Relation between birth weight at term and systolic blood pressure in adolescence

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Abstract

Objective—To examine whether birth weight is related to systolic blood pressure during adolescence.

Design—Retrospective (comparative) cohort study. The observers who traced and studied the subjects were unaware of their case-control status.

Subjects—330 subjects were born in Cardiff in 1975-7. Cases who were low birth weight at term (<2500 g) were matched with controls of normal birth weight (3000-3800 g) at term.

Main outcome measures—Systolic blood pressure measured by random zero sphygmomanometry in the subject's right arm with the subject supine, corrected for size and age.

Results—The mean age at examination was 15.7 years. The mean systolic blood pressure of the cases was 105.8 mm Hg and of the controls 107.5 mm Hg. The corrected difference (95% confidence interval) in systolic blood pressure between the cases and controls was 1 mm Hg (-3 to +1 mm Hg; two tailed probability 0.33).

Conclusion—Systolic blood pressure in adolescents of low birth weight is not significantly different from that of adolescents of normal birth weights.

Introduction

Hypertension is a well accepted risk factor for ischaemic heart disease and stroke. To the known risk factors for hypertension of obesity,^{1,2} lifestyle,^{3,4} and increased salt intake⁵ has recently been added low growth rate in utero with its proxy of low birth weight at term.^{6,9} The hypothesis advanced is that programming of physiological or metabolic events caused by undernutrition¹⁰ at critical or sensitive periods early in life can amplify certain long term changes which eventually manifest in disease, in this case hypertension and ischaemic heart disease. Evidence for this hypothesis comes largely from studies of cohorts born in Britain earlier this century, most of whom were men.^{6,9} A criticism of these studies is that people whose growth has been affected in early development may continue to have imposed during childhood and adult life a hostile environment that leads eventually to disease.¹¹ The confounding influence of these external environmental effects may be evident in advance of the consequences of the intrauterine environment. Also in some circumstances the extrauterine effects may be overwhelming.

We aimed to minimise the effects of different extrauterine environments by investigating the association between low birth weight at term with systolic blood pressure in schoolchildren.

Subjects and methods

STUDY DESIGN

This was a retrospective cohort study in which the subjects were traced and assessed by observers who were unaware of whether they were cases or controls. A strict protocol was drawn up for the identification and selection of subjects, methods of clinical examination, and statistical analysis. Ethical approval was granted

by the South Glamorgan Health Authority and the local family health services authority.

Tracking of systolic blood pressure is well documented throughout childhood and adult life.¹² Raised blood pressure may be amplified throughout life so that any differences associated with low birth weight may become larger with advancing age.¹³ If very young children were studied tracing might be easier as few would have changed address, but any differences in blood pressure due to prenatal experiences might be small. Older children would be more difficult to find, but the differences in blood pressure might be larger. Children in school are a fairly easy group to locate with the availability of computerised school health records, and they are a homogeneous group whose environment is similar for part of the day so that some confounding variables (which might begin to impose themselves when the subject leaves school—for example, occupation) would be lessened.

Cases and controls were identified from the Cardiff birth survey. Every birth in Cardiff and later in South Glamorgan has been documented since 1965. Cases were defined as infants with a birth weight under 2500 g at 38 weeks' gestation or more, a group likely to have suffered retardation of intrauterine growth. Unlike low birth weight and preterm delivery there is no internationally agreed definition of fetal growth retardation. Controls were infants born at 38 weeks' gestation or more and weighing 3000-3800 g. These infants were judged to have grown adequately in utero.

Multiple pregnancies and infants with congenital abnormalities were excluded, as were deaths and medical conditions that might influence systolic blood pressure. Only births at the two large obstetric departments were used as the small numbers born elsewhere would not have sufficient well matched controls. There were 8920 relevant deliveries during the study.

The initial variables used to calculate sample size were $\alpha=0.05$ and $\beta=0.10$, and the smallest difference in systolic blood pressure that could be viewed as clinically important was 4 mm Hg. No estimate of the standard deviation of the differences in systolic blood pressure between cases and controls matched as described was available. The estimated standard deviation of systolic blood pressure for 15 year olds was 12 mm Hg,¹⁴ giving a standardised difference of $4/12=0.33$. Without matched data a sample size of between 173 and 234 in each group at analysis would be required. The data from the birth survey showed that the academic year cohorts from 1 September 1975 to 30 August 1977 would yield 230 potential cases. At the start of the study (March 1991) all were expected to be receiving compulsory full time education. If the matching were to reduce the variance then a greater power would ensue.¹⁵

MATCHING OF CASES AND CONTROLS

For each index case a list of eligible controls was drawn up with a computer. Cases and controls were matched for sex, hospital of delivery, parity of the mother, date of birth, and length of gestation. Length of gestation was calculated from the mother's last menstrual period as ultrasound scanning was not

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routinely available. If there was doubt regarding length of gestation a Dubowitz assessment of the neonate was performed. Prior rules for selection of controls were applied in sequence until a match was found. These rules were designed so that if any bias was introduced by matching it would result in the cases having a higher systolic blood pressure than the controls, which is in favour of the alternative hypothesis.

DATA COLLECTED

Data on the mother collected at delivery included social, lifestyle, and clinical variables associated with the current pregnancy and relevant medical history. Data on the infant included details of the delivery and relevant clinical variables. The following multiple sources were used to trace the subjects: computerised school records, South Glamorgan family health services authority, NHS central register (including armed forces), telephone directory, South Glamorgan Social Services, hospital patient administration system, general practitioners, door to door inquiries, hospital records at the time of delivery (which occasionally contained telephone numbers of relatives), and the medical directory.

The subjects who agreed to participate were visited at home by one of two observers who had been trained in the techniques of anthropometry and measurement of blood pressure.

Weight was measured to the nearest 100 g with the subject lightly clothed and without shoes by using a portable Soehnle scale. Height was measured by using a portable Harpenden stadiometer to the nearest 1 mm. Systolic blood pressure was measured with a random zero sphygmomanometer by following recommendations of the British Hypertension Society.¹⁶

ANALYSIS

The statistical package SPSS/PC was used for analysis.¹⁷ Means were compared by using a paired *t* test. The effect on systolic blood pressure of sex was examined by using one way analysis of variance, and the effect of birth weight, maternal height, and gestation was determined by using the non-parametric Kruskal-Wallis test. Results were all rounded after calculation.

Results

Of 230 potential low birthweight cases identified, eight were ineligible (six were dead and two had

TABLE IV—Differences in adjusted systolic blood pressure by sex*

Sex	No of subjects	Mean (SD)	SE	95% Confidence interval
Male	63	-1.4 (11.9)	1.5	-4.4 to 1.6
Female	101	-0.7 (13.6)	1.4	-3.4 to 2.0

*f/Ratio = 0.11, *df* = 1, *P* = 0.73. Tests for equality of variance in two groups gives *P* = 0.25.

medical conditions not recorded at birth (pseudo-hypoparathyroidism and severe cerebral palsy)), leaving a cohort of 222. The records of five were marked as "confidential—not to be used for research studies"; four subjects were living too far from Cardiff to visit, 17 subjects could not be traced at all, and 19 refused to participate. A total of 177 cases were finally studied.

Of the controls for these cases, 75% (133/177) were found. When it was recognised early in the study that a control would be unavailable a subsequent control was drawn. For the 30 identified subsequent controls, 93% (28) were seen. This left 12 cases who were seen but did not have matched controls and could not therefore be used in the paired analysis. In total 74% (165/222) of the case-control pairs were found. The subjects studied were of mean age 15.7 years. In one control the weight had not been recorded.

We did not consider it appropriate to determine the exact stage of puberty during a home visit. Tables I and II show the closeness of the case-control matching. Table III gives a comparison of the cases and controls. There were important differences in age, height, and weight at examination which influence systolic blood pressure. These differences may be adjusted for by fitting a linear regression model to the data.¹⁸ No existing model was suitable so age, height, weight, sex, body mass index (weight (kg)/(height (m)²)), and case-control status were entered into a stepwise multiple regression to produce a linear model for systolic blood pressure. All the available data were used. The best fit to the data was: systolic blood pressure = 24.7 + 0.31 weight (kg) + 4.1 age (years); *r*² = 0.24; SE weight coefficient = 0.05; SE age coefficient = 0.69.

This model was then used to adjust the paired differences in systolic blood pressure. For one subject the weight was not available and so adjusted systolic blood pressure could not be calculated. The mean (SD) difference in adjusted systolic blood pressure for the 164 remaining pairs of subjects was -1.0 (13.0) mm Hg, with a median of 0.35, minimum of -41.4, and maximum of 33.2. The standard error was 1.01 and 95% confidence interval -3.0 to 1.0, giving a two tailed *P* value of 0.33. Table IV gives the non-significant differences in systolic blood pressure by sex. The effect of birth weight of cases (*P* = 0.85), maternal height for cases (*P* = 0.60), and length of gestation (*P* = 0.66) on systolic blood pressure were all found to be non-significant by using the Kruskal-Wallis test, there being no obvious trend in the results.

Discussion

Infants who weighed less than 2500 g at 38 weeks' gestation or more (cases) are considered to have probably suffered retardation of growth in utero. These infants have a lower than average ponderal index because of fetal undernutrition from various causes.¹⁹ The control infants, on the other hand, were considered to have been adequately nourished during gestation.

This is the first reported population based comparative study to investigate the influence of low birth weight at term on later blood pressure. No link could be found between birth weight and blood pressure in adolescence once the differences in size and age between the two groups at examination were con-

TABLE I—Summary of differences in parity of mothers of cases and controls for each pair of subjects

Parity for mothers of cases	Difference in parity (cases minus controls)			
	-2	-1	0	+1
0			82	
1		1	49	1
2			18	1
3	1		6	2
4		1	2	
5			1	
Total	1	2	158	4

TABLE II—Summary of differences in length of gestation between cases and controls for each pair of subjects

Length of gestation (weeks) for cases	Differences in length of gestation (weeks) for cases minus controls		
	-1	0	+1
38	2	41	4
39		40	
40		62	
41		13	
42	1	2	
Total	3	158	4

TABLE III—Comparison between 165 cases and 165 controls. Data on birth weight and maternal height from Cardiff birth survey, other data from examination of adolescents

	Mean	SD	Median	Minimum	Maximum
Birth weight (coded to nearest 100 g):					
Cases	2250		2300	1600	2400
Controls	3330		3300	3000	3800
Maternal height (inches)*:					
Cases	61.9		68.0	57.0	67.0
Controls	62.7		63.0	57.0	67.0
Body mass index (kg/m ²):					
Cases	20.2	3.1	19.9	12.9	29.0
Controls	21.1	3.7	20.7	13.4	35.8
Difference	-0.93	4.9	-0.4	10.6	-18.1
Age at examination (years):					
Cases	15.6	0.77	15.45	14.1	17.3
Controls	15.8	0.84	15.7	14.0	17.8
Difference (days)	-74.6	286	-53	550	-722
Systolic blood pressure					
Cases	105.8	10.2	105	82	136
Controls	109.5	11.5	108	78	144
Difference	-3.8	14.4	-2	32	-48
Weight (kg):					
Cases	52.1	9.0	51.4	33.4	80.0
Controls	58.4	11.7	57.1	33.0	110.6
Difference	-6.3	14.7	-5.5	28.8	-71.8
Height (cm):					
Cases	160.7	7.72	160.9	134.0	185.0
Controls	166.1	7.96	165.1	148.0	185.1
Difference	-5.4	9.6	-5.0	20.2	-233.3

*Data recorded for 161 cases and 161 controls to nearest inch with extreme values coded as ≤ 57" (145 cm) and ≥ 67" (170 cm).

sidered. Other studies have also failed to find an association. In a previous study mean systolic blood pressure of very low birthweight preterm children aged 8 years was 1.8 mmHg lower than that of normal birth weight controls, but no correction was made for body size.²⁰ Cook *et al* found no association between birth weight and blood pressure in first degree relatives (mean age 43 years) of people with non-insulin dependent diabetes mellitus.²¹ A study from New Zealand showed that corrected systolic blood pressure in 7 year olds was 1 mmHg higher in those who had suffered retardation of intrauterine growth compared with those of birth weight between the 10th and 90th centiles, but at the age of 18 years the differences were not significant.²² In nearly 33 000 men and women aged 17 years Seidman *et al* found that systolic blood pressure was positively associated with birth weight, but the correlation coefficients were low; current body weight was significantly associated with systolic blood pressure, suggesting that an adverse extrauterine environment was a much more important determinant of blood pressure than birth weight.¹⁸

Other studies have found associations between birth weight and later blood pressure. Multiple regression analysis of factors affecting systolic blood pressure in Croatian subjects aged 18-23 years showed an inverse relation with birth weight.²³ In 36 year old subjects from the Medical Research Council's 1946 birth cohort systolic blood pressure fell by 2.6 mmHg in men and 1.8 mmHg in women from the highest to the lowest third of the distribution of birth weight.⁷ A study of men and women aged 50 years from Preston found that a higher systolic blood pressure was related to increased placental weight and lower birth weight.⁶ Barker *et al* found a similar relation between low birth weight and increasing systolic blood pressure in men aged 59-70 years in Hertfordshire.⁸ Higher standardised mortality ratios for ischaemic heart disease in men in Sheffield were associated with low birth weight, small head circumferences, and reduced ponderal indices at birth.⁹ Whincup *et al* studied children aged 5-7 years and found a relation between low birth weight and systolic blood pressure corrected for age, sex, height, and body mass index.²⁴ The effect was similar for prematurity and growth retardation, and one suggested explanation was that increased postnatal growth rates might be more important in determining future blood pressure than prenatal growth.

SOURCES OF BIAS

Many of these studies rely on cohorts born a long time ago, when health care practices, environmental conditions, and diet were probably different.⁶⁻¹⁰ Also only the survivors from times when infant mortality was high have been studied. These factors could be a source of bias. Different factors may now predispose to intrauterine growth retardation—for example, maternal smoking. Several groups have questioned whether the links between intrauterine growth retardation and later disease might be explained by a continuum of social and environmental insults throughout life.²⁵⁻²⁸ Our findings of no link at the age of 15 years may be because any possible adverse life style has not yet had sufficient time to exert an effect.

The possibility of a "false negative" result can never be discounted. There are two sources of bias which may have influenced our study: case ascertainment bias and recruitment bias. Case ascertainment bias may lead to important subgroups being underrepresented in the sample. Here we failed to use 57 cases (25%). Control selection and ascertainment bias may similarly lead to underrepresentation of important subgroups. Attempts were made to control these types of potential bias by keeping the field workers blind to subjects' case-control status. Also the controls were selected by

Clinical implications

- An inverse relation between birth weight and cardiovascular disease in adults has been reported in retrospective cohort studies
- One possible mechanism is via a link with raised systolic blood pressure
- No difference was found between the systolic blood pressure of adolescents who as a group were growth retarded at term and a comparative group of normal birth weight
- This study does not lend support to one suggested mechanism linking intrauterine growth retardation and later cardiovascular disease
- Further studies are required on new cohorts to examine the mechanism by which intrauterine growth retardation might be linked to a risk of adult disease

using prior rules which entailed no additional judgment. The strength of the study lies in the fact that it is based on the population of a well defined geographical location, every member of that population being recorded. While the number of cases (165) may seem small compared with other studies with much larger samples the effective population studied here is over 9000 with just the cases of particular interest selected.

CONCLUSIONS

Our results are inconsistent with adolescents born at term with low birth weight having a systolic blood pressure more than 1 mmHg above or more than 3 mmHg below that of controls with normal birth weight. This result lends no support to the hypothesis that low birth weight at term is related to a higher blood pressure during adolescence.

The hypothesis that hypertension may be initiated in fetal life is fascinating and derives additional support from studies on animals. Before it shifts from hypothesis to established fact, however, further large population studies are required to determine any association between birth weight (a proxy for changes in structure, physiology, and metabolism determined in utero) and blood pressure through the whole span of life.

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Are pre-hospital deaths from accidental injury preventable?

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Abstract

Objective—To determine what proportion of pre-hospital deaths from accidental injury—deaths at the scene of the accident and those that occur before the person has reached hospital—are preventable.

Design—Retrospective study of all deaths from accidental injury that occurred between 1 January 1987 and 31 December 1990 and were reported to the coroner.

Setting—North Staffordshire.

Main outcome measures—Injury severity score, probability of survival (probit analysis), and airway obstruction.

Results—There were 152 pre-hospital deaths from accidental injury (110 males and 42 females). In the same period there were 257 deaths in hospital from accidental injury (136 males and 121 females). The average age at death was 41.9 years for those who died before reaching hospital, and their average injury severity score was 29.3. In contrast, those who died in hospital were older and equally likely to be males or females. Important neurological injury occurred in 113 pre-hospital deaths, and evidence of airway obstruction in 59. Eighty six pre-hospital deaths were due to road traffic accidents, and 37 of these were occupants in cars. On the basis of the injury severity score and age, death was found to have been inevitable or highly likely in 92 cases. In the remaining 60 cases death had not been inevitable and airway obstruction was present in up to 51 patients with injuries that they might have survived.

Conclusion—Death was potentially preventable in at least 39% of those who died from accidental injury before they reached hospital. Training in first aid should be available more widely, and particularly to motorists as many pre-hospital deaths that could be prevented are due to road accidents.

Introduction

In 1988 a retrospective study of 1000 deaths from injury in England and Wales showed that many of the deaths in hospital might have been prevented.¹ The authors expressed concern that in up to 20-30% of fatal cases a delay in diagnosis or treatment or an error in both was an important significant factor in the deaths. In response to this study and the recommendations of the Royal College of Surgeons' working party on the management of patients with major injuries² the

Department of Health funded a pilot trauma centre in Stoke on Trent.³ The study by Anderson *et al*,¹ however, was incomplete. Of the 1000 people who died of injury, 486 did so at the scene of the accident or were dead on arrival at hospital. No further information on these deaths was given, and the authors seem to have assumed that these deaths (about half the population studied) were not preventable.

This assumption is not without precedent. In 1977 Yates investigated airway patency in people in fatal accidents.⁴ Airway patency may be compromised by the aspiration of blood or gastric contents when the gag reflex is lost during coma. This life threatening complication can often be prevented by early basic life support. Yates reviewed all deaths reported to the coroner over five years and studied in detail the findings of the necropsy on people who died in an accident. By correlating the severity of the injury with airway obstruction he concluded that airway obstruction contributed to the death of those who died in hospital but not of those who died before reaching hospital. This seems to suggest a limited scope for increasing the survival rate for those who die before reaching hospital.

Yates used the injury severity score to assess injury.⁵ This had been developed by Baker *et al* during a study of 2128 victims of road traffic accidents in Baltimore, United States, from 1968 to 1969, which included those who died before reaching hospital.⁶ In 1975 Bull used the injury severity score to reanalyse the data on 1333 victims of road traffic accidents treated as inpatients at Birmingham Accident Hospital in 1961.⁶ He used probit analysis⁷ to linearise the mortality data in separate age groups and showed a good correlation between the injury severity score and the probability of survival when this North American method was applied to a British population. Yates used Bull's findings to correct the injury severity score for age.⁴ "Bull's probits" have been used by several other authors to identify preventable deaths from injury.⁸⁻¹⁰

In the United States Trunkey identified a trimodal distribution of deaths from injury.¹¹ The first peak occurred immediately after the injury: over half of all deaths due to trauma occurred within seconds or minutes of injury, owing to overwhelming injury to the brain, heart, or great vessels; death was inevitable and unavoidable. The second peak occurred in the first few hours after injury in about a third of cases; brain injuries and haemorrhage were the principal causes.

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