findings can be applied to female genital mutilation occurring in a totally different cultural setting.

Finally, as paediatricians we condemn male circumcision; we were putting it into the context of the more extensive operations in females. It is now more than 46 years since Gairdner, in a classic paper, demolished the case for routine male circumcision.[5]

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Faecal incontinence in hospitals and residential and nursing homes for elderly people

EDITOR,—S M Peet and colleagues report a high prevalence of faecal incontinence in hospitals and residential and nursing homes for elderly people.[1] Faecal incontinence causes distress to patients and increases the amount of care they require. It also presents problems for infection control as it facilitates the spread of infectious intestinal disease to residents, patients, and their carers.

Between January 1992 and December 1994 the Public Health Laboratory Service Communicable Disease Surveillance Centre received detailed information on 1275 general outbreaks of infectious intestinal disease. Three hundred and sixty of these occurred in nursing or residential homes, geriatric or psychogeriatric hospitals, or wards for this subset of patients in general hospitals. The mean attack rate was 38%, with 7966 people affected and 37 deaths. There were 179 admissions to hospital from the 282 outbreaks that occurred outside hospital settings. Pathogens or toxins were identified in 306 of the 360 outbreaks: 186 outbreaks were due to small round structured virus, 56 to *Salmonella* sp, 24 to *Clostridium perfringens*, 18 to rotavirus, and 22 to other pathogens. Three quarters (271) of these outbreaks were reported to have been transmitted principally by the person to person route.

The duration of the outbreaks ranged from one to 71 (median seven) days in the 324 outbreaks for which duration was reported (fig 1). One hundred and twenty five outbreaks lasted 10 days or longer.

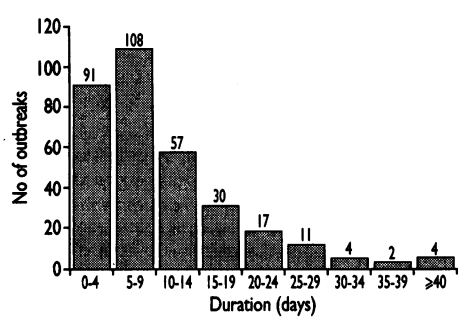

Fig 1—*Duration of outbreaks of infectious intestinal disease in nursing and residential homes and in hospitals or wards for elderly people reported to Communicable Disease Surveillance Centre, 1992-4 (n=324; 36 values are missing)*

The prolonged duration of many outbreaks with a foodborne component suggests that the initial cases were due to foodborne infection and that secondary spread maintained infection in the units.

Outbreaks in these settings are common because of the vulnerable populations that the units contain. Faecal incontinence is likely to be an important factor in these outbreaks, and adequate management of incontinence and the maintenance of good hygiene are essential if such outbreaks are to be avoided or rapidly controlled. Recently published guidelines on infection control in these settings should prove useful.[2,3]

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1 Peet SM, Castleden CM, McGrother CW. Prevalence of urinary and faecal incontinence in hospitals and residential and nursing homes for older people. *BMJ* 1995;311:1063-4. (21 October.)
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Weightings for analysing general practices' prescribing

Pooling of data from practices was inappropriate

EDITOR,—In their paper presenting cost weightings based on patients' age and sex for general practice prescribing within therapeutic groups Lloyd and colleagues[1] use methods that they suggest are "slightly different" from those that colleagues and I used in a study.[2] In fact, the methodology is crucially different. Although Lloyd and colleagues obtained data separately for each practice, the prescribing costs and items by age, sex, and therapeutic group were then pooled across practices. This pooling does not allow any modelling of practice effects, which should certainly be taken into account, given the known variability in prescribing habits among practices. By contrast, statistical modelling to derive the weighting known as the ASTRO-PU (age, sex, and temporary resident originated prescribing units)[2] treated practices as fixed effects and derived estimates of relative rates of prescribing between the age-sex groups in practices, thus adjusting for interpractice variability. Given that data for individual practices were available, the pooled approach used by Lloyd and colleagues is inappropriate.

The differences between practices supplying data to the General Practice Research Database and those supplying data to the MediPlus database, as shown by figures 1 and 2 in the authors' study, are appreciable in view of the large numbers of patients. Nationally, about a tenth of women are aged 65-74, which means that about 150 000 and 38 000 patients are in this age-sex group in the practices supplying data to the General Practice Research Database and the MediPlus database, respectively. For drugs acting on the central nervous system the difference in rates is about 0·25 items per patient (4·38 as against 4·12, estimated from figure 1), and for endocrine drugs it is over 0·5 items (1·22 as against 0·68). No indication is given of the standard errors, but these differences are considerable and cast doubt on the typicality of participating practices and the completeness of the recording systems.

Finally, I note that in table III the headings "male" and "female" for ASTRO-PU weights have been incorrectly ascribed, and I wonder whether this also applies to the comparative columns for the IMS weights.

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Authors' reply

EDITOR,—Sarah J Roberts is right in saying that the analysis that resulted in the ASTRO-PU weightings was more sophisticated, involving a log linear model, than that used to produce the STAR-PU weightings. We do not agree, however, that our simpler model, which used aggregated data, was inappropriate. We had a much larger sample than that available for the ASTRO-PU work, and the costs were direct rather than inferred from other data. Since we had these advantages we thought that the simpler analysis was appropriate.

The difference in items per patient for drugs acting on the central nervous system for women aged 65-74 (the rate mentioned by Roberts) is actually smaller than that suggested by the figures. The mean (SD) values are 4·30 (1·71) for the MediPlus database (n=112) and 4·41 (1·51) for the General Practice Research Database (n=510). A t test with unequal variances gives t=0·68 (620 df), which is not significant.

We thank Roberts for pointing out the typesetting error in table III: the headings "male" and "female" should be reversed for both pairs of columns in this table.

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Early controlled clinical trials

EDITOR,—Carla L van der Wijden and John A Overbeke state that the publication of randomised clinical trials started in the late 1940s.[1] They cite a Dutch trial of paludrine in malaria and the Medical Research Council's first trial of streptomycin in pulmonary tuberculosis, carried out by the council's Tuberculosis Research Unit; both trials were reported in 1948. I wish to question this priority, favoured though it is by many authorities (for example, Cochrane[2]), and propose that the Medical Research Council's trial of patulin in the common cold, published four years previously,[3] deserves a place among the trials that initiated this new era of medical investigation and treatment.

The patulin trial was carried out in over 1000 British factory workers and civil servants. It was double blind and placebo controlled, and the controls were similar to the subjects in the intervention group on entry to the study and received similar instructions. A strict alternation scheme was devised by the clinical organisers (of whom I was one), which ensured an effectively random allocation of the subjects to patulin and placebo. Why has this trial been overlooked? Is it because attention to the validity of therapeutic trials was generally stimulated by the scheme based on random sampling numbers provided by Bradford Hill to Marc Daniels and me for our use in the

Tuberculosis Research Unit for the streptomycin trial,[4] which subsequently (from 1948) served as a model for randomisation in many later randomised controlled trials? Or is it because the results of the patulin trial were negative and those of the streptomycin trial made medical history?

I have previously quoted Bradford Hill's recollection that his statistical, and my medical, experiences in this field converged when we prepared for the streptomycin project.[5,6] The patulin trial certainly influenced my thinking. Yet, so far as I am aware, the literature on the modern clinical trial contains no recognition of this trial, despite its many deserving features. Should not this omission be rectified?

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Recent advances in obstetrics

Testing for Down's syndrome carries too much stress

EDITOR,—I would like to highlight some of the points that Philip Steer made about serum screening for Down's syndrome,[1] in the light of personal experience.

My first reaction to the offer of an antenatal appointment to discuss a risk factor of 1 in 165 for Down's syndrome was one of surprise. I thought that this was too low a risk to contemplate amniocentesis. My second reaction was to consider the implications seriously for the first time and to realise my reluctance to go through with the late abortion of a fetus that might have the potential for a reasonably independent and fulfilled life. I thought it important, however, that my non-medical husband should have more information, so we attended the clinic. Ironically, I missed a postgraduate course on antenatal screening in order to do so.

The visit was far from satisfactory. I was persuaded to have more detailed ultrasound scanning, despite having declined amniocentesis. The ultrasound scan showed the presence of chorion villus cysts, a marker for Down's syndrome but present in 3% of normal fetuses. Suddenly the risk of Down's syndrome was increased, but in an unquantifiable way. The consultant now strongly recommended amniocentesis, even if only to prepare or reassure us, and enjoyment of my normal pregnancy was spoiled. I was anxious and confused. What in fact was the risk of Down's syndrome? My husband felt sure that he could not cope with a child with learning disabilities and preferred not to think about it, perhaps hoping that he would feel differently in the event if he had to. He respected my decision not to have a termination, and we both agreed to decline further testing.

Our anxiety persisted to the point of delivery and beyond. I was convinced for a few awful minutes, before reason intervened, that my perfectly normal baby (shocked after a rapid delivery) did have features of Down's syndrome. Now 2 years old, he is generally healthy but has had more than his fair share of medical problems. I wonder whether my experience in pregnancy had some affect on my ability to copy with his subsequent illnesses.

Serum screening has low sensitivity (0-48% in the studies quoted), causes parents high levels of anxiety, puts normal fetuses at risk from amniocentesis, and is financially expensive. Routine use should be stopped, and adequate counselling of patients is obligatory.

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Figures on screening for Down's syndrome are inaccurate

EDITOR,—Philip Steer's comments about serum screening for Down's syndrome[1] should not go unchallenged. Firstly, it is inappropriate to cite a 48% detection rate. For a 5% false positive rate the estimated detection rate is 59% when dates are used to estimate gestational age and 65% if an estimate based on an ultrasound scan is used.[2] The Barts Down's syndrome demonstration project, in which 48% of cases were detected, was not designed to estimate the detection rate and was not large enough to do so.

Secondly, a serum screening test carried out on a community basis costs much less than the £80 cited. There are existing NHS programmes which are provided at a quarter of this sum.

Thirdly, contrary to Steer's statement, we know that screening has had a considerable effect on reducing the birth prevalence of Down's syndrome. In the absence of screening and at a time of increasing maternal age, the birth prevalence of Down's syndrome would have increased from 1·41/1000 in 1989 to 1·47/1000 in 1993,[3] assuming a natural fetal loss rate of 27% among the women who had terminations. In fact it has decreased from 1·11/1000 in 1989 to 0·92/1000 in 1993. This is equivalent to an increase in the percentage of affected births avoided through screening from about 21% to 37%.

Finally, it is acknowledged that the provision of adequate information before the screening test and appropriate counselling afterwards is important and is not always carried out satisfactorily. This is not the fault of the screening test but is a fault in the way the screening test is delivered to women. It underlines the fact that the test itself is but one component of the screening procedure.

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Epidural analgesia in labour

EDITOR,—The comments on epidural analgesia for labour in Philip Steer's review overlooked important new evidence relating to postpartum backache and a potential hazard of ambulatory epidural analgesia.[1]

Steer implies that the use of epidurals in labour results in increased rates of long term backache. However, the reference supporting this is a retrospective review of only 39% of a population of women who delivered their baby at a given hospital over an eight year period.[2] Apart from the inherent drawbacks in making causal associations in a retrospective study, recall bias over such a protracted period would tend to render its conclusions unreliable.

On the contrary, a more recent prospective study of 1042 women interviewed during their admission for delivery and again two months later found no difference in the incidence of new postpartum back pain between those who received epidural analgesia for labour (44%) and those who did not (45%).[3] The design of this study is more appropriate to assess cause and effect, and the results impressively refute the suggestion that epidural analgesia is a risk factor for "long term backache."

Furthermore, although Steer correctly states that the combined spinal-epidural technique of ambulatory labour analgesia may cause a greater degree of maternal hypotension than conventional epidural analgesia, he does not mention its potential impact on posterior column sensation, particularly proprioception.[4] This may undermine safe ambulation, although there is no doubt that the ambulatory technique improves maternal satisfaction and that retention of lower limb mobility is an advance, even if actual walking may not be advisable.

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Transmission of HIV from mother to infant depends on many factors

EDITOR,—I was disturbed at the many unfounded assertions about preventing the transmission of HIV from mother to infant in Philip Steer's review of recent advances in obstetrics.[1] He asserts that transmission rates could be halved if women did not breast feed. This oversimplifies a very complicated reality, in which transmission to infants depends on many other factors as well; women who have HIV related illnesses may be at high risk of transmitting HIV to their infants whether they breast feed or not.

Secondly, Steer asserts that caesarean section reduces transmission by a "further 40%." The evidence for this is far from clear cut, and the study cited does not draw this conclusion. No large controlled trials have ever been done, and no one has ever recommended routine caesarean section for HIV positive pregnant women on the basis of the existing evidence. Nor does Steer even consider that women with HIV might be at higher risk of complications from caesarean section, when there is at least anecdotal evidence that this is so.[2]

Thirdly, the role of zidovudine during pregnancy to prevent transmission to infants needs to be confirmed. One trial in one country is not enough. Just as the use of zidovudine in most developing countries for this purpose is highly unrealistic, given the cost,[3] we might ask whether the NHS would pay for all pregnant women with HIV infection to use it. If not, there is no benefit.

Thus for Steer to reach the conclusion in a few sentences that, with these three (in his eyes) apparently simple precautions, vertical transmis-