Genitourinary tract infections in pregnancy and low birth weight: case-control study in Australian Aboriginal women

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Abstract

Objective—**To investigate the association between genital and urinary tract infections in pregnant Aboriginal women and low birth weight.**

Design—**Retrospective case-control study controlling for potential confounding variables.**

Setting—**Western Australia from 1985 to 1987.**

Subjects—**All Aboriginal women (n=269) who had given birth to singleton infants weighing 2250 g or less (cases), and 269 randomly selected Aboriginal women who had given birth to singleton infants weighing 3000 g or more (controls).**

Main outcome measures—**Proportions of women in case and control groups who had had genital and urinary tract infections; odds ratios for low birth weight when genitourinary tract infection was present; population attributable fraction of low birth weight to genitourinary tract infection.**

Results—**At the time of delivery 51% of women in the case group (109/215) had genitourinary tract infections compared with 13% of controls (35/266). After controlling for potential confounding variables the odds ratio for giving birth to infants weighing 2250 g or less when genitourinary tract infection was present was 4·0 (95% confidence interval 2·3 to 7·0). The proportion of infants with low birth weight attributable to genitourinary tract infection in the whole population of Aboriginal women was 32% (95% confidence interval 17% to 49%).**

Conclusions—**There was a strong association between low birth weight and the presence of genitourinary tract infections in Aboriginal women both during pregnancy and at the time of delivery. A community intervention trial of screening and treatment of genitourinary infections in this population is recommended.**

Introduction

The poor state of health of Australian Aborigines has been well documented,[1,2] and perinatal health in particular is worse in Aborigines than in non-Aboriginal Australians.[3] Although only about 1% of the population of Australia is Aboriginal, 2% of live births are to Aborigines and 7%, 4%, and 8% of stillbirths, neonatal deaths, and postneonatal deaths respectively occur in Aborigines.[3] Preterm birth is thought to be an important factor in Aboriginal perinatal morbidity and mortality, some 16% of Aboriginal infants born in Western Australia being preterm during 1980-6 compared with 7% of all Western Australian infants.[4] Recent research has focused on maternal genitourinary tract infections as potentially treatable causes of preterm birth.[5]

Aboriginal women in Western Australia have a high prevalence of many established risk factors for preterm birth and high rates of genitourinary tract infections. Evidence of an association between genitourinary tract infections and preterm birth in Aboriginal women was suggested in a study which found that the regions of Western Australia with the highest proportions of preterm births to Aboriginal women were those in which the reported prevalence of genitourinary infections during pregnancy was also highest.[6]

This study was designed to investigate whether genital and urinary tract infections in pregnant Aboriginal women in Western Australia were associated with preterm birth, low birth weight (2250 g or less) being used as a measure of preterm birth.

Subjects and methods

The Western Australian maternal and child health research database was used as a sampling frame. This collection of data is based on the Western Australian midwives' notification of case attended form 2, which is a statutory document completed by the attending midwife on all deliveries of liveborn and stillborn infants in the state with a gestational age of 20 weeks or more or a birth weight of at least 400 g. Demographic, medical, obstetric, and perinatal information is recorded for each birth, and about 99% of the 25 000 births each year in Western Australia are recorded in this system.[7] The notification system was validated in 1986[8] and found to be accurate for most of the variables used in this study. Mother's race is determined by the midwife by prior knowledge, observation, or questioning. No distinction is made regarding degree of Aboriginality.

Ideally the case group would have consisted of mothers of all Aboriginal infants born before 37 weeks' gestation (preterm infants) and the control group of a matched random selection of mothers of Aboriginal infants born at 37 or more weeks' gestation (term infants). However, as data on gestational age for Aboriginal infants are unreliable and often unavailable[6,9] it was necessary to infer maturity from birth weight. Thus cases were all Aboriginal women who had given birth to singleton infants weighing 2250 g or less in Western Australia from 1985 to 1987 (n=269). We decided on three years of data as power calculations showed that this would be sufficient (80% power) to detect a 20% difference in the prevalence of infection between the two groups at the 5% level of significance.

The accepted international definition of low birth weight is less than 2500 g.[10] However, the difference in mean birth weight between infants born to white women and those born to Aboriginal women in Western Australia during the study years was roughly 250 g (maternal and child health research database, unpublished data). Thus a birth weight of 2250 g or less was chosen in an attempt to ensure that most births in this group were actually preterm.

Controls were randomly selected from all Aboriginal women who had given birth to singleton infants weighing 3000 g or more from 1985 to 1987. This birth weight was chosen in an attempt to ensure that all the births in the group were full term. Controls were frequency matched[11] with cases for year of birth and gender of infant.

A standard data collection sheet was used for collecting information from the hospitals where births occurred. Data for most of the pregnancies were collected by visiting hospitals (243 cases, 238 controls),

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but for rural hospitals with fewer than five births in the study (26 cases, 31 controls) data were collected by mail. Data included information on genital and urinary tract infections during pregnancy and at delivery, other medical and obstetric conditions, previous obstetric history, smoking and alcohol consumption, and social and demographic variables. Hospital records, including paediatric assessments, were scrutinised to obtain the best estimate of gestational age for all infants, including previous births.

DEFINITION OF INFECTION

The doctors' diagnoses, as recorded in the notes, were used for all medical and obstetric conditions. For every genital tract, urinary tract, and other infection information was recorded about the site of the infection, the method and date of diagnosis, any organism(s) cultured or otherwise identified, whether and how treatment was given, and the results of any follow up tests. When no laboratory tests had been performed the method of diagnosis was recorded as "clinical signs." The coding of genitourinary tract infection as being present at the time of delivery was based on an incomplete course of treatment or a diagnosis made within one week of delivery for which no treatment was given.

We recognised that identifying an organism does not necessarily implicate that organism in infection, that infection may be present when no organism can be identified, and that infections may go unrecorded in pregnancy. However, as the above method is widely used in the diagnosis and treatment of infections in clinical practice we considered that it was appropriate to define infection in this way.

Data from hospital records, particularly those concerning tobacco and alcohol use, were supplemented by collecting information from antenatal records held by Aboriginal Medical Services. These organisations aim at providing culturally appropriate primary and preventive health care for Aboriginal people.[12]

Hospital and Aboriginal medical service data were then merged with data from the maternal and child health research database. Before analysis infants who were recorded as having birth weights of less than 500 g were excluded (18 cases) as these weights are known to be measured inaccurately and so that results would be comparable with those of other studies.[10] Infants with major congenital malformations (21 cases, three controls) were also excluded as such malformations are known to be an independent cause of low birth weight.[13] These infants were identified from the birth defects registry of Western Australia,[14] which uses multiple sources of ascertainment up to the age of 6 years so that identification is as complete as possible. A further 15 women in the case group had no hospital records, so full data were available for 215 cases and 266 controls. For the logistic regression analyses this sample size of 481 was reduced to 410 because of missing data on maternal height, hypertension, or whether previous infants were preterm.

TABLE I—*Maternal characteristics*

Characteristic	No (%) of cases (n=215)	No (%) of controls (n=266)	
Maternal age (years):			
<20	70 (32·6)	73 (27·4)	
20-24	69 (32·1)	103 (38·7)	
25-29	49 (22·8)	54 (20·3)	$\chi^2=2·96$; df=4; p=0·56
30-34	20 (9·3)	26 (9·8)	
≥35	7 (3·3)	10 (3·8)	
Parity:			
0	67 (31·2)	71 (26·7)	
1	59 (27·4)	68 (25·6)	
2	33 (15·3)	55 (20·7)	
3	22 (10·2)	35 (13·2)	$\chi^2=5·78$; df=5; p=0·33
4	21 (9·8)	17 (6·4)	
≥5	13 (6·0)	20 (7·5)	
Marital status:			
Married/cohabiting	111 (51·6)	145 (54·5)	$\chi^2=0·29$; df=1; p=0·59
Single/divorced/widowed	104 (48·4)	121 (45·5)	
Maternal height (cm):			
<149	3 (1·4)	8 (3·0)	
150-159	81 (37·7)	89 (33·5)	
160-169	88 (40·9)	143 (53·8)	$\chi^2=7·76$; df=3; p=0·05 (known values only)
>169	6 (2·8)	19 (7·1)	
Unknown	37 (17·2)	7 (2·6)	

STATISTICAL METHODS

For categorical variables significant differences in proportions between the cases and controls were identified by χ^2 test or Fisher's exact test when numbers in any cell were less than five. Student's *t* test was used for continuous variables.[15] Multiple logistic regression analysis with the CATMOD procedure in the SAS/PC statistical package[16] was performed to determine the odds ratio for preterm birth when genitourinary tract infection was present at the time of delivery, and 95% confidence intervals were calculated.

Various models were fitted to the data simultaneously controlling for potential confounding factors which included known risk factors for preterm birth, such as adverse obstetric history and hypertension in pregnancy,[17] and factors such as maternal height which had different distributions in the cases and controls. Screening for genitourinary tract infections was included in the model as a potential confounding factor as some women (for example, those with problems in previous pregnancies) are more likely than others to have been screened and thus identified as having infections. The matching variables of gender and year of birth of infant were included in the full regression models and subsequently eliminated as they did not significantly alter the odds ratios. Similarly, a quadratic term for maternal height was included in the models to allow for the possibility of a non-linear relation between maternal height and birth weight, but this also was removed as it did not significantly change the odds ratios.

In an attempt to ascertain the public health significance of genitourinary tract infections with regard to low birth weight in the total Aboriginal population in Western Australia the percentage of preterm births attributable to genitourinary tract infections was calculated from the formula[11]: attributable fraction=$(OR-1)p/[(OR-1)p+1]$, where OR=odds ratio and p=proportion of total population exposed (0·158 (from reference 6)).

Results

Determination of the best estimate of gestational age from hospital records, including early ultrasonography and Ballard assessments[18] when these were available, showed that 86% of infants born to mothers in the case group were judged to be preterm compared with 2% of infants born to controls. Hence our choice of birthweight criteria for selecting women having preterm births as cases and term births as controls was considered satisfactory.

Maternal characteristics of the cases and controls are shown in table I. There were no significant differences between the groups in maternal age, parity, or marital status, but the women in the case group were significantly shorter than controls (difference in mean height 1·2 cm; $p<0·05$).

Data on smoking were available for only 146 (68%) of women in the case group and 161 (61%) women in the control group. Among this subsample there was no significant difference in smoking habits between the two groups, 96 (66%) women in the case group and 93 (58%) controls being smokers. Alcohol use was significantly better documented for women in the case group than the control group ($p<0·05$), data being available for 108 (50%) women in the case group and 96

TABLE II—*Complications of pregnancy (some women had more than one complication)*

Complication	No (%) of cases (n=215)	No (%) of controls (n=266)	p Value
Anaemia*	85 (45·7)	90 (45·9)	0·97
Antepartum haemorrhage	39 (18·1)	21 (7·9)	<0·01
Diabetes†	9 (4·6)	11 (4·5)	0·97
Hypertension‡	52 (24·5)	36 (13·7)	<0·01
Prolonged premature rupture of membranes (longer than 24 hours before delivery)	45 (20·9)	7 (2·6)	<0·001
Threatened spontaneous abortion	11 (5·1)	1 (0·4)	<0·01

*Data missing for 29 cases and 70 controls.
†Data missing for 20 cases and 24 controls.
‡Data missing for three cases and three controls.

TABLE III—*Genital and urinary tract infections diagnosed on separate occasions during pregnancy*

No and site of infections	No (%) of cases (n=215)	No (%) of controls (n=266)	
Genital tract infections:			
0	100 (46·5)	176 (66·2)	χ^2 for trend=16·38; df=1; p<0·001
1	85 (39·5)	68 (25·6)	
≥2	30 (14·0)	22 (8·3)	
Urinary tract infections:			
0	141 (65·6)	205 (77·1)	χ^2 for trend=6·08; df=1; p<0·01
1	55 (25·6)	44 (16·5)	
≥2	19 (8·8)	17 (6·4)	

TABLE IV—*Prevalence of genital and urinary tract infections during pregnancy and at delivery*

	No (%) of cases (n=215)	No (%) of controls (n=266)	
Infections present during pregnancy:			
None	69 (32·1)	150 (56·4)	χ^2=28·4; df=3; p<0·001
Genital tract infection	72 (33·5)	55 (20·7)	
Urinary tract infection	31 (14·4)	26 (9·8)	
Both infections	43 (20·0)	35 (13·2)	
Infections present at delivery:			
None	106 (49·3)	231 (86·8)	χ^2=79·94; df=3; p<0·001
Genital tract infection	69 (32·1)	23 (8·6)	
Urinary tract infection	27 (12·6)	8 (3·0)	
Both infections	13 (6·0)	4 (1·5)	

(36%) women in the control group. For the women for whom data were available significantly more women in the case group (43; 40%) than in the control group (23; 24%) reported any alcohol consumption (p<0·05).

Many different medical conditions were recorded, including asthma, epilepsy, thromboembolism, and rheumatic heart disease, but the recorded prevalences of individual conditions were too small for analysis. A higher proportion of women in the case group than the control group had histories of preterm birth and spontaneous abortion. With regard to multiparous women data on previous births were unavailable for 12 cases and 12 controls, and for multigravid women data on previous pregnancies were unavailable for three cases and one control. Of the multiparous cases in which there were available data, 67 out of 133 (50%) women had had a previous preterm birth compared with 23 out of 183 controls (13%) (p<0·001); 51 out of 151 (34%) multigravid women among the cases had had a spontaneous abortion compared with 40 out of 198 (20%) controls (p<0·005). More women among the cases than among the controls had hypertension, including both pregnancy induced and essential conditions; premature rupture of the membranes; antepartum haemorrhage; and threatened spontaneous abortion. Diabetes and anaemia, when recorded, were of a similar high prevalence in both groups of women (table II).

The most striking differences between the groups were in genital and urinary tract infections. A significantly higher proportion of women among the cases than among the controls had been diagnosed as having genital or urinary tract infections, or both, during pregnancy (146 (68%) and 116 (44%) respectively), and the women in the case group had had a significantly higher number of infections than the controls (tables III and IV). At the time of delivery 109 (51%) women in the case group had a genitourinary tract infection compared with 35 (13%) controls (table IV). There was no difference in the incidence of other infections between the two groups.

The organisms responsible for colonisation and infection of the genital and urinary tracts at the time of delivery are shown in table V. *Trichomonas vaginalis*; vaginosis with any of *Gardnerella vaginalis*, bacteroides species, or anaerobic streptococci; and group B streptococcus were significantly more common in the genital tract of women in the case group (p<0·05). *Escherichia coli* was the only individual urinary tract pathogen significantly more commonly cultured from urine samples of women in the case group (p<0·05).

Table VI shows the odds ratios for low birth weight among women with complications of pregnancy and other confounding variables when maternal height and age were controlled for. The odds ratio for low birth weight when genitourinary tract infection was present at delivery was 3·99 (95% confidence interval 2·28 to 6·99) after controlling for hypertension, antepartum haemorrhage, prolonged premature rupture of the membranes, threatened spontaneous abortion, adverse obstetric history, and screening for infection.

The attributable fraction of low birth weight to genitourinary tract infection in the Aboriginal population was calculated to be 32·1% (95% confidence interval 16·8% to 48·6%).

It has been suggested that threatened spontaneous abortion and prolonged premature rupture of the

TABLE V—*Genital tract colonisation and bacteriuria: organisms reported at delivery (in many women colonisation was polymicrobial)*

Site and organism	No (%) of cases (n=215)	No (%) of controls (n=266)	p Value
Genital tract colonisation:			
Candida albicans	17 (7·9)	15 (5·6)	0·42
Trichomonas vaginalis	17 (7·9)	3 (1·1)	<0·001
Vaginosis with any of *Gardnerella vaginalis*, *Bacteroides* sp, or anaerobic streptococci	13 (6·0)	3 (1·1)	<0·01
Group B streptococcus	12 (5·6)	4 (1·5)	<0·05
Neisseria gonorrhoeae	3 (1·4)	2 (0·8)	0·40
Calymmatobacterium granulomatis	1 (0·5)	1 (0·4)	0·69
Staphylococcus aureus	2 (0·9)	0	0·20
Others*	7 (3·3)	1 (0·4)	<0·05
Unspecified or unknown organisms†	18 (8·4)	2 (0·8)	<0·001
Bacteriuria:			
Escherichia coli	14 (6·5)	6 (2·3)	<0·05
Gram positive bacilli	2 (0·9)	0	0·12
Gram negative bacilli	3 (1·4)	0	0·08
Klebsiella sp	2 (0·9)	2 (0·8)	0·60
Others‡	4 (1·9)	0	<0·05
Unspecified or unknown organisms†	15 (7·0)	5 (1·9)	<0·05

*Others were all those detected once only—namely, *Clostridium perfringens*, *Klebsiella* sp, human papillomavirus, *Treponema pallidum*, *Mycoplasma hominis*, herpes simplex virus, *Torulopsis glabrata*, *Staphylococcus epidermidis*.
†Includes all organisms detected in only one sample—namely, unclassified streptococci, Gram positive cocci, *Enterobacter* sp, *Proteus* sp.
‡Includes culture negative and uncultured urinary tract infections diagnosed by signs and symptoms.

TABLE VI—*Logistic regression of genitourinary tract infection and potential confounding variables on low birth weight (based on n=410). (Results controlled for maternal height and age)*

Variable	Odds ratio	95% Confidence interval
Hypertension	2·23	1·22 to 4·07
Antepartum haemorrhage	3·04	1·37 to 5·54
Prolonged premature rupture of membranes	7·36	2·52 to 21·5
Threatened spontaneous abortion	14·63	1·55 to 137·9
Adverse obstetric history (previous preterm birth or spontaneous abortion)	3·66	2·13 to 6·30
Screening for infection (urine sample, vaginal swab, or both)	2·24	1·16 to 4·34
Genitourinary tract infection at delivery	3·99	2·28 to 6·99

TABLE VII—*Logistic regression of genitourinary tract infection and potential confounding variables on low birth weight excluding possible early manifestations of preterm birth (prolonged premature rupture of membranes and threatened spontaneous abortion) (based on n=410). (Results controlled for maternal height and age)*

Variable	Odds ratio	95% Confidence interval
Hypertension	1·88	1·03 to 3·43
Antepartum haemorrhage	3·53	1·63 to 7·66
Adverse obstetric history (previous preterm birth or spontaneous abortion)	3·69	2·18 to 6·23
Screening for infection (urine sample, vaginal swab, or both)	2·35	1·23 to 4·50
Genitourinary tract infection at delivery	4·83	2·83 to 8·24

membranes may be early manifestations of preterm birth[19-21] and hence outcomes rather than confounding variables. Logistic regression analysis excluding these variables (table VII) showed a higher point estimate for the odds ratio of low birth weight with genitourinary tract infection at delivery of 4·83 (95% confidence interval 2·83 to 8·24). When threatened spontaneous abortion and premature rupture of the membranes were excluded as separate risk factors then the fraction of infants with low birth weight attributable to infections was 37·7% (95% confidence interval 22·4% to 53·4%).

Logistic regression analysis was also performed for the subsample of women who had had spontaneous labour (n=304) as women with induced labour or no labour may have had infants with low birth weight solely because of the induction or caesarean section. Infections remained a significant predictor of low birth weight with an odds ratio of 4·64 (95% confidence interval 2·31 to 8·61).

Discussion

Established risk factors for preterm birth include extremes of maternal age, small stature, maternal smoking, unmarried status, low socioeconomic status, hypertension, previous preterm birth, and spontaneous abortion.[17] Many of these factors cannot be altered after conception and others may be markers for a predisposition to preterm birth rather than causative factors. However, 60% of preterm births remain unexplained by these risk factors and efforts to identify and treat women at high risk of preterm birth have not significantly changed the proportion of women giving birth before term.[21] In our study genitourinary tract infections were significant predictors of low birth weight—and, by implication, preterm birth—after controlling for many of these factors.

Our study showed a strong association between low birth weight and maternal genitourinary tract infections both during pregnancy and at delivery. This association remained when controlling for potential demographic, obstetric, and selective screening confounding factors. Infections were stronger predictors of low birth weight than any demographic or social factors measured, adverse obstetric history, and complications of pregnancy other than threatened spontaneous abortion and prolonged premature rupture of the membranes. Although socioeconomic status is difficult to measure, maternal height, which may be an indicator of this variable,[22] was controlled for throughout.

When women who had had induced labours were excluded from the analysis infections remained a significant predictor of low birth weight, with an odds ratio higher than that for the total group. When threatened spontaneous abortion and prolonged premature rupture of the membranes were excluded as confounders from the analysis genitourinary tract infections became the strongest predictor of low birth weight of all the variables considered. These results support those of other studies which have shown a clear causative link between genitourinary tract infections and low birth weight or preterm birth.[23-26]

Cultures of *T vaginalis*, organisms of bacterial vaginosis, and group B streptococcus were significantly more frequent from women in the case group. These conditions have all been associated with preterm delivery and low birth weight in previous studies and share the characteristic of being sexually transmissible.[21 23 27]

The high proportion of women in the case group with previous preterm deliveries or spontaneous abortions suggests an aetiological agent which could be recurrent or persistent, as is frequently the case with genital and urinary tract infections. This finding supports other studies which have shown that infectious organisms can cause spontaneous abortion by similar mechanisms to those which cause preterm labour.[28]

In this sample of Aboriginal women treatment and follow up of infections was inadequate. Further research to determine the specific role of genitourinary tract infections in low birth weight and preterm birth in Aboriginal women is important if effective preventive measures are to be instituted. Clinical trials of screening for genitourinary infections and giving antibiotics in pregnancy would show whether there is a causal and preventable relation between infections and preterm birth in this population. The prevention and treatment of genitourinary tract infections during pregnancy have the potential to reduce the prevalence of low birth weight and preterm birth and to have an important impact on Aboriginal maternal, infant, and child health.

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Risk of epilepsy after febrile convulsions: a national cohort study

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Abstract

***Objective*—To identify children with febrile convulsions, classify their febrile convulsions into simple and complex, and determine the number and type of subsequent afebrile seizures in those children.**

***Design*—National population based study.**

***Setting*—United Kingdom.**

***Subjects*—16 004 neonatal survivors born during one week in April 1970.**

***Main outcome measures*—Information about febrile and afebrile seizures obtained from questionnaires at 5 and 10 years of age and from hospital records.**

***Results*—Information was available for 14 676 of the cohort children. 398 (2·7%) of them had had at least one febrile convulsion. 16 children were known to be neurologically or developmentally abnormal before the first attack. Of the remaining 382 children, 305 had had a simple first febrile convulsion and 77 a complex first febrile convulsion. Thirteen of the 382 had had one or more afebrile seizures, nine of whom had developed epilepsy (recurrent afebrile seizures). A higher proportion of children with complex febrile convulsions (6/95) rather than simple febrile convulsions (3/287) developed epilepsy, the risk being highest for those who had had focal febrile convulsions (5/17; $\chi^2=39{\cdot}9$, $p<0{\cdot}001$). Three of the 32 children who had prolonged febrile convulsions developed afebrile complex partial seizures.**

***Conclusions*—The risk of epilepsy after febrile convulsions is much less than reported in many hospital studies, and if febrile convulsions cause brain damage that leads to later epilepsy this is a rare occurrence.**

Definitions of convulsions

Febrile convulsion—An event in infancy or childhood associated with fever but without evidence of intracranial infection or defined cause. This is similar to the National Institutes of Health definition.[7] Children with previous afebrile seizures were excluded. Suspected seizures in the first four weeks of life were excluded but convulsions during vaccination fevers were included.

Complex febrile convulsion—Longer than 15 minutes, focal, or multiple (more than one convulsion per episode of fever).

Simple febrile convulsion—Not complex.

Recurrent febrile convulsion—More than one episode of fever associated with convulsions.

Afebrile convulsion—Classification based on proposals of the International League Against Epilepsy.[8] Children with more than one afebrile attack defined as having epilepsy.

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Introduction

Hospital based studies have reported a high incidence of epilepsy in children after febrile convulsions—up to 40%.[1] In contrast, population based studies suggest that the outcome is better, with incidences of 2% and 3·5% in two large American studies.[2,3] Febrile convulsions may cause later afebrile seizures or some children may be predisposed to both febrile convulsions and subsequent afebrile seizures.

The child health and education study,[4,5] which began as the British births survey,[6] is one of the few cohort studies large enough to study outcome after febrile convulsions without the bias inherent in hospital based studies. We report the risk of afebrile seizures and epilepsy after febrile convulsions using data obtained when the cohort of children were aged 10.

Subjects and methods

The British birth survey enrolled 16 004 neonatal survivors—98·5% of the infants born in the United Kingdom in one week in April 1970. Of these children, 13 135 (82%) were assessed at 5 years of age and 14 902 (93%) at 10 years. At the assessments parents were asked: "Has the child ever had any form of convulsion, fit, seizure or other turn in which consciousness was lost or any part of the body made an abnormal movement?" After the 10 years assessment replies to this question were available for 14 676 children, of whom 1318 were judged to have had a suspicious event. Reported suspicious events were validated by sending questionnaires to general practitioners and by obtaining hospital records. Questionnaires were returned for 1212 (92%) children and records obtained for 1173 (89%). Children with confirmed febrile convulsions were classified according to the type of convulsion (box). Comparisons between groups were analysed by the χ^2 test with Yates's correction.

Results

AGE OF ONSET, INCIDENCE, AND RECURRENCE OF FEBRILE CONVULSIONS

Of the 1318 children who had had suspicious events, 398 had had at least one febrile convulsion by 10 years of age (median (range) age of onset 1 year 7 months (2 months—7 years 9 months)). The remaining children had had other attacks such as breath holding attacks and afebrile seizures and will be discussed elsewhere. Two children who had had seizures and