that of Dr Toon and colleagues, show a much higher isolation rate of organisms (table). We found no difference in contraceptive habit or smoking habit between these groups. We agree that many of these isolates can be regarded as normal lower genital tract commensals, but we regard *Chlamydia trachomatis*, *Trichomonas vaginalis*, and *Gardnerella* as pathogenic organisms.

Recent data from Dundee¹ and Gateshead² indicate that many women with mildly atypical findings on cervical cytology have cervical intra-epithelial neoplasia requiring treatment. Nevertheless, colposcopic facilities are not infinite and it should be remembered that some women with inflammatory smears will have a microbiological infection, which may respond to simple antimicrobial therapy without the need for colposcopic examination.

S A WALKINSHAW

Department of Obstetrics and Gynaecology,
Princess Mary Maternity Hospital,
Newcastle upon Tyne NE2 3BD

A D G ROBERTS

Leicester Royal Infirmary,
Leicester

JAMES W CORDINER

Western Infirmary,
Glasgow

1 Walker EM, Dodgson J, Duncan ID. Does mild atypia on a cervical smear warrant further investigation? *Lancet* 1986; ii:672-3.

2 Souttar WP, Wisdom S, Brough AK, Monaghan JM. Should patients with mild atypia in a cervical smear be referred for colposcopy? *Br J Obstet Gynaecol* 1986;93:70-4.

Childhood respiratory infection and adult chronic bronchitis in England and Wales

SIR,—Professor D J P Barker and Dr C Osmond (15 November, p 1271) used the method of Osmond and Gardner¹ to analyse deaths from lung cancer and chronic bronchitis in England and Wales during 1941-80. For both diseases they found a similar period of birth (cohort) pattern with a peak around 1900-5 for men and 1925 for women: they attributed this to the different smoking habits of successive generations. They found little evidence of a period of death effect for lung cancer but a sharp decline in both sexes for chronic bronchitis, which they attributed partly to the advent of antibiotics and partly to the decline in infant bronchitis and pneumonia in the early decades of this century.

These conclusions must be treated with extreme caution. One reason is that the estimated period of birth patterns are impossible to reconcile properly with data on smoking habits, particularly for women. Consider the smoking habits of two cohorts of women, born in 1905 and 1945, with a similar estimated period of birth value. As shown in the table, the cumulative cigarette consumption of these cohorts at any given age is vastly different. While epidemiological studies can be interpreted in terms of some benefit from tar reduction² it is difficult to see how this could explain this large anomaly. Consideration of the detailed data suggests that the declining cohort trends in risk of lung cancer and chronic bronchitis, seen in both sexes, must, at least in part, have been due to a decline in the effect of some risk factor other than smoking, quite possibly air pollution.

Cumulative cigarette consumption (thousands per adult) in two cohorts of women

Age:	20	30	40	60	80
Born 1905	0.2	2	12	43	63
Born 1945	8	36	65		

²Estimated from Lee³ with annual supplements.

A second problem lies in relating the declining period of death effect for chronic bronchitis to childhood respiratory infection. If childhood infection does have an important role it should emerge from the Osmond and Gardner analysis as a birth cohort effect, not as a period of death effect.

Arguably, the Osmond and Gardner technique is useful for discriminating those diseases which are mainly characterised by birth cohort changes and those which are mainly characterised by period of death effects. Where both effects emerge from the analysis, as for chronic bronchitis, it is important, firstly, to test whether the model fits the data and, secondly, to remind readers that there are a range of other (very different) period and cohort variables that fit the data as well. Barker and Osmond do neither and overinterpret the results of these analyses.

PETER N LEE

Sutton,
Surrey SM2 5DG

1 Osmond C, Gardner MJ. Age, period and cohort models applied to cancer mortality rates. *Statistics in Medicine* 1982;1:245-59.

2 Lee PN, Garfinkel L. Mortality and type of cigarette smoked. *J Epidemiol Community Health* 1981;35:16-22.

3 Lee PN, ed. *Statistics of Smoking in the United Kingdom*. 7th ed. London: Tobacco Research Council Research, 1976.

SIR,—We disagree with Mr Lee but are grateful for the opportunity to expand on a necessarily abbreviated section of our paper.

Following suggestions made by Breslow¹ and by Day and Charnay² we derived a curve which described the cohort (generation) effects of both lung cancer and chronic bronchitis (figure). For lung cancer this one curve was an adequate description of the time trends. For chronic bronchitis, however, it was necessary to add a further curve which we show as a steeply falling period of death effect (figure).

This analysis helped to determine the choice of identification^{2,3} because a submodel was adequate for lung cancer. We returned to a standard presentation of these results in the paper for simplicity. The identifications were chosen to correspond to the earlier analysis by equating gradients. Goodness of fit statistics and parameter estimates are available on request.

Mr Lee chose inappropriate cohorts for comparison. The figure and those in our paper suggest that lung cancer mortality rates for women are 25% greater in the 1945 cohort than in that of 1905. Disease rates in the two cohorts cannot be directly related because there were no overlapping ages

during the period for which data are available (1941-80). There are a number of reasons why we do not think that Mr Lee's cumulative cigarette consumption measure is helpful. For example, it fails to take account of changes in tar consumption and in the use of filters, and it is based on an overly simple model for carcinogenesis. The average number of cigarettes smoked annually by the 1905 cohort of women when aged 35-39 (that is, during 1941-5) was 1360.⁴ The corresponding figure for the 1945 cohort (that is, during 1982) is 2630.⁵ When tar corrected this reduces to 1230. This illustrates one weakness of Mr Lee's analysis.

Turning to bronchitis, our analysis showed that its trend could be decomposed into a lung cancer component, which we related to smoking, and a component that decreased regularly within each age group, which we related to childhood respiratory infection. Such a decreasing trend may be represented equally well by a cohort or period of death curve.³ Thus we find no contradiction in the appearance of a childhood related trend component in the period curve.

For these and other reasons given in our paper we restate our conclusion that the "decline in infant bronchitis and pneumonia in the early decades of this century may have determined the subsequent fall in age specific mortality from bronchitis in adults."

D J P BARKER
CLIVE OSMOND

MRC Environmental Epidemiology Unit,
Southampton General Hospital,
Southampton SO9 4XY

1 Breslow NE. Extra-Poisson variation in log-linear models. *Appl Stats* 1984;33:38-44.

2 Day NE, Charnay B. Time trends, cohort effects and ageing as influence on cancer incidence. In: Magnus K, ed. *Trends in cancer incidence, causes and practical implications*. New York: Hemisphere Publishing Corporation, 1982.

3 Osmond C, Gardner MJ. Age, period and cohort models applied to cancer mortality rates. *Statistics in Medicine* 1982;1:245-59.

4 Lee PN, ed. *Statistics of smoking in the United Kingdom*. 7th ed. London: Tobacco Research Council, 1976.

5 Office of Population Censuses and Surveys. *General household survey 1982*. London: HMSO, 1984.

Dignity in hospital

SIR,—As a lay reader (albeit a GP's wife) I was particularly interested in the section on "dignity" in Dr Roger Homan's article (29 November, p 1417) as I have just been considering these points after a visit as an outpatient to a general hospital's x ray department.

Dr Homan states that inpatients are conditioned

Mortality trends from bronchitis and lung cancer, men and women, England and Wales 1941-80.

by their hospital surroundings and by the process of depersonalisation into accepting any indignities without embarrassment. That may be so, but what of the outpatient who has just walked in off the street and is still bound by the taboos of the outside world? Although my own examination was one that did not require me to undress, I was disturbed to see other outpatients being obliged to strip off in cubicles situated in a waiting area occupied by patients (and accompanying relatives) of both sexes, on a public corridor. It must have felt like having to undress at a bus stop. Within earshot of all and sundry they were told by the nurse which garments they had to remove, and then had to emerge attired in skimpy hospital gowns (no dressing-gowns being provided), their own clothes bundled in their arms. I felt particularly humiliated on behalf of an elderly man who looked ludicrous in a little white nightie.

I am sure the medical and nursing care given to hospital patients is exemplary, but I am worried by what seems to be an indifference towards patients' susceptibilities. One reason why its "regulars" so cherish the Elizabeth Garret Anderson Hospital is because it goes to great pains to avoid embarrassing patients. Surely it must be possible, without any trouble or financial outlay, to put undressing cubicles in places more secluded than a public highway. Most outpatients are already nervous about their condition and intimidated by hospital surroundings; they should not be subjected to unnecessary embarrassments and indignities.

PAMELA HARRIS

Romford, Essex

Asthma and climatic conditions: experiences from Hong Kong

SIR,—There seems little doubt from the contributions to your journal (4 October, p 843; 8 November, p 1239) and other studies that asthmatic attacks are far from uniform in their frequency over the year.

In 1984 we investigated the pattern of admission of children with asthma to the wards of a busy general hospital in Kowloon, the Caritas Medical Centre, over the four years 1980-3.¹ In 993 patients we found a clear bimodal distribution of admissions for asthma with an autumn wave peaking in November and a smaller spring wave reaching a peak in May (figure).

Pursuing possible explanations for these seasonal variations we analysed the admissions in relation to six climatic factors: temperature, air pressure, humidity, rainfall, sunshine, and windspeed² (information obtained by courtesy of the Royal Observatory, Hong Kong). With a multiple stepwise regression analysis the best model containing two climatic variables was found: number of admissions = $51.41 - 0.26 D_{\text{rain}} + 14.2 AD_{\text{temp}}$ (D_{rain} = difference in monthly rainfall; AD_{temp} = absolute difference in monthly temperature; F value 0.75).

Thus about 75% of the variation in the number of admissions can be attributed to rainfall and temperature (figure). Any change in rainfall would affect asthmatic admissions in the opposite direction—that is, increased rainfall lessening the number of admissions, reduced rainfall increasing them. Any change in environmental temperature in either direction would increase admission rate.

The mechanism(s) whereby these climatic changes bring about asthmatic attacks is (are) unknown. They may simply exist concomitantly with, but not directly cause, asthmatic attacks. The smaller admission peak in late spring might be due to an increase in pollen, fungal spores, and other aeroallergens at a time when the temperature is changing from cool to warm and when rainfall is only moderate. In Hong Kong the summer months bring relatively little fluctuation in monthly temperature but a consistently steady

amount of rainfall. Regular rainfall will wash airborne allergens from the air, thereby lessening chances of inhalation. Rainfall is also accompanied by temperature inversions which tend to force particles nearer to the ground. These factors might explain the trough of asthmatic admissions in July and August. (It is relevant here to recall Derrick's experience in Brisbane, in which he describes fewer attendances with asthma on wet days.³) With the coming of autumn in October and November the gradual influx of a cold dry air mass resulting in much less rainfall might be responsible for a big wave of admissions from a resultant increase in levels of fungal and pollen spores and even air pollutants, which gain less impeded entry into the lungs. The colder weather itself might also have an added effect in increasing bronchial lability either from direct action on the respiratory mucosa or indirectly by reactivating dormant viruses. The small peak in May, which appears to parallel the increase in rainfall at this time, might seem to contradict the rainfall hypothesis. However, a particular type of pollen (or other airborne allergen) might have its principal effect in the spring despite steadily increasing rainfall.

Monthly asthmatic admissions (1980-3) to paediatric wards of Caritas Medical Centre, Hong Kong, in relation to mean monthly rainfall and temperature.² (Reproduced by courtesy of the editor, *Hong Kong Journal of Paediatrics*.)

These ideas are but an endeavour to account for the well defined seasonal variations in admissions to hospital for asthma. There are probably other interpretations. They do, however, conform in large part with other opinions to explain seasonal changes in asthma.⁴ Nevertheless the interesting theories put forward to explain the seasonal variations should not detract from their important clinical implications—namely, that children (and adults) who suffer from asthma must have special attention given to prophylaxis and early treatment of attacks during a season of particular vulnerability.

K W WONG

Department of Paediatrics,
Caritas Medical Centre,
Kowloon

D P DAVIES

Department of Paediatrics,

E M C LAU

Department of Community Medicine,
Chinese University of Hong Kong,
Shatin, NT, Hong Kong

1 Wong KW, Davies DP. Seasonal variations in childhood asthma. *Hong Kong Journal of Paediatrics* 1985;2:19-23.

2 Wong KW, Davies DP, Lau EMC. Asthmatic admissions. *Hong Kong Journal of Paediatrics* 1985;2:272-4.

3 Derrick EH. The short term variation of asthma in Brisbane: its relation to the weather and other factors. *Int J Biometeor* 1969;13:295-301.

4 Derrick EH. Asthma and the Brisbane climate. *Aust NZ J Med* 1972;3:235-46.

Why women are not receiving anti-Rh prophylaxis

SIR,—Dr Marcela Contreras and colleagues (22 November, p 1373) suggest that failure to give anti-Rh immunoglobulin after spontaneous abortion is common and that it accounts for many cases of Rh sensitisation. Tovey showed that 22% of new cases of sensitisations were due to failure of administration, and of these one half arose after spontaneous or therapeutic abortion.¹ However, a recent audit conducted in the Mersey Region does not confirm these observations.

Medical records of new sensitisations arising in the Mersey region in 1983 and 1985 were examined. There were 81 cases and of these 36 (44%) arose because of failure of protection—that is, the patients were given the standard postnatal or postabortion dose of anti-D immunoglobulin but nevertheless developed antibodies by or during the next pregnancy; 11 cases (14%) arose in primigravidae during the pregnancy; and six (7%) were in women whose last pregnancy was before the widespread use of anti-D immunoglobulin. However, only five cases (6%) arose because of a failure to administer anti-D immunoglobulin—three were failures of administration after a full term pregnancy and the remaining two were after spontaneous and therapeutic abortions. The remaining 23 patients "booked" with antibodies but their history with regard to anti-D immunoglobulin was unknown. Some of these cases may have been due to failure to administer anti-D immunoglobulin when indicated.

Although failure to administer anti-D immunoglobulin still accounts for some new sensitisations, this does not seem to be due particularly to failure of administration after early spontaneous abortion. A substantial number of sensitisations arise from failure of protection, which may be the result of insufficient anti-D immunoglobulin having been given. As there is currently a shortage of anti-D immunoglobulin perhaps attention should be focused on the reasons for failure to administer after full term delivery and therapeutic abortion as well as on cases where insufficient anti-D immunoglobulin has been given.

RUTH M HUSSEY

Department of Community Health,
University of Liverpool,
Liverpool L69 3BX

1 Tovey LAD. Haemolytic disease of the newborn—the changing scene. *Br J Obstet Gynaecol* 1986;93:960-6.

Hip fractures in healthy patients

SIR,—Mr R Villar and colleagues put forward a case for treating subcapital femoral neck fractures as "middle of the night" emergencies (8 November, p 1203).

Assessing the results of hip surgery must not be based entirely on social and rehabilitation grounds. There are other reasons for a protected environment that can confuse the hip result. The three recovery categories of Hall and Ainscow are disheartening if related purely to the fracture treatment. In many cases geriatric falls and fractures are the first outward manifestation of what should be an already lowered grading of this type—that is, the changes in the patient or her environment that require a drop in grading are often the precipitating factors of the fracture. Mr R Villar and colleagues suggest the patient's failure to give consent as one reason for delay in surgery, but this strongly suggests that preoperatively factors are already at work necessitating a lowering of independence grade. Delayed diagnosis was another reason given, but in many of these cases the