

chemical findings stable. The packed cell volume was 0.27. With hypertonic peritoneal dialysis his weight decreased by 1.6 kg, but blood pressure remained raised at 230/130 mm Hg, and the next day he suffered two generalised seizures. Packed cell volume was 0.35. Phenytoin was started. He was discharged on 17 December, normotensive with no neurological sequelae, and taking no antihypertensive medication. Phenytoin was subsequently withdrawn. He had a cadaveric renal transplant in 1987 and remained well with a blood pressure of 130/80 mm Hg.

Comment

Neither patient had previously suffered seizures. In both fitting occurred after a sudden rise in blood pressure in association with normal fundi and computed tomograms and in the context of recent blood transfusion and increase in packed cell volume. These features closely resemble those of seizures reported in patients treated with erythropoietin while undergoing dialysis.² As with erythropoietin, the factors precipitat-

ing fits in these patients remain unclear. In case 1 the haemoconcentration associated with haemodialysis may have been relevant. The second patient was not fluid overloaded at the time of the first seizure but was at the time of the second, when rapid fluid removal by peritoneal dialysis might have contributed. Perhaps a rapid rise in packed cell volume resulted in both increased blood viscosity and a loss of hypoxic vasodilatation³ and thus a rise in vascular resistance. These events, superimposed on a possibly abnormal cerebral vasculature, may have resulted in hypertensive encephalopathy.⁴

- 1 Raine AEG. Hypertension, blood viscosity and cardiovascular morbidity in renal failure: implications of erythropoietin therapy. *Lancet* 1988;ii:97-9.
- 2 Edmunds ME, Walls J, Tucker B, et al. Seizures in haemodialysis patients treated with recombinant human erythropoietin. *Nephrology, Dialysis, Transplantation* (in press).
- 3 Neff MS, Kim KE, Persoff M, Onesti G, Swartz C. Hemodynamics of uremic anemia. *Circulation* 1971;43:876-83.
- 4 Byrom FB. Pathogenesis of hypertensive encephalopathy and its relation to the malignant phase of hypertension: experimental evidence from the hypertensive rat. *Lancet* 1954;ii:201-11.

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Hay fever, hygiene, and household size

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Hay fever has been described as a "post industrial revolution epidemic,"¹ and successive morbidity surveys from British general practice suggest that its prevalence has continued to increase over the past 30 years.² Other evidence suggests a recent increase in the prevalence of asthma² and childhood eczema.³ This paper suggests a possible explanation for these trends over time.

Subjects, methods, and results

I studied the epidemiology of hay fever in a national sample of 17 414 British children born during one week in March 1958 and followed up to the age of 23 years (the National Child Development Study). Three outcomes were investigated: (a) self reported "hay fever during the past 12 months" at age 23; (b) parental

report of "hay fever or allergic rhinitis in the past 12 months" at age 11; (c) parental recall of "eczema in the first year of life" elicited when the child was 7. Cross tabulations were performed with the SAS statistical package, and multiple logistic regression models were fitted with the LR program in the BMDP statistical package.

Of the 16 perinatal, social, and environmental factors studied the most striking associations with hay fever were those for family size and position in the household in childhood. The table shows that at both 11 and 23 years of age hay fever was inversely related to the number of children in the household at age 11 (when it is assumed most families were complete). When prevalence figures were adjusted by multiple logistic regression for other significant determinants of hay fever in this cohort (see table) the associations with numbers of older and younger children in the household persisted. These trends in adjusted prevalence were independent of one another and each was significant ($p < 0.01$, see table), but the trends by number of older children were significantly steeper ($\chi^2 = 11.6$, $df = 1$, $p < 0.01$ at age 11; $\chi^2 = 19.5$, $df = 1$, $p < 0.01$ at age 23). A further analysis of hay fever occurring at 23 by birth

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Prevalence of hay fever and of eczema in infancy by position in the household. Numbers in parentheses

| | Prevalence of hay fever in previous year | | | | | | | | Prevalence of eczema in first year of life | | | |
|-----------------------------------------------------------|------------------------------------------|-----------------------|-----------|----------|-----------------------|---------------------|-----------|----------|--------------------------------------------|--------------------|-----------|----------|
| | At age 23 | | | | At age 11 | | | | | | | |
| | Crude* | Crude† | Adjusted‡ | χ^2 | Crude* | Crude† | Adjusted‡ | χ^2 | Crude* | Crude† | Adjusted‡ | χ^2 |
| No of older children (under 21) in household at age 11 : | | | | | | | | | | | | |
| 0 | 20.4 (910/4 470) | 20.5 (810/3 942) | 20.4 | | 9.6 (542/5 622) | 10.0 (389/3 895) | 10.0 | | 6.0 (308/5 096) | 6.2 (245/3 952) | 6.1 | |
| 1 | 15.7 (583/3 703) | 15.5 (515/3 323) | 15.0 | | 8.4 (398/4 721) | 8.3 (273/3 286) | 7.9 | | 5.2 (225/4 331) | 5.3 (177/3 320) | 5.2 | |
| 2 | 11.6 (172/1 478) | 12.1 (157/1 301) | 12.5 | 80.0 | 5.4 (106/1 953) | 4.8 (62/1 290) | 5.0 | 55.4 | 3.9 (68/1 757) | 4.4 (57/1 298) | 4.6 | 12.5 |
| 3 | 9.6 (58/606) | 9.2 (48/520) | 10.6 | | 3.7 (29/777) | 3.3 (17/511) | 4.0 | | 3.6 (25/692) | 3.3 (17/517) | 3.7 | |
| 4+ | 6.5 (21/322) | 6.7 (18/270) | 8.6 | | 2.8 (12/436) | 1.9 (5/268) | 2.6 | | 2.1 (8/381) | 2.2 (6/273) | 2.8 | |
| No of younger children in household at age 11 : | | | | | | | | | | | | |
| 0 | 17.2 (643/3 746) | 17.1 (575/3 354) | 17.9 | | 8.8 (422/4 770) | 8.6 (286/3 319) | 8.9 | | 5.1 (221/4 366) | 5.2 (174/3 356) | 5.3 | |
| 1 | 17.7 (626/3 544) | 17.7 (559/3 151) | 16.9 | | 8.8 (387/4 414) | 8.8 (273/3 120) | 8.3 | | 5.7 (228/4 030) | 5.9 (186/3 145) | 5.7 | |
| 2 | 16.0 (303/1 898) | 16.3 (273/1 678) | 15.7 | 13.4 | 7.3 (179/2 436) | 7.5 (125/1 657) | 7.3 | 10.7 | 5.3 (118/2 222) | 5.4 (91/1 686) | 5.3 | 0.19 |
| 3 | 13.9 (117/841) | 13.0 (93/714) | 13.4 | | 5.9 (67/1 144) | 6.1 (43/707) | 6.5 | | 4.0 (40/997) | 4.3 (31/715) | 4.6 | |
| 4+ | 10.0 (55/550) | 10.5 (48/459) | 12.3 | | 4.3 (32/745) | 4.3 (19/447) | 5.4 | | 4.2 (27/642) | 4.4 (20/458) | 5.3 | |
| Total | 16.5 (1 744/10 579) | 16.5 (1 548/9 356) | | | 8.0 (1 087/13 509) | 8.1 (746/9 250) | | | 5.2 (634/12 257) | 5.4 (502/9 360) | | |

*Using all available information.

†For subjects with complete covariate data included in the multiple logistic regression.

‡Adjusted by multiple logistic regression for the other factor in the table, plus father's social class, housing tenure and shared household amenities in childhood, breast feeding, region of birth, and cigarette smoking at 23.

§Test for linear trend ($df = 1$) from the multiple logistic regression model.

||Includes children of the family living away from home in 1969.

order and number of older children in the household (not shown) suggested that the number of older children was a more influential variable.

Eczema in the first year of life was also independently related to the number of older children in the household (see table). There was no association between eczema in infancy and younger children of the family (who were not yet born).

Comment

Variations in labelling respiratory symptoms probably exist among socioeconomic classes, but it is unlikely that differential reporting could explain the strong relation between hay fever and position in the household, which was independent of the social class of the father. Although the recall by parents of eczema occurring in infants seven years previously might be influenced by total family size, it is less likely to have been affected specifically by the number of older children in the household. Similar gradients in hay fever and eczema with increasing family size were reported at 5 years of age among British children born in 1970.⁴

These observations do not support suggestions that viral infections, particularly of the respiratory tract, are important precipitants of the expression of atopy.⁵ They could, however, be explained if allergic diseases were prevented by infection in early childhood,

transmitted by unhygienic contact with older siblings, or acquired prenatally from a mother infected by contact with her older children. Later infection or reinfection by younger siblings might confer additional protection against hay fever.

Over the past century declining family size, improvements in household amenities, and higher standards of personal cleanliness have reduced the opportunity for cross infection in young families. This may have resulted in more widespread clinical expression of atopic disease, emerging earlier in wealthier people, as seems to have occurred for hay fever.¹

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- 1 Emanuel MB. Hay fever, a post industrial revolution epidemic: a history of its growth during the 19th century. *Clin Allergy* 1988;18:295-304.
- 2 Fleming DM, Crombie DL. Prevalence of asthma and hay fever in England and Wales. *Br Med J* 1987;294:279-83.
- 3 Taylor B, Wadsworth J, Wadsworth M, Peckham C. Changes in the reported prevalence of childhood eczema since the 1939-45 war. *Lancet* 1984;ii:1255-7.
- 4 Golding J, Peters T. Eczema and hay fever. In: Butler N, Golding J, eds. *From birth to five. A study of the health and behaviour of Britain's five-year-olds*. Oxford: Pergamon, 1986:171-86.
- 5 Busse WW. The relationship between viral infections and onset of allergic diseases and asthma. *Clin Exp Allergy* 1989;19:1-9.

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Spare artificial legs

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Providing every amputee with two prostheses as an entitlement was started during the first world war. Prostheses were then made of wood by craftsmen. Conditions and circumstances have changed since, and many doctors and paramedical staff now think that one modern prosthesis would serve the patient better.

Most prostheses made in Britain in the 1940s and 1950s were metal. These took several months to manufacture, several weeks to repair or modify, and several days to adjust. The amputees were young and justifiably required two prostheses so that while one prosthesis was being repaired they could use the "duplicate." Patients today with leg amputations are mostly elderly. In the 1970s "modular" prostheses were introduced, and plastic and carbon fibre were used to make prostheses. Prostheses can now be made within days and resocketed or repaired "while you wait." Thus with faster production and an older patient population many doctors think that only one prosthesis should be provided.

Patients and results

I interviewed 100 patients, aged 22 to 85, who were attending a limb fitting centre about their prostheses. Only adults with unilateral below knee or above knee amputations who had been provided with two prostheses of the same prescription and still had both were included. I excluded patients who, though having been

provided with two prostheses, stated that they had only one, or had lost or mislaid one, or had returned one to the clinic.

Thirty eight patients said that they used both prostheses; 62 used only one and rarely used the other. Twenty six said the unused prosthesis was not comfortable; 18 simply preferred one over the other; 11 said that the unused one was a poor fit; and seven gave no reason or said that they thought one was to be kept in reserve.

Comment

Patients who have had a leg amputated know that two prostheses will be provided, one of which will be a "spare leg"—a term which should be dropped. This is reinforced, perhaps unwittingly, by the paramedical staff and sometimes by the doctor.

The incentive to make a comfortable prosthesis the first time is often lacking because it is assumed that there will soon be an opportunity to make a duplicate. The patient receives the first prosthesis and awaits the "second, even better" leg. One is inevitably more comfortable, and the other is rejected. In practice no two prostheses are identical, and even if both are comfortable one prosthesis is favoured. Sometimes attempts are made to make them identical, and in fact they become less comfortable. One leg is destroyed and a new one made, perhaps with a new component or a new type of foot. The quantity of prosthetic hardware should never overtake quality of patient care. A second prosthesis should be provided only in exceptional circumstances. The patient's views are important, but the final decision is the doctor's, who is answerable as the prescriber.

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