 IU daily. Dietary intake in early pregnancy is usually 1000-4000 IU daily.[2]

(4) Some studies suggest that vitamin supplements (including vitamin A) may reduce the recurrence of cleft lip with or without cleft palate in humans but not of isolated cleft palate.[3]

Dr Bound suggests that dietary advice or treatment with folic acid as suggested by Laurence *et al*[4] is preferable to Pregnavite Forte F. The relative efficacy of these treatments in preventing neural tube defect has not yet been determined, and no claim has been made that either prevents cleft palate. It is, however, worth noting: (*a*) that if the problem is essentially a dietary one it is unlikely that a single nutrient is responsible[2]; and (*b*) that Laurence *et al*[4] advocate giving folic acid in a dose of 2 mg twice daily, which is about 10 times the recommended daily allowance.

There is no good evidence of vitamin A teratogenicity in the human, and no evidence at all with doses below 10 000 IU daily. It would be unfortunate if Dr Bound's report was interpreted as evidence of a teratogenic risk from Pregnavite Forte F.

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[1] Woollam DHM, Millen JW. Effect of cortisone on the incidence of cleft-palate induced by experimental hypervitaminosis-A. *Br Med J* 1957;ii:197-8.
[2] Smithells RW, Ankers C, Carver ME, Lennon D, Schorah CJ, Sheppard S. Maternal nutrition in early pregnancy. *Br J Nutr* 1977;**38**:497-506.
[3] Tolarova M. Periconceptual supplementation with vitamins and folic acid to prevent recurrence of cleft lip. *Lancet* 1982;ii:217.
[4] Laurence KM, James N, Miller MH, Tennant GB, Campbell H. Double-blind randomised controlled trial of folate treatment before conception to prevent recurrence of neural-tube defects. *Br Med J* 1981; **282**:1509-11.

Occult pneumococcal bacteraemia and febrile convulsions

SIR,—The review by Dr P McIntyre and others (15 January, p 203) highlights how little is known of the incidence of paediatric bacteraemia. I have reviewed cases of bacteraemia in children aged 1 month to 16 years from 1979 to 1981 at the East Birmingham Hospital, a large district general hospital with an active infectious diseases unit. The range of organisms isolated was very different from that seen at Alder Hey Hospital. During my study 84 significant isolations were made. *Staphylococcus aureus* was the commonest organism—25 cases (30%)—and 17 patients had a bacteraemia due to salmonella. These patients had been admitted to the infectious diseases unit. *Haemophilus influenzae* was the third most common isolate. *Streptococcus pneumoniae* accounted for only 8% of isolates (seven cases). Apart from *Escherichia coli* (five cases) the remaining organisms were represented by single cases.

The national incidence of paediatric pneumococcaemia is 10·2% (Young S, personal communication). In five of our seven cases the source of infection was known, but in the remaining two this was not apparent. Both these patients, aged 5 and 16 years, had an upper respiratory tract infection, and the latter had meningism but normal cerebrospinal fluid.

No papers have been published on the incidence of paediatric bacteraemia in the United Kingdom. The only papers from the United States concentrate on occult pneumococcaemia, which accounts for about 60% of isolates from children. Most of these patients come from "walk in" clinics. In Britain most of these children would be seen by their general practitioners, who would not initiate investigations. Only the minority who have complications are seen in hospitals.

The association between febrile fits and occult pneumococcaemia is interesting and has not been seen in this hospital. It is important to know if this is a consistent finding. The difference in incidence of paediatric bacteraemia between Alder Hey and East Birmingham hospitals shows how important a multicentre trial is in determining the true extent of the problem.

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Deaths and dental anaesthetics

SIR,—While not wishing to become involved in a discussion of the issues raised in Dr Brian Lewis's leading article (1 January, p 3), there are three major statistical errors, partly caused, I suspect, by the transfer of data from other papers, which require correction.

(1) In common with many others Dr Lewis has assumed that the figures published by the Dental Estimates Board represent the number of general anaesthetics given in general practice. This is not so: the estimates submitted for payment under item 13 are not required to specify the agent used or the depth of anaesthesia or sedation achieved. The figure of 10 167 000 over 10 years includes all National Health Service anaesthetics and sedations but not the number of general anaesthetics administered by private contract.[1] The proportion of anaesthetics to sedations cannot readily be determined and will certainly vary from year to year. The Dental Estimates Board figure cannot therefore be used as an estimate of the population at risk of death from dental anaesthesia.

(2) In calculating the figure of 10 deaths for every million anaesthetics given in general dental practice it seems that the Dental Estimates Board item 13 has been divided by 100. This figure, apparently taken from table II in the paper "Deaths associated with dentistry,"[2] represents the number of general anaesthetic deaths reported between 1970 and 1979 for all dentistry and includes those occurring in hospital and the community dental service. These are subdivided in table V of the same paper, where the figure of 49 is recorded for those which occurred in general dental practice. If the assumption made in this paper is accepted the figure of 11 430 000 in table VII can be taken as an estimate of the population at risk in general dental practice for the 10 years 1970-9. As 49 deaths were recorded, in 36 of which the general anaesthetic was deemed to be the sole factor, the rates become 4·3 and 3·1 for every million anaesthetics given respectively.

(3) Dental Estimates Board figures are quoted again later in the same article, where the assumption is made that the 52 960 payments under item 13 to operating dentists were all for the administration of a general anaesthetic. A proportion of these would certainly be for sedation or relative analgesia. A further survey would be required to determine current practice.

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[1] Dinsdale RCW, Dixon RA. Anaesthetic services to dental patients: England and Wales 1976. *Br Dent J* 1978;**144**:271-9.
[2] Coplans MP, Curzon I. Deaths associated with dentistry. *Br Dent J* 1982;**153**:357-62.

Breast prostheses and seat belts

SIR,—The Klunk Klip, recommended by Dr M J Minton (8 January, p 147) for patients who have had a mastectomy or breast or chest wall surgery, obstructs the retraction of the belt at the upper anchorage and thus introduces a degree of slackness in the belt as it lies across the body. This may be dangerous.

In a road accident the forces created on impact which hurl the body forward in a way which would maim or kill if no seat belt were being worn develop in a minute fraction of time—a few milliseconds—and are transferred to the car occupant in the same kind of time scale. It is essential that the seat belt should lock (in the case of an inertia reel belt) and restrain the seat occupant before his body develops the tremendous forward momentum which would do much more harm to the chest when it hits a slack belt than any conceivable discomfort experienced in ordinary wear. It is not true that the Klunk Klip does not interfere with the working of the belt. It is true that it does not prevent the belt from locking or restraining the wearer, but it does delay the moment when it begins to restrain the occupant so that the body is moving with greater momentum than it need be.

It does not require much imagination to visualise the effect of this greater impact between the body and a slack belt on people who have undergone surgery in an area where the belt is in contact. There is a margin of safety in seat belt fit which allows a small amount of slack without risk, and the makers of the clip take advantage of this to advise purchasers that they should limit the slack to that amount. The wisdom of sacrificing this safety margin in return for an unnecessary and doubtful addition to comfort must be questionable. There must be other ways of protecting people who have had chest surgery if this is necessary, without meddling dangerously with the seat belt, which is made to fit with only the necessary degree of light pressure on the body.

A particularly unacceptable feature of devices like Klunk Klip and others which interfere with the working of seat belts is that once they are in use the initial instructions are soon lost or forgotten and the importance of readjusting the clip when a different occupant takes the car seat is overlooked. A small wife following a large husband in the driving seat may well not even notice that the belt is too