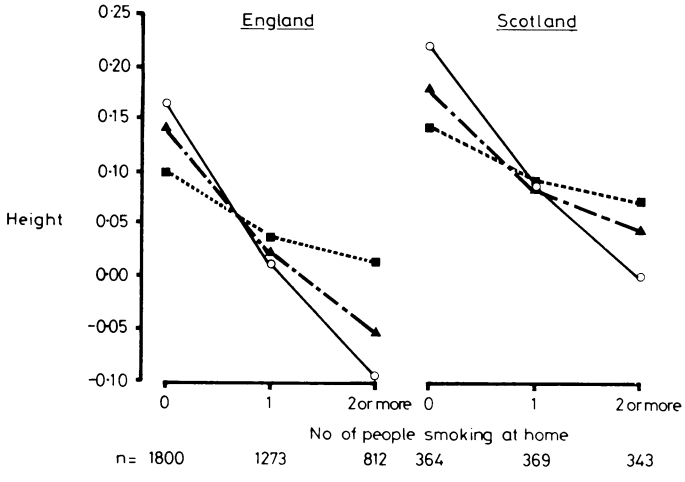
Parental smoking at home and height of children

It is recognised that smoking by mothers during pregnancy is associated with low birth weight[1] and that the subsequent physical growth of children born to these mothers is impaired.[2] Babies with parents who smoke also have more respiratory illness in the first year of life.[3] Data from a study in Cleveland of 539 6- and 7-year-old children suggested an association between short stature in the child and number of smokers in the home. We tested this hypothesis using data from the National Study of Health and Growth, which provided a large sample of primary schoolchildren.

Methods and results

In the National Study of Health and Growth the heights of children in 28 study areas in England and Scotland were measured. Each child's parents gave information on the father's occupation, parents' reported height, number of siblings of the child, and child's birth weight. In 1977 we also included questions on the number of people smoking at least five cigarettes a day at home and the child's respiratory symptoms. The study design and measurement technique have been described elsewhere.[4] The children were divided into three groups according to whether there were no smokers, one smoker, or two or more smokers in the home. In an analysis of variance we tested for a trend of height across the three smoking categories and allowed for variables that might be related to both smoking behaviour and stature. Standardised height by age, sex, and country of residence was obtained for each child as the difference between his height and the mean height in his group divided by the standard deviation for that population.[5]

A strong inverse association was found between height and number of smokers at home ($p<0.001$ in England and $p<0.01$ in Scotland). Children with one smoker at home had an intermediate mean height compared with the two other groups in both countries (figure). After adjustment for the child's birth weight to allow for the mother's smoking during pregnancy the trend was still significant in England ($p<0.001$) but borderline in Scotland ($p<0.1$). After further adjustments for the father's social class, the mother's and father's heights, and the number of siblings a significant trend remained in the English sample ($p=0.01$) and a non-significant trend in the Scottish sample (the Scottish sample was one-third the size of the English). A similar analysis by sex showed a significant trend for boys but not for girls in England after all other independent variables had been allowed for. In Scotland the trend was observed only in girls ($p<0.05$). The number of respiratory symptoms in the previous 12 months did not explain the association between height and smoking behaviour at home.



Mean standardised height of children by number of people smoking five or more cigarettes a day at home.
○——○ = Unadjusted. ▲- - -▲ = Adjusted for child's birth weight. ■- - -■ = Adjusted for child's birth weight, number of siblings, father's social class, and parents' height.

Comment

The association between smokers at home and children's height cannot be explained by smoking in pregnancy because adjusting for each child's birth weight did not eliminate differences in height between the groups. The significant gradient found in England after adjustment for the parents' height, number of siblings, and father's social class is further evidence that the association is due to passive smoking. In Scotland a trend of the same magnitude as in England was not significant.

We investigated whether respiratory symptoms in childhood were a necessary step between passive smoking and a child's impaired growth. Our analysis indicated that this is unlikely. Respiratory symptoms before the child entered school, however, may have played a part.

Passive smoking at home, therefore, seems to affect the growth of children. The maximum difference in mean height between groups was about 1 cm. This was the value reported by Butler and Goldstein[2] in 7- and 11-year-old children whose mothers smoked during pregnancy.

We thank Professor W W Holland and our helpers in the study areas. This study is funded by the Department of Health and Social Security and the Scottish Home and Health Department.

[1] Butler NR, Goldstein H, Ross EM. Cigarette smoking in pregnancy: its influence on birth weight and perinatal mortality. *Br Med J* 1972;ii: 127-30.
[2] Butler NR, Goldstein H. Smoking in pregnancy and subsequent child development. *Br Med J* 1973;iv:573-5.
[3] Colley JRT, Holland WW, Corkhill RT. Influence of passive smoking and parental phlegm on pneumonia and bronchitis in early childhood. *Lancet* 1974;ii:1031-4.
[4] Rona RJ, Altman DG. National Study of Health and Growth: standards of attained height, weight and triceps skinfold in English children 5 to 11 years old. *Ann Hum Biol* 1977;**4**:501-23.
[5] Rona RJ, Florey CduV. National Study of Health and Growth: respiratory symptoms and height in primary schoolchildren. *Int J Epidemiol* 1980; **9**:35-43.

(*Accepted 27 July 1981*)

Department of Community Medicine, St Thomas's Hospital Medical School, London SE1
R J RONA, MED-CIR, PHD, lecturer
C DU V FLOREY, MD, FFCM, professor
G C CLARKE, statistical assistant
S CHINN, MA, senior lecturer in medical statistics

Human papovavirus isolated from urine of a child with acute tonsillitis

The human papovavirus BK has been isolated exclusively from people who were immunologically impaired[1] and from pregnant women.[2] In these cases the presence of infective virus seemed to result from virus reactivation as shown by the presence of BK virus antibodies before virus excretion. Isolation of BK virus has not been reported during the primary infection of immunologically competent individuals even though antibody surveys have shown a 60-80% prevalence of BK virus antibodies in adults. Antibodies to BK virus usually occur in early childhood.[3] Serological studies have suggested a possible association between primary infection with BK virus and acute upper respiratory tract disease.[4 5] We report the isolation of a human papovavirus from urine of an immunologically competent child with acute tonsillitis.

Case report

On 16 October 1980 a 2-year-old mentally retarded boy was admitted to the department of paediatrics of the university clinic with dyspnoea and high fever. Physical examination showed nasal discharge, swollen inflamed tonsils, enlarged cervical lymph nodes, and conjunctival irritation. His medical history included several hospital admissions for acute respiratory and gastrointestinal illnesses. Laboratory investigations showed normal biochemical values for blood and urine, normal serum immunoglobulin concentrations, and normal lymphocyte functions. Throat cultures were negative for haemolytic streptococci. Bacterial cultures of urine and faeces showed normal flora. Within 11 days he had recovered from the tonsillitis, and he left the hospital on 27 October in good health. On the third day of the illness a throat swab and urine sample were collected for virological examination and inoculated into cultures of primary thyroid cells.

After 28 days urine cultures showed cytopathic changes characterised by cytoplasmic vacuolisation. Electron microscopy of the supernatant showed icosahedral virus particles with a diameter of about 45 nm. Supernatants from the urine cultures agglutinated human type O erythrocytes and the agglutination could be inhibited by a specific anti-BK virus serum. No cytopathic effect was observed in control cells and throat swab cultures. Seroconversion against BK virus between the third and 14th day of the disease (haemagglutination inhibiting antibody titres 1:8-1:256) was shown. Similar titres were obtained when the patient's virus was used as the antigen. No appreciable rises in antibodies to adenovirus, respiratory syncytial virus, influenza virus types A and B, *Mycoplasma pneumoniae*, and *Chlamydia psittaci* were observed as measured by the complement fixation test. Analysis of the viral DNA by cleavage with several restriction endonucleases showed a pattern compatible with BK virus-like human papovaviruses.

Comment

This report describes seroconversion against BK virus and subsequent isolation of virus from the urine of a child with acute tonsillitis. The child's humoral and cellular immunity was not impaired and no immunosuppressive treatment was given. Our findings should encourage further attempts to isolate BK virus from patients with acute respiratory disease.

[1] Gardner SD, Field AM, Coleman WV, Hulme B. New human papovavirus (BK) isolated from urine after renal transplantation. *Lancet* 1971;i:1253-7.

[2] Coleman DV, Daniel RA, Gardner SD, Field AM, Gibson PE. Polyoma virus in urine during pregnancy. *Lancet* 1977;ii:709-10.

[3] Gardner SD. Prevalence in England of antibody to human polyomavirus (BK). *Br Med J* 1973;i:77-8.

[4] Mäntyjärvi RA, Meurman OH, Vihma L, Berglund B. A human papovavirus (BK), biological properties and seroepidemiology. *Ann Clin Res* 1973;5:283-7.

[5] Van der Noordaa J, Wertheim-van Dillen P. Rise in antibodies to human papovavirus BK and clinical disease. *Br Med J* 1977;i:1471.

(*Accepted 31 July 1981*)

Laboratorium voor de Gezondheidsleer, University of Amsterdam, Mauritskade 57, 1092 AD Amsterdam, The Netherlands

J GOUDSMIT, MD, research associate
M L BAAK, technician
K W SLATERUS, MD, clinical virologist
J VAN DER NOORDAA, MD, professor of virology

Prognosis of isolated seizures in adult life

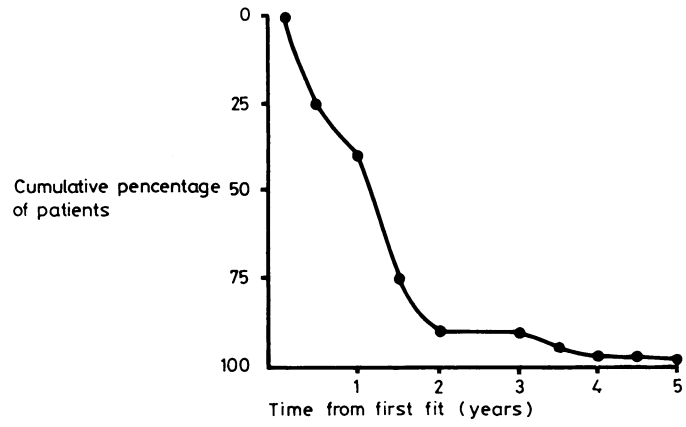
An isolated epileptic fit is a matter of concern to the patient, his family, and his doctor. While the problem for the doctor is to determine any underlying cause, the patient is naturally anxious about further fits. We report here our findings on the follow-up of a group of 70 adults who suffered a single fit.

Subjects, methods, and results

We analysed the notes of all patients aged 16 to 65 with a single blackout referred to the Regional Neurological Centre, Newcastle General Hospital. Only those referred before 1978 and who had suffered a definite witnessed major convulsion were included in the study. Patients whose convulsion had been associated with head injury or drug overdose or who had been started on anticonvulsants were excluded. The suitable patients were contacted and their notes analysed for prognostic indicators.

Eighty-four patients fulfilled the criteria, of whom 14 could not be contacted. Of the remaining 70, 30 were women and 40 men with a mean age of 36. The mean time between the fit and the outpatient visit was six and a half weeks (range two to 10 weeks but 80% were between five to seven weeks) and the mean follow-up period was 4 years 9 months (range three to 10 years). Twenty-seven (39%) patients subsequently developed epilepsy while 43 (61%) had no further fits. The time intervals between the first and second fits are shown in the figure.

There was no difference between those who had single and recurrent fits in age, sex, or period of follow-up. In three (12%) of the former and nine (22%) of the latter group there were possible precipitating factors and these were the same for the two groups—namely, exertion, infection, alcohol, lack of sleep, and anaesthesia. None of the patients had a family history of epilepsy or any abnormal neurological signs. Thirty-nine per cent (15 out of 38) of those who had single fits and 68% (17 out of 25) of those who had recurrent fits had abnormal electroencephalograms (EEGs). This difference was significant (χ^2; 1 df = 4·91; $p < 0·05$). The abnormalities were the same in the two groups and were either non-specific abnormalities, focal slowing over the temporal lobe, or generalised slow activity. One patient, aged 20, who had a second fit two months after the first was subsequently found to have a cerebral tumour (grade II astrocytoma).



Cumulative percentage of 27 patients who developed epilepsy and time between first and second fits.

Comment

The development of epilepsy in adult life is a sinister event, for about 10% of such patients will turn out to have an intracranial tumour.[1] On the other hand, a single fit is usually regarded as a one-off occurrence, not warranting concern or investigation.[2] Little has been written on the prognosis of patients presenting with an isolated fit. Clearly all first fits are "isolated" until a second one occurs and then the patient is, by definition, suffering from epilepsy. We thought that a convenient time to label a fit as isolated was at the outpatient visit, about six weeks after the event.

This study, broadly confirming other reports,[3,4] shows that 39% of patients with an isolated fit developed epilepsy after a mean follow-up period of 4 years 9 months. Moreover, significantly more of this group had an abnormal EEG at the time of their first fit. Thus, though a single EEG recording is of limited value in diagnosing epilepsy, it is a useful indicator as to the likelihood of further fits.

An interesting finding is that the risk of further fits remains high for two years after an isolated fit. Thus the chances against a second fit six weeks, six months, one year, and two years after an isolated fit are respectively 2·6:1, 3·2:1, 4·1:1, and 15:1. Given an incidence[5] of 20 per 100 000, the annual chance of an adult having a fit is one in 5000.

We believe that an adult with an isolated fit should be investigated in the same way as an adult with epilepsy of recent onset. Though six months is a reasonable time to withhold a driver's licence, the patient should be warned that the risk of further fits remains high for another 18 months.

Dr I Mosquera is supported by Fundayacucho, Venezuelan Scholarship. Requests for reprints should be addressed to Dr P G Cleland, Department of Neurology, Newcastle General Hospital, Westgate Road, Newcastle upon Tyne NE4 6BE.

[1] Raynor RB, Paine RS, Carmichael EA. Epilepsy of late onset. *Neurology (Minneapolis)* 1959;**9**:111-7.

[2] Williams D. The border land of epilepsy revisited. *Brain* 1975;**98**:1-12.

[3] Johnson LC, DeBolt WL, Long MT, *et al.* Diagnostic factors in adult males following initial seizures. *Arch Neurol* 1972;**27**:193-7.

[4] Saunders M, Marshall C. Isolated seizures: an EEG and clinical assessment. *Epilepsia* 1975;**16**:731-3.

[5] Hauser BH, Durland LT. The epidemiology of epilepsy in Rochester, Minnesota, 1935 through 1967. *Epilepsia* 1975;**16(1)**:1-67.

(*Accepted 27 July 1981*)

Regional Neurological Centre, Newcastle General Hospital, Newcastle upon Tyne NE4 6BE

P G CLELAND, MRCP, senior registrar in neurology
I MOSQUERA, MD, honorary research fellow in neurology
W P STEWARD, MRCP, senior house officer
J B FOSTER, MD, FRCP, consultant neurologist