increased since 1988, every year nine in 100 000 children under 5 develop diabetes, each with the daily burden of diabetes management and a risk of early death and subsequent morbidity in adult life.

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Abnormal liver growth in utero and death from coronary heart disease

D J P Barker, C N Martyn, C Osmond, G A Wield

MRC Environmental Epidemiology Unit (University of Southampton), Southampton General Hospital, Southampton SO16 6YD
D J P Barker, *director*
C N Martyn, *clinical scientist*
C Osmond, *statistician*
G A Wield, *systems analyst*

Correspondence to:
Professor Barker.

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Evidence is growing that the metabolic abnormalities which lead to coronary heart disease are programmed by undernutrition in utero. Undernutrition of the fetus leads to small size and disproportionate body form at birth, which are now known to be linked to metabolic abnormalities in later life.[1] Abdominal circumference may be measured routinely at birth and indicates liver growth in utero as well as the fatness of the abdominal wall. A recent study showed that men who had a small abdominal circumference at birth had raised serum concentrations of total and low density lipoprotein cholesterol.[2] They also had raised plasma concentrations of fibrinogen, another major risk factor for coronary heart disease that is regulated by the liver.[3] These associations were independent of social class, current body weight, cigarette smoking, and alcohol consumption. They suggest that impaired liver growth in utero may be an early stage in the pathogenesis of coronary heart disease.

Methods and results

A standardised record form was kept for each woman admitted to the Jessop Maternity Hospital in Sheffield. From 1922 onwards, abdominal circumference was included among the measurements made on the baby at birth. We have traced 1973 (79%) of 2513 singleton boys born alive during 1922-30. The method of follow up has been described.[2,3] The abdominal circumference of 1819 of the 1973 boys had been recorded. A total of 174 of them had died from coronary heart disease (*International Classification of Diseases* (9th revision) codes 4100-4149).

The men's mean birth weight was 7·5 pounds (3400 g) and their mean abdominal circumference was 12·3 inches (31·2 cm). The table shows their death rates from coronary heart disease, expressed as standardised mortality ratios with the average for England and Wales as 100. Men with below average birth weight had higher death rates, as would be expected from previous findings.[1] Mortality ratios showed a U shaped relation with abdominal circumference. Further examination showed that this was the result of opposing trends at birth weights below and above the average. Below the mean birth weight standardised mortality ratios fell with increasing abdominal circumference whereas above it they rose. The difference in these two trends was significant ($\chi^2=8·2$, $P=0·004$ interaction term in log-linear model). P values for the individual trends were 0·11 and 0·02, respectively.

Standardised mortality ratios for coronary heart disease according to abdominal circumference at birth among 1819 men born during 1922-30. Values in parentheses are numbers of deaths unless stated otherwise

Abdominal circumference at birth (inches (cm))	All men			Men born at >37 weeks' gestation		
	Average birth weight or less	Above average birth weight	All	Average birth weight or less	Above average birth weight	All
≤11·5 (29·2)	123 (39)	48 (2)	114 (41)	102 (17)	35 (1)	92 (18)
-12·0 (30·5)	113 (36)	58 (11)	93 (47)	110 (23)	43 (6)	83 (29)
-12·5 (31·8)	79 (12)	102 (14)	90 (26)	73 (7)	95 (9)	84 (16)
-13·0 (33·0)	68 (7)	110 (26)	97 (33)	74 (5)	112 (18)	101 (23)
>13·0 (33·0)	101 (3)	125 (24)	122 (27)	49 (1)	126 (16)	115 (17)
All	106 (97)	97 (77)	101 (174)	95 (53)	91 (50)	93 (103)

Average birth weight = 7·5 lb (3400 g).

The trends with chest circumference were similar, but weaker (χ^2 for difference = 3·3, P = 0·07). There were no significant trends with head circumference or crown-heel length. Opposing trends in mortality from all causes with abdominal circumference corresponded to those in coronary heart disease mortality but were not significant.

Gestation period was known in 1278 (70%) of the men, estimated from the mother's last menstrual period. We examined coronary heart disease death rates in the 1183 who were born at term. The opposing trends in standardised mortality ratios with abdominal circumference were similar to those of all men (table).

Comment

We have found that among men whose birth weights were below average those who had a small abdominal circumference at birth had raised death rates from coronary heart disease. This is consistent with the association between small abdominal circumference at birth and raised serum cholesterol and plasma fibrinogen concentrations, found in a separate sample of men.[2,3] In late gestation the fetus responds to undernutrition by sustaining growth of the brain at the expense of growth of the trunk.[4] This adaptation compromises the liver and seems permanently to alter low density lipoprotein cholesterol and fibrinogen metabolism.

Among men with above average birth weight those who had a large abdominal circumference at birth had raised death rates from coronary heart disease. The babies of women who develop impaired glucose tolerance in pregnancy become macrosomic. During their accelerated growth in late gestation the fetus's abdomen enlarges rapidly.[5] The processes underlying this are not known, though deposition of glycogen in the liver has been suggested.

Our findings suggest that both reduced and accelerated liver growth in late gestation are early determinants of coronary heart disease.

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Correction

Predictors of ratio of placental weight to fetal weight in multiethnic community

An authors' error occurred in this article by Ivan J Perry and colleagues (pp 436-9, 18 February). In the first line of page 438 the data are geometric mean (95% confidence interval) and not geometric mean (range) as printed.

A SUMMONS THAT CHANGED MY PRACTICE

A lower threshold for referrals

In retrospect that morning had seemed deceptively safe. A full surgery was punctuated by a welcome coffee break. Paperwork was tackled with a fair degree of success, if not pleasure. The normal background noise of familiar general practice abounded: prescriptions, interruptions, housecalls.

The summons was delivered by two uncomfortable and visibly embarrassed men in ill fitting business suits. One read a legal statement as if it were a school poem, learnt by rote and with no inflection. The summons and copies were then handed to each partner from the practice. I remember us standing in a row with a variety of expressions: angry, perplexed, bemused, humiliated.

I was the doctor who had failed to diagnose the testicular torsion some three years earlier. The patient was a warm young chap, with whom I had always maintained a good rapport. Two days before he saw me he had been seen by the urologist in the casualty department of the local teaching hospital. He was complaining of loin and testicular pain with nausea and dysuria. A diagnosis of epididymo-orchitis had been made and antibiotics started. He had walked in to see me with a smile and a slightly embarrassed look, as the problem was "down below." I took a history, examined him, and agreed with the diagnosis that had been made. I arranged review for 24 hours. By then he was worse and I referred him back to the hospital. The torted testis was not recoverable and was removed the same day.

On discharge I continued to see and treat him. I offered counselling and advised him on prostheses. I tried to explain why the diagnosis had proved difficult to make. At no point did he show animosity or loss of confidence in me. He did not seem inclined to apportion blame and my initial anxieties around the case settled as our relationship was quickly re-established. That was until the summons arrived. I felt angry, betrayed, and insecure. It turned out that it was three years to the day since the event had occurred, the absolute deadline for the issue of and delivery of the summons to instigate proceedings. I doubted my own clinical judgment. At times I despaired. For a while I was sleepless and depressed. For many months I was preoccupied with the details of the case, my mind reviewing them endlessly like a continuous film loop.

Some two years later the case was dropped. My defence society indicated that because I had seen, taken a history from, and thoroughly examined the patient, I had done all the things a reasonable general practitioner could be expected to do. In addition I had written full notes and recorded that I had considered the diagnosis of torsion. The presenting symptoms and signs had been unusual. There was little case to answer. After the initial relief I continued to be troubled by the whole affair. Was there not an element of my being at fault? Intellectually I knew that this was not the case, but I felt unremitting sadness and a sense of betrayal.

I elected to make some changes to my own practices to protect against a recurrence of the circumstances. I have lowered my threshold for referring. I try to accept my own fallibility and that of others. I try to balance the opinion of other doctors with the evidence that is in front of me. Sadly, I place less importance on how good a relationship seems to be with a patient.

Tennessee Williams said, "We have to distrust each other. It's our only defence against betrayal." There is a loss of faith in human nature behind this sentiment that I would rather not embrace, yet part of me thinks there may be some truth in it.