## Increased risk of sudden infant death syndrome in older infants at weekends

SIR,—I enclose a table to support the suggestion of Dr M F G Murphy and colleagues that cot death (sudden infant death syndrome) occurs more frequently at weekends (9 August, p 364). It combines data from the only studies citing the day of the week of such deaths published before the article of Murphy *et al.*<sup>14</sup> The numbers are self evident. When the data are combined there is a significant excess of cot deaths on Saturday to Monday compared with Tuesday to Friday  $(\chi^2=18\cdot34, df=1, p<0\cdot001)$ .

Cot deaths by day of week in four studies

Study	Sat	Sun	Mon	Tues	Wed	Thur	Fri
Peterson <sup>1</sup>	30	24	27	23	19	27	23
Emery <sup>2</sup>	14	23	25	14	20	11	13
Froggatt <sup>3</sup> Cameron and	21	27	25	16	21	18	20
Asher <sup>4</sup>	26	43	35	20	19	21	19
Total	91	117	112	73	79	77	75

The article that disputes this observation, 5 cited by Dr Murphy and colleagues, provided no data and included only a brief statement to the effect that there is no variation by day of the week. This is clearly in error. Why do more cot deaths occur on weekends? I suppose because *everyone* takes weekends off.

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- 1 Peterson DR. Sudden unexpected deaths in infants. Am J Epidemiol 1966;85:478-82.
- Emery JL. Epidemiology of "sudden unexpected, or rapid" deaths in children. Br Med J 1959;ii:925-8.
   Froggatt P. Epidemiologic aspects of the Northern Ireland study.
- 3 Froggatt P. Epidemiologic aspects of the Northern Ireland study. In: Bergman AB, Beckwith JB, Ray CG, eds. Sudden infant death syndrome. Seattle: University of Washington Press, 1970: 32-46.
- 4 Cameron AH, Asher P. Cot deaths in Birmingham 1958-61. Med Sci Law 1965:5:187-99.
- 5 Peterson DR. Evolution of the epidemiology of sudden infant death syndrome. Evidemiol Rev 1980;2:97-112.

## Antiviral treatment in chronic infection with hepatitis B virus

SIR,—We have estimated that 1-2% of the population of the United Kingdom is positive for hepatitis B surface antigen (HBsAg), a figure that was challenged by Dr M A Vickers (28 June, p 1742). We do not feel, however, that we have overestimated the extent of the problem. Although surveys of blood donors in the 1970s showed a prevalence of 0·1-0·4%, these figures are almost certainly an underestimate, as drug addicts, patients with a history of hepatitis or jaundice, and those with liver disease are unlikely to volunteer blood.

In our present society ethnic origin is the main factor that will determine the overall rate of carriage of HBsAg as many immigrants will have come from areas with a carriage rate of 10%. In a study from Holland based on three separate areas in which all women were screened antenatally for HBsAg regardless of risk factors, 0·8% (of 28 412) were found to be positive. A breakdown showed that in Rotterdam and Utrecht 2·1% and 2·3% respectively were positive for HBsAg while in a more rural area, Twente, 0·3% were positive (S W Schalm, personal communication). The differences between the urban and rural areas were accounted for by the relative proportions of immigrants.

Assuming, firstly, that a similar pattern obtains in the United Kingdom and Holland and, secondly, that women with significant liver disease rarely conceive, and knowing that adult males become carriers more commonly than adult females, our estimate of HBsAg carriage in the UK as being 1-2% appears to be a reasonable one.

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1 Mazel JA, Heijtink RA, Schalm SW, et al. Gecomineerde passieve en actieve immunisatie van Zuigelingen van HBsAgpositieve moeders. Ned Tijdschr Geneeskd 1985;129:590-4.

## Royal Medical Benevolent Fund Christmas appeal

SIR,—Christmas and charity have much in common, as both are about giving to others. The Royal Medical Benevolent Fund was founded in 1836 as the medical profession's own charity. The idea of making a special effort for Christmas is more recent. Thanks to the generosity of the medical profession, all our beneficiaries receive some recognition. This may be just a card or money for a gift of up to £80. Many letters are received in the office as evidence of how much this is appreciated. It would be wonderful if the Christmas appeal in this, the fund's 150th year, resulted in a record sum. It takes many gifts to achieve the amount required to give each beneficiary a Christmas box. Gifts may be sent, marked "Christmas appeal," either to the Secretary (B), at the address below, or to the medical representative or treasurer of the local guild of the fund.

JOSEPHINE BARNES

Royal Medical Benevolent Fund, London SW19 8QN

## Prevalence of polycystic ovaries in women with anovulation and idiopathic hirsutism

SIR,—The paper by Dr J Adams and colleagues (9 August, p 355) interested us because we have used ultrasound to study the ovaries of women with anorexia nervosa.1 We agree that ultrasonography is extremely useful in investigating women with dysmenorrhoea and oligomenorrhoea but are concerned that the technique is being stretched beyond its limits in being used to assess ovarian stroma density. Tissue characterisation is notoriously difficult to assess ultrasonically, being dependent on many factors including the machine settings and initial attenuation of the beam by superficial adipose tissue. There is also enhancement of the sound beam distal to a cystic structure, and hence apparently dense ovarian stroma in polycystic ovaries may be artefactual.

The paper implies that the patients underwent only one ovarian scan. We feel that one scan is inadequate to assess ovarian morphology, which in premenopausal women is continually changing. We have now studied the ovaries of 55 patients with anorexia nervosa. All such patients have, at one stage of their illness, ovaries containing many cysts. We found that with weight gain the ovaries became normal when the patient reached her premorbid weight.

The clinical and biochemical features of anorexia nervosa are appreciably different from those of the polycystic ovary syndrome, and we are concerned that the authors appear to diagnose the syndrome on the appearance of the ovaries alone. Ultrasound gives information on the uterus. In all our patients with anorexia nervosa the uterine area was half that of normal women and was one of the final features to be restored at optimum weight. This is undoubtedly due to the low oestrogen concentrations encountered in these patients as opposed to the high concentrations seen in the polycystic ovary syndrome. The authors make no mention of uterine dimensions.

In conclusion, we believe that the wide range of endocrine results noted in this paper support our view that many different populations of women are being included under the heading "polycystic ovaries."

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1 Treasure JL, King EA, Gordon PAL, Wheeler M, Russell GFM. Cystic ovaries: a phase of anorexia nervosa. *Lancet* 1985;ii: 1379-81.

AUTHORS' REPLY—The answer to most of the points raised by Dr Gordon and her colleagues may be found either in our paper in the BMJ (9 August, p 355) or in an earlier report.<sup>1</sup>

We concede that it is unwise to attach too much importance to the echogenicity of the ovarian stroma on ultrasonography, but, as is stated in our two papers, it is the increased volume of stroma that is the most important feature in distinguishing polycystic ovaries from the multifollicular appearance of the ovaries in women with amenorrhoea related to weight loss. The stroma is frequently hyperreflective—"dense"—but this is not the primary criterion for diagnosis.

Nowhere is it implied that only one scan was performed. Nearly all patients underwent at least two scans. One of the important points of distinction between polycystic and multifollicular ovaries, which we have been at pains to explain in our papers, is that the polycystic appearance-multiple cysts and increased stroma-persists even in the presence of normal cyclical changes in the ovary. In contrast, the multiple follicular appearance of the ovaries of underweight women changes when the patient gains weight or is given luteinising hormone releasing hormone, a finding entirely consistent with that of Treasure et al.2 Another important objective of the paper of Adams et al,1 which appears to have been missed by Dr Gordon and her colleagues, was to emphasise the pronounced differences in clinical and biochemical features between women with amenorrhoea related to weight loss and multifollicular ovaries and those with polycystic ovaries.

We are well aware that ultrasound gives information on the uterus. In our previous paper, not only were uterine sizes in women with multifollicular or polycystic ovaries documented but, in contrast to their study, serum oestradiol concentrations were also given and the mechanism of oestrogen metabolism fully discussed.

Finally an important thesis of our paper is that the clinical and biochemical presentation of women with polycystic ovaries is indeed wide ranging. We do not claim a precedent for this. Goldzieher and Green showed this to be the case in women with polycystic ovaries proved at wedge biopsy. The problem arises in the use of the term polycystic ovary "syndrome." There is no general agreement as to what constitutes the syndrome, and that is why we were careful to use this term sparingly in our paper. The classic Stein-Leventhal syndrome of anovulation, obesity, and hirsutism with raised serum concentrations of luteinising hormone and androgens is but one end of the range