THE INFLUENCE OF MATERNAL HAEMOGLOBIN LEVEL AND OBSTETRIC HAZARD ON THE INTELLIGENCE OF THE CHILD

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INTRODUCTION

The causes of the variation in the intelligence of the population have been classified into two main categories, heredity, over which there is no control, and environment, which theoretically is subject to some control. The individual owes his ability to the constant interaction between the innate capability transmitted to him by his parents, and the particular circumstances in which he is born and develops, and to which he tends with varying success to adapt and adjust himself.

Burt (1955) has suggested that heredity might account for as much as 75% of the variation in ability, whereas Knobloch and Pasamanick (1959) with the directly opposing view, suggest that all the observable differences in intellectual ability are the result of prenatal damage, the birth process or post-natal conditions.

It has been suggested that a reduction in mental retardation will accompany a reduction in perinatal mortality (Clifford, 1964). This suggestion is based on the idea that perinatal mortality is the observed portion of the iceberg, and that the submerged and much the larger portion is the subclinical, unrecognised, minimal brain damage which results from adverse prenatal and perinatal factors.

Various authorities have listed the principal obstetric factors which are thought to increase the likelihood of mental subnormality (Baird, 1960; Richards, 1963; Illingworth, 1964), and it will be the purpose of this work to determine the relative effect of each of these factors, and supporting the theory that their damaging effect is caused by foetal anoxia, to show that a high level of maternal haemoglobin is a protective factor.

REVIEW OF RELEVANT PAST WORK

That certain gross mental and neurological defects are positively associated with obstetric hazards, such as prematurity, foetal hypoxia due to severe complications of pregnancy, or cerebral damage at delivery, is not in doubt. Of the children born in these adverse conditions, only a minority sustain this severe damage, the majority escaping serious sequelae. However, there is now increasing evidence that some of the less serious impairments of motor and intellectual function, and some specific educational problems may result from these obstetric complications (Drillien, 1964).

Several studies have shown an increased incidence of "prematurity" amongst mentally sub-normal and backward children (Ascher and Roberts, 1949; Boskow, 1949; Baird, 1959; Fairweather and Illsley, 1960; Drillien, 1963; Lubchenko Et Al, 1963; Zappella, 1963; Berenberg and Ong, 1964; Rostafinski, 1964). Other workers have found that the apparent differences in intelligence between children born prematurely and matched control cases, disappeared when due allowance was made for inadequate matching (Howard and Worrell, 1952; Douglas, 1960; MacDonald, 1964), and that premature birth unassociated with intra-cranial injury did not affect mental development (Tredgold and Soddy, 1963).

It is accepted that prematurity is one of the more dangerous obstetric complications (Baird, 1960; Butler and Bonoham, 1963) but few authorities when discussing prematurity have differentiated between babies of low birth weight for gestation and purely immature babies.

About one third of all babies classed as "premature" on a weight basis are in fact born after a pregnancy of at least thirty-eight weeks' duration (Butler and Bonham, 1963), and so the term "dysmaturity" has been introduced to describe the condition of those babies born underweight for the period of gestation (Sjöstedt Et Al, 1958; Scott, 1966).

Studies into cerebral myelination have shown that the vulnerable period for human infants would be from the seventh intrauterine month to the end of the first few months of post-natal life (Davison and Dobbing, 1966). Therefore the full-term newborn infant, and even more so the immaturely born, would thus be at special risk from external factors, especially anoxia.

Consideration of the findings of Butler and Bonham (1963) shows that babies premature according to weight after mature or prolonged pregnancy, i.e. dysmature, have a mortality ratio which is eight times higher than that for babies weighing over 5½ lbs., whereas babies weighing 5½ lbs. and over, and born before the thirty-eighth week of pregnancy have a mortality ratio which is only twice that of all babies weighing over 5½ lbs.

Recent work on placental anatomy has shown that the weight of the baby varies directly with the weight of the placenta and the chorionic villous surface area (Aherne and Dunnill, 1966A). This work would support the view that dysmaturity results from placental insufficiency, which causes, amongst other nutritional deprivations, a reduced oxygen supply to the foetus.

To differentiate such distinct conditions as immaturity, birth trauma and other obstetric hazard, is sometimes impossible, since frequently one condition may result from another, e.g. trauma causing cerebral oedema, leading to compression of the cerebral vessels, resulting in ischaemia and anoxia (Rostafinski, 1964). Indeed, immature infants sustain birth injuries in higher proportions than full-term infants (Douglas, 1956) due perhaps in some measure to the fragility of the blood vessels and the hypoprothrombinaemia (Maunter, 1959).

Brain damage can occur either during intrauterine life or post-natally. Anoxia or haemorrhage is the usual cause of the damage which, if it is sufficiently extensive, results in mental subnormality (Holt, 1965). It has been estimated that birth injury could account for 5% of all mental defectives (Kirman, 1952; Tredgold and Soddy, 1963), but in 1966 Kirman raised this estimate to 11%.

This relationship has found support from many authorities since Little's original work in 1862, and it has been concluded that deficits in complex verbal and visuomotor tasks may be indicative of "minimal" brain damage (Klatskin, 1964).

Recent work has shown that birth injuries are due primarily to intrauterine asphyxia rather than the actual trauma (Reid, 1959; Ahvenainen, 1964). Butler and Boneham (1963) reported that in deaths following breech delivery there was three times more intrapartum anoxia than pure cerebral birth trauma; and the annual report of the Registrar-General for Scotland (1962) showed that paranatal anoxia caused more than four deaths to every one by mechanical injury, and this is confirmed by Courville (1963) who found a similar proportion in 466 neonatal deaths. It is now established that mechanical injuries at birth, such as cord prolapse and malpresentation, as well as high or mid forceps deliveries, are of little significance in the aetiology of mental retardation; but rather that those prenatal and perinatal factors which produce anoxia are of greater importance than mechanical injury in relation to post-natal neurological sequelae (Solnitzky, 1964).

It is commonly thought that the first-born child is more likely to be mentally retarded (Tredgold and Soddy, 1963) due to the increased incidence of birth injury (Penrose, 1949), and it has been said that the greater the number of siblings preceding the baby expected, the better are his prospects of being born safely (Schwartz, 1961). Although Butler and Bonham (1963) confirm that there is in fact an increased risk to the first-born compared to the second-born, they show that subsequently the risk rises with each birth. However, it is known that the average birth weight rises with parity (McKeown and Gibson, 1951; Karn and Penrose, 1951) and this advantage, due perhaps to improved placental function in later pregnancies, may protect the intellectual development of the later children in the family. Also, Minkowski (1957) has suggested that in multiparae the uterine contractions are less powerful and act for a shorter time, and therefore have less of an adverse effect on the placental blood flow. Nevertheless, Fairweather and Illsley (1960) did find that the higher parities were over-represented in their series of mentally handicapped children.

There is an ever-increasing library of evidence that asphyxia at birth is a cause of mental retardation. Several retrospective studies have found a higher incidence of perinatal anoxaemia amongst mentally retarded children (Darke, 1944; Rostafinski, 1964), and other prospective studies of children who were asphyxiated at birth have shown a higher incidence of mental retardation (Fraser and Wilks, 1959; Walker, 1964; MacDonald, 1964; Mastyukova, 1964). Penrose (1949) points to the results of animal experiments to support this, experiments which showed that induced anoxia at birth caused cerebral damage.

Although there is some evidence that the neonate has an increased resistance to asphyxia (Smith, 1948), paranatal anoxia is in fact the commonest cause of perinatal death - 22.9% (Butler and Bonham, 1963). Increasing anoxia causes foetal tachycardia, then bradycardia, then meconium is passed and these signs of foetal distress are associated with increased perinatal mortality (Walker, 1954). Paranatal anoxia can also be induced by pressure on the foetal head causing bradycardia (Yagi, 1954; Minkowski, 1957), and it is claimed that only slow accommodation of the head is physiological and without danger to the child; otherwise, in prolonged and high forceps deliveries or precipitate delivery, especially caesarean birth, there is an increased probability of inhibition of normal blood flow through the cerebral vessels (Grontoft, 1954). Again, anoxia can cause distention of intracranial blood vessels, with stagnation of the blood, and this, aggravated by hypoprothrombinaemia and the underdevelopment of the vessels themselves, is probably the reason for such severe damage resulting from minimal trauma in immaturely born babies (Howard and Warroll, 1952; Goerttler and Draisbach, 1963).

The relationship between low oxygen saturation of the blood in the umbilical vein at birth, and infant neurological symptoms and signs, is well demonstrated by Minkowski (1957), who showed that out of 206 cases, 72 had had an oxygen saturation below 80%, and of these more than half had abnormal neurological signs.

Heyns (1962) has no doubt that even normal labour has an adverse effect on the foetus, mainly because of anoxia, and he has described in detail a technique of reducing the atmospheric pressure around the abdominal wall during labour. He found that the babies born by this decompression technique were on average superior in mental and physical development to those not having experienced decompression. In his discussion he says that the decompression may do no more than improve the circulation in the placenta in pregnancy and during labour when there is a very real danger of hypoxia (Ingalls, 1950).

Certainly abnormal features in the immediate post-natal period, such as delay in the establishment of respiration, cyanotic attacks and cerebral crying, are signs of intracranial damage (Hess Et Al, 1934). But, although it is an accepted criterion, the interval between birth and the first breath is an extremely crude estimation of the severity and duration of perinatal asphyxia.

Studies of placental blood flow, using radioactive sodium, have shown that certain obstetric complications could affect the foetus during pregnancy (Browne and Veall, 1953). These studies have shown the reduced intervillous blood flow in late pregnancy complicated by pre-eclamptic toxaemia and hypertension; and more recent studies into placental anatomy have shown that the total placental volume and chorionic villous surface area were each reduced by over 25% in cases of hypertension (Aherne and Dunnill, 1966B).

Solnitzky (1964) has stated that toxaemia is not associated with mental retardation, but rather with minor degrees of cerebral damage that lead to behaviour and specific learning disabilities. Complementary to this are the views held in the U.S.S.R. that there is a relationship between the oxygen saturation of the blood to the foetus and pre-eclamptic toxaemia and, in support of Heyns (1964) that psychoprophylaxis, which is a method of preparation for active participation in labour involving the dissociation of voluntary muscles, improves oxygen saturation to the foetus and reduces intrapartum asphyxia (W.H.O. Chron., 1966).

Reduced placental sufficiency may account for the high incidence of twins resident in mental deficiency institutions; this incidence actually rises to 4%, whereas in the general population it is only 1.2%. Certainly it is agreed that the mean intelligence quotient of twins tends to be lower (Illingworth and Woods, 1960; MacDonald, 1964; Illingworth, 1964

Anaemia of Pregnancy

From a review of the literature, Hytten and Duncan (1956) quoted incidences of anaemia of pregnancy which ranged from 5% (Medical Research Council, 1945) to 20.1% (Scott and Govan, 1949). Butler and Bonham (1963) showed that 14.6% of pregnant women had a haemoglobin level below 70%, and that the risk to the baby was higher in these cases. In fact, the risk of perinatal foetal death was twice the normal in those cases where the haemoglobin level was below 60%.

However, because of differences in technique of blood sampling and of estimating and recording, and differences in the stages of pregnancy at which the samples were taken, published surveys are rarely comparable and allow only a rough assessment of the incidence of anaemia in pregnancy.

The principle that supplementary iron should be given during pregnancy to prevent anaemia has been questioned by Hankin (1963), who found that there was no significant difference in the mean haematocrit levels in the treated and untreated groups at any period, but his sample was small and his methods of selecting his two groups are open to criticism. Hytten and Leitch (1964) demonstrated what appeared to be the normal anaemia of pregnancy due to haemodilution, but cited the findings of Gerritsen and Walker (1954) who found that Bantu women, who have an exceptionally high intake of iron in their diet, do not show this haemodilution effect. It is the opinion of Briscoe (1963) that the term "physiological anaemia of pregnancy" should be abolished, that a drop in the haemoglobin value during pregnancy is not normal, and that all women should receive iron supplementation during pregnancy, regardless of their haemoglobin level. From his investigations into the amount of stored iron (details of his methods are not given), Briscoe has estimated that 20% of pregnant women have normal stores, 60% have reduced stores and 20% have no storage iron. He found that the incidence of "significant anaemia" ranged from 20% to 30%. By using the differential ferrioxamine test, which is based on the iron chelation of desferrioxamine ("DESFERAL"), Fielding, O'Shaughnessy and Brunstrom (1965) have accurately measured the iron stores of apparently healthy women with normal haemoglobin levels, and have found that one-third of them have no significant iron stores. Add to this group the 20% of women who are clinically anaemic, and it will be seen that 50% of women have no stored iron.

No matter what the haemoglobin state of the mother might be, there is n significant difference of mean haemoglobin levels during the first twenty-four hours of life or at three months of age on comparing infants born to either anaemic or non-anaemic mothers (Lankowski, 1961). To accomplish this, the foetus requires 300 mgs. of iron; the increase in the maternal circulating blood volume requires a further 450 mgs. of iron to maintain the non-pregnant haemoglobin level (Briscoe, 1963). It is therefore obvious that in 50% of women, the increased demand for iron in pregnancy must inevitably lead to iron deficiency anaemia.

Social Class

The mean I.Q. rating of children falls regularly as the mean intelligence of the parents descends (Penrose, 1938; Illsley, 1961), and the intelligence of parents can be differentiated with regard to occupation class (Jones and Carr-Saunders, 1927; Burt, 1926). The correlation between social class of parents and the I.Q. scores of their children has been estimated at -0.35, with groups from class 3 families having a mean I.Q. score of about 15-25 points higher than that of class 5 families (Wells Et Al, 1951).

Social class affects pregnancy directly in several ways. With regard to birth weight, the percentage of babies weighing under $5\frac{1}{2}$ lbs. rises from 3.6% in social class 1 to 8.2% in social class 5. This relationship is also seen when the length of pregnancy is considered, for the incidence of pregnancies ending before the thirty-eighth week is 50% higher in social class 5 than in social class 1, and also the risk to the baby rises as the social class declines (Illsley, 1961; Butler and Bonham, 1963).

When Fairweather and Illsley (1960) considered their findings after a retrospective survey of 66 mentally handicapped children, they found the families were characterised by poor social conditions, low average intelligence and high fertility; indeed the higher parities were over-represented in the series; only 34 were first-born (45 were expected), and 18 were fifth-born or later (6 were expected). Thompson (1949) had previously shown that there is a definite negative association between the size of the family and average intelligence score, this association being a linear relationship with a correlation factor of -0.28 on group testing, and -0.32 on Terman Merrill I.Q scale.

This correlation closely matches that obtained by Burt in 1946, which was between -0.19 and -0.33.

It has been claimed that this drop in intelligence with declining social class and the larger family is due in some measure to the less stimulating home environment and lack of parental interest in schooling (Douglas, 1960; Kushlick, 1964), for it is agreed that an exceptionally good environment can raise the I.Q by as much as 20 points (Burks, 1928).

MATERIALS AND METHOD

The survey was carried out in the Eastern Area of Warwickshire, which is the catchment area for St. Mary's Maternity Hospital, situated in Harborough Magna, a village close to Rugby, the administrative centre for the area.

Between 1st September, 1951, and 31st August, 1954, 2,122 women were confined in St. Mary's Hospital. There were 68 stillbirths, 33 neonatal deaths, 25 sets of twins and 1 set of triplets; therefore 2,051 babies were discharged home. These were to be the potential population of this prospective survey. However, of this number, only 1,123 were still resident in the area and attending ordinary schools at the time of the 11+ examination. Because in this survey the measurement of intelligence is based on the result of the 11+ examination, no account has been taken of those children attending special schools for the handicapped (1.9%) nor of those excluded from school under Section 57 of the Education Act, 1944 (0.4%), nor, finally, of those who were attending private schools (approximately 7%).

Permission was sought to search and extract information from the hospital medical (obstetric) records of the mothers of those 1,123 children. Unfortunately, 201 of these records were incomplete due, in the majority of cases, to the mother being admitted in emergency, without previously attending the ante-natal clinic, and consequently without a record of her haemoglobin state.

The survey was limited therefore to a population of 922 children. Since all severely mentally retarded children and physically handicapped children had been excluded from this survey, this sample represents over 97% of the population.

The relevant information on each child was extracted from three main sources:-

A. The hospital medical (obstetric) record of the mother;

B. The Education Authority's record of the result of the 11+ examination (Moray House), and

C. The official Register of Births, from which was extracted the occupation of the child's father.

It was recognised that the use of these records gave a marked degree of consistency in recording certain parameters which are open to observer variation; for instance, haemoglobin estimations, taken on the first visit to the ante-natal clinic, usually between the twelfth and sixteenth week of pregnancy, were all recorded by one technician, the details of labour and condition of the baby at birth were observed and recorded by one of three or four labour room Sisters, and the I.Q scoring of all the children was by the standardised Moray House group intelligence tests.

The school health records and the infant welfare records, where available, were searched, and it was found to be unnecessary to exclude any child on account of a history of any medical condition which might have had a deleterious effect on the I.Q score.

A card, as illustrated in Appendix I, was prepared for each child and the following variables recorded on it:-

Maturity was the duration of the pregnancy measured in days:- Computer Code = "DAYS".

Length of the first stage of labour was measured in hours:- Computer Code = "HOURS".

Length of the second stage of labour was measured in minutes:- Computer Code = "MINS".

Age of the mother was recorded as her age on the birthday prior to her first visit to the ante-natal clinic:- Computer Code = "AGE".

Gravidity of the mother was recorded at her first visit to the ante-natal clinic and included stillbirths and any known miscarriages:- Computer Code = "PAR".

Condition of the baby at birth was a numerical evaluation of the description given by the midwife at the time of delivery.

Good = 1; Fair = 2; Blue = 3; Blue and given oxygen = 4; White asphyxia = 5 :- Computer Code = "CONDIT".

Weight of the baby was measured in pounds:- Computer Code = "WT".

Social class was determined by the occupational unit of the father (Census 1951, Great Britain (1% sample) Part I, Section II):- Computer Code = "CLASS".

Haemoglobin was recorded as a percentage of 14.8 Gm haemoglobin per 100 ml:- Computer Code = "HB".

Type of delivery was recorded only if completed by forceps:- Computer Code = "FORCEP"; or by caesarean section:- Computer Code = "CAESAR"

The "at risk" space was to contain any complications in pregnancy, such as hypertension:- Computer Code = "H.B.P.", or pre-eclamptic toxaemia:- Computer Code = "P.E.T.", or multiple pregnancy:- Computer Code = "MULT".

These variables can be separated into three groups.

Group 1 consists of those variables which might be related to the I.Q in a continuous linear fashion, i.e. a change in the variable will cause a corresponding change in the I.Q score. This is expressed by the equation y = ax. Within this group would be the Age of the mother, the Condition of the baby at birth, the Social Class and the Haemoglobin estimation.

Group 2 consists of those variables which might be related to the I.Q in a "quadratic" fashion, i.e. a rise in the value of the variable causes a rise in the value of the I.Q, till the optimum is reached and then any further rise in the value of the variable causes a fall in the value of the I.Q. This is expressed by the equation $y = a + bx + cx^2$, and so for the purposes of the proposed analysis, the squares of the values of the variables in class 2 were added to the data, and are recognised in computer code by the repetition of the last letter. Within this group are five variables: Maturity, since postmaturity might be as damaging as prematurity; Length of first and second stages of labour, since precipitate labour might be as damaging as prolonged labour; Gravidity, since both the first and last born in a large family might be equally at risk to perinatal damage; and the Weight of the baby, since there may be as much danger for a very large baby as for a very small one.

Group 3 consists of those conditions which are not true variables, but rather "factors" which are either present or absent. Contained in this final group would be those given the computer codings of "H.B.P.", "P.E.T.", "FORCEP", "CAESAR" and "MULT".

The basic data was first punched on to five-hole-tape in Elliott code. Then the addition of the "squares" of the variables in group 2 was made, a constant term of 1 ("CONST") inserted, the numbers checked and the layout arranged by using simple ALGOL programs and submitting the data to an Elliott 803 digital computer.

The final stage in the preparation of the data tape was to convert the data into Ferranti Pegasus code.

The print-out of the complete data is presented in Appendix II.

Method of Analysis

In industry there are many processes where the nature or amount of an output from a process is dependent upon a variety of factors, some of which are under control and some of which are not; improved control of such processes can only be obtained by discovering more about the extent to which the various factors actually influence the output. This is done by recording the value which each factor takes in practice, and this is done without interfering in any way with the normal methods and fluctuations in production. It is then possible to analyse such records to show the connection between the variation in the factors and that in the resultant output; the method used for doing this is MULTIPLE REGRESSION ANALYSIS.

Where there are many factors suspected of simultaneously affecting the output of a process, it is necessary to measure the effect of each factor, paying due regard to the associated effects of all the other factors. The aim therefore of multiple regression analysis is to obtain from the recorded values an equation of the form

$$y = b_0 + b_1x_1 + b_2x_2 + b_3x_3 + ----- + b_nx_n$$

where y is the dependent variable (e.g. output or, in our case, the I.Q) and x_1, x_2, x_3, --- x_n, the independent factors or variables, each with its own influence on y, the amount of which is measured by the regression coefficients b_1, b_2, b_3 ---- b_n. b_0 is a constant factor which is that part of y that is not explained by the independent variables in the equation.

In practice, it is necessary to discover whether an equation so calculated has any real value. This is done by establishing how likely it would be for such values of "b" to occur by chance circumstance in spite of there being no real effect of x on y, i.e. by establishing the statistical significance of the result. It is usual to accept as meaningful any result which has a greater than 5% probability of occurring by chance. Results where the probability is less than this value are said to be significant at the 5% level. In some cases where a large number of variables is under consideration, a significance level of 1% or less is used.

One computer program for multiple regression analysis is the FERRANTI PEGASUS PROGRAM MK. 1.B., which was devised for the British Iron and Steel Research Association (B.I.S.R.A.). This program is designed to carry out multiple regression analysis involving any number of variables up to 26, designated by the code letters A - Z. Significance testing of the variables included in a regression equation with the elimination of non-significant variables is carried out according to certain rules, as follows:-

During the calculation with this program, if the regression coefficient of a particular variable (x) is found to be not significant (e.g. its probability of occurring by chance is higher than 5%), then this particular variable is removed from the subsequent analysis of the regression analysis. However, if several variables qualify for exclusion when considered individually, it is necessary to make sure that they nevertheless do not have a combined effect which is significant. If this were to be the case, these particular variables are returned to the analysis, in decreasing order of their individual significance, until the combined effect of the ones still excluded is no longer significant.

In interpreting the results from a regression analysis it should be remembered that the regression equation is derived from a certain observed range of values of each of the variables, and in consequence there may be considerable risk in accepting the equation's applicability outside this range.

In addition to calculating the regression coefficients together with their corresponding standard errors and t-ratios, other quantities are evaluated. These comprise a summary of the minimum, mean and maximum values of each variable, together with its standard deviation, and also a matrix of the simple correlation coefficients between pairs of variables.

Unfortunately this program had been written specifically for the Ferranti Pegasus computer to which access was not immediately available, but since the program appeared to be so well suited to the purpose of the survey, enquiries were made, and in time access was granted by the United Steel Companies of Sheffield to their Ferranti Pegasus and Elliott 503 combined computer.

RESULTS

The statistical analysis by the computer is based on the formation of a matrix of the sums of squares and products, and in the build-up of this matrix, quantities may be formed which overflow the capacity of the computer store. A binary scale factor of 2^{-10} is introduced to overcome this difficulty. However, on the first attempt to run the program, this scale factor proved to be inadequate to prevent overflow.

On the second run the scale factor was lowered to 2^{-15}, but even this was inadequate to prevent overflow when an analysis of the full complement of variables was sought.

It is now known that an even lower scale factor could have been introduced into the program, but, since access time to the Pegasus computer was limited, a compromise was made, which entailed excluding the quadratic expressions (except "PARR"), and dividing the variables into two unequal groups. These were analysed separately and the results are displayed in Tables 3 and 4.

MULTIPLE REGRESSION PROGRAM

TABLE 1

SUMMARY	MINIMUM	MEAN	MAXIMUM	SIGMA
I.Q	70	106.27	140	14.60
DAYS	149	278.89	314	12.93
DAYSS	22201	77949.43	98596	6932.42
HOURS	.00	10.6735	86.00	10.6746
HOURSS	.00	227.8713	7396.00	647.8509
MINS	0	42.85	295	43.90
MINSS	0	3763.93	87025	8578.81
AGE	16	27.98	45	5.40
PAR	1	2.06	12	1.29
PARR	1	5.90	144	9.72
CONDIT	1	1.26	5	.73
HB	60	87.32	113	8.74
WT	3.00	7.3816	12.80	1.0717
WTT	9.00	55.6358	163.84	15.9274
CLASS	1	3.06	5	.77
CONST	1	1.00	1	.00
H.B.P	0	.05	1	.22
P.E.T	0	.08	1	.27
FORCEP	0	.04	1	.19
CAESAR	0	.06	1	.24
MULT.	0	.02	1	.14

(SETS) n = 922

Table 1 is a summary of each variable with its minimum, mean and maximum value and standard deviation.

The validity of the significance tests depends on the assumption that all the variables are normally distributed, or nearly so, and to check this, use can be made of this table.

Applying the definition of a normal distribution, that the curve should be symmetrical about the mean, it will be seen that seven variables, namely, Gravidity ("PAR") and its square "PARR", Length of first stage ("HOURS") and its square "HOURSS", Length of second stage ("MINS") and its square "MINSS" and Condition of the child at birth ("CONDIT"), do not completely satisfy this requirement, and therefore the results of the analysis, with regard to these variables, must be accepted warily.

Use is made in Table 5 of the range between the minimum and maximum values in calculating the range of effect of each significant variable.

Finally, reference to this table will prevent the interpretation of the results of this analysis beyond the range of its applicability.

TABLE 2

I.Q									
-007	DAYS								
049	116	HOURS							
053	033	343	MINS						
-000	-014	-168	-157	AGE					
-197	033	-219	-323	487	PAR				
-099	-010	119	169	002	-080	CONDIT			
137	041	054	034	-054	-095	029	HD		
070	363	-022	029	027	132	-026	060	WT	
-247	-025	-027	-019	-079	048	055	-074	-048	CLASS
-034	-026	-035	064	048	070	-014	048	066	H.B.P.
-019	-067	015	077	045	-093	002	083	-003	P.E.T.
034	000	365	508	-020	-127	177	018	-012	FORCEP
-078	-161	-247	-249	119	-051	073	-033	-083	CAESAR
-081	-196	-031	158	076	030	-008	-037	-229	MULT.

CLASS					
-056	H.B.P.				
-040	-066	P.E.T.			
023	-017	032	FORCEP		
-060	-038	044	-049	CAESAR	
020	-032	047	-027	-026	MULT.

<u>Table 2</u> is a matrix of the simple correlation coefficients.

The coefficient of correlation between two variables is found by taking the element common to the column of the variable with the lower subscript, and the row corresponding to the variable with the higher subscript.

The coefficients are printed to three decimal places and the decimal point has been omitted.

The highest positive correlation in the matrix is between the length of the second stage of labour and forceps delivery (0.508). Other high positive correlations are between age of the mother and gravidity (0.487) and between duration of pregnancy and weight of the baby at birth (0.363).

The highest negative correlation is between the length of the second stage of labour and gravidity (-0.325); and two negative correlations of special interest are those between I.Q and social class (-0.247) and between I.Q and gravidity (-0.197).

Dependent Variable				Sum of Squares of Dependent Variables			
I. Q.				196473			
Level of *"p"	Degrees of Freedom	F Ratio	Independent Variables	Regression Coefficient	Standard Error	t Ratio	Residual sum of squares
-	915	6.20	Constant term	88.60	4.810	18.42	
			CONDIT	-2.149	0.6574	-3.27	
			HB	0.2380	0.05442	4.37	
			H.B.P	-3.071	2.182	-1.41	
			P.E.T	-1.781	1.788	-1.00	
			FORCEP	3.786	2.595	1.46	
			CAESAR	-3.838	1.978	-1.94	188795
10%	918	10.75	Constant term	88.86	4.805	18.49	
			CONDIT	-1.960	0.6471	-3.03	
			HB	0.2306	0.05421	4.25	
			CAESAR	-4.013	1.973	-2.03	189805
5%	918	10.75	Constant term	88.86	4.805	18.49	
			CONDIT	-1.960	0.6471	-3.03	
			HB	0.2306	0.05421	4.25	
			CAESAR	4.013	1.973	-2.03	189805
1%	919	14.01	Constant term	88.40	4.808	18.39	
			CONDIT	-2.058	0.6464	-3.18	
			HB	0.2345	0.05427	4.32	190661
0.1%	920	17.70	Constant term	86.25	4.784	18.03	
			HB	0.2294	0.05451	4.21	192764

* "p" = Probability

Table 2 details the progress of an analysis, mainly of those variables previously described as obstetric factors which are either present or absent in a pregnancy.

The most noticeable feature in this analysis is that the influence of forceps delivery is eliminated as a non-significant factor at such an early stage.

The influence of the level of maternal haemoglobin persists in significance after the elimination of all the other factors, and this is due, no doubt, to the inclusion in "maternal haemoglobin level" of some influence more properly ascribed to "social class", which had not been included as a variable in this analysis.

Dependent Variable				Sum of Squares of Dependent Variables			
I.Q.				196473			
Level of *"p"	Degrees of Freedom	F Ratio	Independent Variables	Regression Coefficient	Standard Error	t Ratio	Residual sum of squares
-	909	12.41	Constant factor	101.0	7.724	13.07	
			DAYSS	-0.0001108	0.0000709	-1.57	
			HOURS	0.02884	0.04597	0.63	
			MINS	-0.0003813	0.01171	-0.03	
			AGE	0.3217	0.09768	3.29	
			PAR	-3.208	0.9865	-3.25	
			PARR	0.04763	0.1200	0.40	
			CONDIT	-2.232	0.6247	-3.57	
			HD	0.1841	0.05215	3.53	
			WT	1.427	0.4599	3.10	
			CLASS	-4.030	0.5894	-6.84	
			H.B.P	-3.684	2.123	-1.74	
			P.E.T	-4.089	1.722	-2.37	168814
10%	913	18.28	Constant factor	94.48	6.436	14.68	
			AGE	0.3144	0.09654	3.26	
			PAR	-2.897	0.4117	-7.04	
			CONDIT	-2.184	0.6152	-3.55	
			HD	0.1837	0.05201	3.53	
			WT	1.150	0.4250	2.70	
			CLASS	-4.032	0.5886	-6.85	
			H.B.P	-3.368	2.079	-1.62	
			P.E.T	-3.846	1.708	-2.25	169352

* "p" = Probability

Dependent Variable	Sum of Squares of Dependent Variables
I. Q.	196473

Level of "p"*	Degrees of Freedom	F Ratio	Independent Variables	Regression Coefficient	Standard Error	t Ratio	Residual sum of squares
5%	914	20.48	Constant factor	94.93	6.436	14.75	
			AGE	0.3114	0.09661	3.22	
			PAR	-2.928	0.4117	-7.11	
			CONDIT	-2.177	0.6157	-3.54	
			HB	0.1792	0.05199	3.45	
			WT	1.114	0.4248	2.62	
			CLASS	-3.981	0.5883	-6.77	
			P.E.T	-3.656	1.706	-2.14	169839
1%	915	23.03	Constant factor	95.65	6.440	14.85	
			AGE	0.2905	0.09630	3.02	
			PAR	-2.819	0.4093	-6.89	
			CONDIT	-2.162	0.6169	-3.51	
			HB	0.1711	0.05195	3.29	
			WT	1.108	0.4256	2.60	
			CLASS	-3.949	0.5893	-6.70	170692
0.1%	916	26.12	Constant factor	103.3	5.747	17.98	
			AGE	0.2785	0.09649	2.89	
			PAR	-2.666	0.4063	-6.56	
			CONDIT	-2.181	0.6188	-3.52	
			HB	0.1804	0.05199	3.47	
			CLASS	-4.032	0.5903	-6.83	171955

* "p" = Probability

Table 4 details the progress of an analysis of 12 independent variables.

The squared values of the duration of gestation were used in preference to the unsquared values, in the hope that by exaggerating the effect of immaturity, any minor relationship between immaturity and the I.Q score would be revealed.

The squared values of gravidity ("PARR") were included, since of all the possible quadratic relationships, the one between I.Q and gravidity seemed to be the most probable. However, the influence of this variable, "PARR", on the I.Q of the child was eliminated immediately, and this suggests that the relationship between gravidity of the mother and the I.Q of the child is, in fact, linear.

Other variables eliminated immediately were duration of gestation and the lengths of the first and second stages of labour.

Hypertension ("H.B.P.") is present as a negative influence at the 10% level of significance but not at the 5% level. However, the influence of pre-eclamptic toxaemia ("P.E.T.") persists into the 5% level of significance.

The "HB" variable is still a significant influence at the 0.1% level, i.e. $P < 0.001$.

TABLE 5

Variable	Range	Correlation coefficient	Standard deviation	Mean range of effect on I.Q score	Mean percentage effect, i.e. relative effect
Age of mother	16-45 yrs.	+ 0.3114	0.09661	+ 9.0	9.5%
Gravidity Amended	1 - 12 1 - 5	- 2.928	0.4117	- 11.7	13%
Condition at birth	1 - 5	- 2.177	0.6157	- 8.7	9%
Maternal haemoglobin level	60-113%	+ 0.1792	0.05199	+ 9.5	10%
Weight at birth	3-12.8 lbs.	+ 1.114	0.4248	+10.9	11.5%
Social class	1 - 5	- 3.981	0.5883	-15.9	16%
Pre-eclamptic toxaemia	Present or absent	- 3.656	1.706	- 3.7	4%
Constant term	82.06 to 107.80	-	6.436	25.7	27%
Total				95.1	100%

Table 5 is an extension of the regression equation at the 5% level of significance in Table 4. The multiple correlation factor of this equation is 0.35.

In this table the mean range of effect on the I.Q score of each variable, is calculated by multiplying the correlation coefficient by the range of applicability of each variable.

The amendment to the range of the variable "Gravidity" was introduced because the results with regard to this variable were to be accepted with reserve, due to the distortion of its distribution curve, and the range of gravidity in normal practice more closely resembles 1 to 5.

The constant term in the equation represents the variation in the I.Q score which is not explained by the variables, and has a range of four times its standard deviation.

The total possible range of effect of all the variables is 93 points on the I.Q scale, and the mean percentage (i.e. relative) effect of each variable is shown in the last column.

N.B. The unallocated portion of the total variance in the data is still considerable, in spite of the number of independent variables being increased to twelve. This would suggest that the use of the technique of multiple regression analysis in this type of study is capable of further development.

DISCUSSION

In view of the total mass of data and of the intricacies of the interrelationships between each of the variables, an analysis by digital computer was appropriate.

The program used for the analysis was chosen primarily because it included tests of significance. Other considerations were the clear presentation of the results and the well documented program manual. Unfortunately this program could not be run on the Elliott 803 digital computer, at the Rugby College of Advanced Technology, to which access was available. However, this access, amounting to many hours of computer time, allowed full preparation of the data tapes, so that when access to the Elliott 503 - Pegasus combined computer of the United Steel Companies was granted, only the actual running time of the program was required, amounting to three hours in all.

The final results detailed in Table 5 are an extension of the findings of the computer.

The regression equation composed of those variables with significant influences at the 5% level, has a constant factor of 95, with a standard deviation of 6.436. This factor represents the variation in the I.Q score which is not explained by the variables, and could perhaps be identified with the "basic inherited intelligence" which Burt (1955) has suggested is now randomly distributed amongst most of the social classes.

If this hypothesis is accepted, then the distribution of the constant factor, which has a range of four times the standard deviation (25.7 points in the I.Q score), would represent the distribution of "basic inherited intelligence" in the population, and would account for 27% of the variation in the I.Q score (Table 5).

There is no doubt that a major influence on the intellectual development of a child, is the social class into which he is born. If we accept Burt's suggestion, that inherited intelligence is randomly distributed throughout the population, then the benefit conferred by being born into an upper social class, must result from environment, a composite of home environment, nourishment, educational interest and culture. Burks (1928) studied the intelligence of children in foster homes in California, and she found that an exceptionally good environment could raise the I.Q. by 20 points. Illsley (1961) has dissected this influence, and he found that the character of the mother is the most important feature in determining the benefit a child obtains from his social class.

However, some benefit must accrue in pregnancy from better diet, more rest and more responsible interest in prenatal care, which is evident in the upper classes, and one would suspect that there is also a genetic loading in favour of a higher intelligence in the children of upper class families.

The simple correlation factor (Table 2) of the relationship between I.Q score and social class of the child is -0.247, which is less than that obtained by Nells (1951) who found a correlation of -0.35. However, this discrepancy may be explained by the separation in this analysis of the associated adverse feature of gravidity.

From Table 5, the relative influence of social class on the total variation in the I.Q score in the population, is seen to be 16%, and the difference in the I.Q score between a child born into class 1, and one born into class 5, could be as much as 16 points.

There is little evidence in the literature to support the findings in this analysis that advancing age in the mother is an advantageous feature, with regard to the child's intelligence. Indeed, most authorities agree that as maternal age increases, so does the risk to the baby, with the rise in the incidence of very severe mental defects, e.g. Down's syndrome, microcephaly (Tredgold and Soddy, 1963). This discrepancy may also arise from a confusion of correlations, since the simple correlation between age and gravidity (which is a disadvantageous factor) is very high (0.487); but this analysis has separated the effects of these two variables, and shows that the age of the mother alone, with other factors remaining constant, could account for 9% of the variation in the I.Q scoring of a population from which the very severely mentally retarded have been excluded.

Since no associated beneficial obstetric features can be found which would suggest that the influence of this variable is effective during pregnancy or labour, it is suggested that this beneficial effect is due to the separation within the social classes of groupings dependent on maternal age, in which the standard of living and emotional stability are directly related to the age of the mother.

The finding that the relationship between gravidity and I.Q score is linear, suggests that the first born, in spite of his increased risk of birth trauma, is not at a disadvantage to his siblings.

However, the distribution of the values of gravidity in the survey did not closely resemble the normal curve, and so the results of the analysis must be accepted with some reserve, and in fact, in Table 5, the results have been extended only to gravida 5, and this not only halves the apparent significance of this particular result, but brings

TABLE 6

Obstetric factor	Relative influence on the variation in the I.Q score of the population
Condition of child at birth	9%
Weight of child at birth	11.5%
Presence of pre-eclamptic toxaemia	4%
Maternal haemoglobin level	10%

the possible range of effect within that of normal experience.

The simple correlation factor between gravidity and I.Q score is -0.197 (Table 2) which agrees with the lower estimate (-0.19) of Burt (1946), but is below the estimate (-0.28) made by Thomson (1949) on group testing; here again the influence of social class may account for the discrepancy.

The results of the analysis suggest a drop of 2½ points in the I.Q score of succeeding children and that the influence of gravidity accounts for 13% of the variation in the I.Q score of the population. Using the same arguments when the influence of the age of the mother was discussed, it is suggested that there must be further sub-divisions in the social class groupings whose standard of living is dependent on the size of the family.

These three influences, most of whose effect will be after delivery, have a combined influence of over 30% on the variation in the I.Q scoring of the population.

The analysis has shown that the remaining 35% of the variation in the I.Q scoring of the population can be attributed to obstetric factors (Table 6).

Although Table 2 suggests that the length of neither the first nor second stage of labour could bear a significant relationship to the subsequent I.Q of the child, it does show that the relationship which the condition of the child at birth has with the length of the first stage is 0.119 and with the length of the second stage is 0.169. This would confirm that the length of labour has an effect in determining the condition of the child at birth.

Since the relationship of 0.177 between forceps delivery and the condition of the child at birth is not significantly different from the relationship of the length of the second stage of labour to the condition of the child at birth, and since "FORCEP" itself was eliminated as a non-significant variable, at an early stage in the analysis (Table 3), it would appear that it is the state of the baby when the application of forceps becomes necessary, rather than the instrumental delivery itself, which is the determining factor in the condition of the child at birth, and the subsequent development of the child's intelligence.

When the effect on the child of birth by caesarean section is considered, it can be seen from Table 2 that there are no significant simple correlation factors; but Table 3 shows that "CAESAR" is still an influence at the 5% level of significance, but that the child's condition at birth is much more significant.

In Table 4 both the variables for the lengths of the first and second stages of labour are eliminated even before the 10% level of significance is reached; this would suggest that the condition of the child at birth is due more to other obstetric features than to the actual length of labour.

The possibility of foetal damage due to abnormalities in the placenta is often put forward but evidence for this is scanty. Indeed, it has been suggested that there is a marginal safety factor of 50% in the normal placenta (Browne and Veall, 1953), and this may be the reason that the gross placental changes, both functional and histological, in hypertension and pre-eclamptic toxaemia, appear to have such a small effect on the foetus.

Hypoxia is the normal state during the birth process (Walsh and Lindenberg, 1961). Smith (1948) has shown that the neonate has an increased resistance to asphyxia but that anoxia occurring before, during or after birth is accompanied by the likelihood of permanent intellectual impairment.

Therefore, to help the foetus survive pregnancy and labour, there are two protective features; the large safety margin of placental function, and the innate resistance of the human foetus to asphyxia before the onset of spontaneous respiration.

It was Darke (1944) who suggested that it is the combination of degree and duration of anoxia which is damaging; and so chronic pre-natal hypoxia due to placental insufficiency - demonstrated by the degree of dysmaturity of the neonate - is a significant factor.

Sir Dugald Baird (1960) expressed anxiety when he discussed the problem of the sudden unexpected foetal death in elderly primigravidae. The postmortem findings in these cases were of asphyxia, due presumably to placental insufficiency, with the margin between adequate compensation and sudden decompensation very narrow, allowing, in the present state of medical knowledge, no warning of the imminence of foetal death.

Those conditions which act only during labour, such as the lengths of first and second stages of labour, and delivery by forceps, appear to have no direct effect on the final I.Q score; it is therefore suggested that the condition of the child after delivery, which <u>has</u> an effect on the subsequent I.Q. is more dependent on the amount of placental reserve before labour begins, than on labour itself.

Heyns (1962) has compared the danger of definite anoxia when the effects can be gross, with hypoxia where the physical and mental effects are imponderable. He suggests that the improved foetal oxygenation before the onset of labour by the use of decompression technique, prevents loss or impairment of the neurones or their inter-communications - the very anatomy of intelligence. In his series, the children born by the decompression technique did appear to be more intelligent.

Ernhart (1960), after finding that of a group of three-year-old children with a history of neonatal anoxia compared unfavourably with a control group, suggested that an attempt should be made to measure "anoxia", since the current criteria are so crude. It would be useful for future statistical surveys, if the method of measurement would cause the values to be distributed as a normal curve, or nearly so.

Considering only the condition of the child immediately after delivery, as assessed by the midwife, the influence of this factor could account for 9% of the total variation in the I.Q scoring of the population.

Tredgold (1963) suggests that as many as 20% of babies suffer some degree of asphyxia at birth. In this survey the percentage was just over 13%.

The condition of the child at birth is therefore of considerable importance to the subsequent development of the child's intelligence, and recently there have been attempts to detect foetal hypoxia before birth. The electrocardiographic recording of the foetal heart rate during maternal exercise would show any lowering of foetal tolerance, and this would be a measurement of placental efficiency. The use of direct amnioscopy to inspect the colour of the amniotic fluid to determine whether meconium (a late occurrence in foetal distress) had already been passed was introduced in 1962 and recently it has been shown that assessment of the acid-base status of the foetus is a practical possibility, by analysing either the amniotic fluid or a foetal blood sample.

The problem of assessing the influence of prematurity on the intelligence of the child is made more difficult by the use of inaccurate terminology. More use should be made of the term "dysmaturity", when the weight of the baby does not correspond to its gestational age, to contrast with "immaturity".

Although in Table 2 there is little direct correlation between the variable "weight of child at birth" and the I.Q score, the correlation with the "duration of pregnancy" is very high (0.363). Karn and Penrose (1951) found this correlation to be 0.4.

With the elimination of "duration of pregnancy" at an early stage in the analysis, emphasis is laid on the "weight of the child at birth" as the significant variable - stressing the importance, in the determination of the eventual I.Q. of maturity by weight rather than gestational age.

A recent survey by Barker (1966) has found that low intelligence is associated with both a slower rate of intrauterine growth, and birth before the thirty-eighth week. Drillien (1964) has implied that the baby who is small (low birth weight) merely because of immature birth may be subjected to influences after birth similar to those affecting the dysmature foetus in utero, with the same long-term results, and he has found support in the findings of Widdowson and McCance (1960) and Wigglesworth (1966).

However, Baird (1959) and Fairweather and Illsley (1960) have inferred from their findings that dysmaturity, rather than immaturity, is more likely to have an adverse effect on the development of the intelligence.

This argument could possibly be settled by including in a multiple regression analysis a variable representing the weight-maturity ratio of the baby. But the interrelationships between the weight of the baby and the parity of the mother, and the weight of the baby and the social class of the mother may detract from the usefulness of the result.

As dysmaturity increases in degree, the oxygen saturation in the cord blood decreases (Sjöstedt et Al, 1958), suggesting that dysmaturity results primarily from placental insufficiency, although recent work has shown some similarity between the dysmature child and neonatal conditions suggestive of an immunological aetiology (Scott, 1966).

The close relationship between birth weight and placental weight and chorionic villous surface area, applies whether the pregnancy is mature or immature, normal or complicated by hypertension or by the dysmaturity syndrome (Aherne and Dunnill, 1966 A). These findings suggest that there is a balance between the transfer area of the placenta and the mass of the foetus, to the detriment of the foetus, if the placenta is unduly small. Browne and Veall (1953) claimed that in a normal placenta there was a functional safety margin of 50%; it can therefore be presumed that, if the placental function is reduced, due to some obstetric complication, such as pre-eclamptic toxaemia, by 50%, then the foetus will fail to gain weight, and may even lose weight in utero (Gruenwald, 1964).

There is no accurate method of assessing the mass of the foetus in utero; some obstetricians judge that when the total maternal weight fails to increase near term, then that is the optimum time for delivery, but this may be obscured by the weight gain of progressing pre-eclamptic toxaemia. Perhaps the use of bi-focal ultrasonics will be developed in this field.

If it were known that the weight of the foetus was increasing satisfactorily and that its acid-base balance was in equilibrium, it could then be assumed that the placental function must be efficient and that there was no danger of sudden foetal anoxia.

In this analysis, it has been shown that the weight of the baby at birth is a highly significant factor in the determination of the subsequent intelligence of the child, accounting for just over 11% of the variation in the I.Q score of the population (Table 5).

The fact that pre-eclamptic toxaemia was included by the analysis and that hypertension was eliminated, at the 5% level of significance, supports the opinion of Klatskin (1964) who, after she had reviewed previous work, considered that pre-eclamptic toxaemia must be a major, and hypertension a minor obstetric stress.

However, Solnitzky (1964) stressed his finding that, in the absence of prematurity, toxaemia was not associated with mental retardation, and Barker (1966) has suggested that the recognised complications of pregnancy, such as pre-eclamptic toxaemia, play little part in the aetiology of low intelligence.

However, Butler and Bonham (1963) found that the risk of perinatal death was increased by 25% in hypertension and doubled in severe toxaemia. Also, the evidence from investigations into placental function shows that the intervillous blood flow in late pregnancy is slowed in cases of pre-eclamptic toxaemia, even to 30% of the normal, and therefore the effect of pre-eclamptic toxaemia must be to reduce placental efficiency.

Table 4 shows that at the 5% level of significance pre-eclamptic toxaemia is a significant variable, and its relative influence is displayed in Table 5 as 4% of the total variation in the I.Q scoring of the population.

Other obstetric complications such as accidental haemorrhage, which has a perinatal mortality risk of seventeen times the normal (Butler and Bonham, 1963), must have an adverse effect on the development of the child's intelligence, more through the disturbance of the placental circulation than through the methods of treatment. Only by analysing a very large sample could their relative influence be estimated.

With a maternal haemoglobin level of below 60%, the risk of foetal death is almost doubled (Butler and Bonham, 1963). In a recent paper, Barker (1966) found a "small increase" in the incidence of anaemia during pregnancy which preceded the birth of children with I.Qs of below 80, even after adjustment for social class.

The estimated incidence of iron deficiency anaemia in pregnancy varies widely; Butler and Bonham (1963) reported that just under 15% of pregnant women had a haemoglobin level below 70%. But recently Fielding (1965) has shown that of the women who are not clinically anaemic, 35% have no iron stores. This means that for 50% of women, the increased demand for iron in pregnancy, must lead to iron deficiency anaemia.

Although Table 2 gives a simple correlation factor of 0.137 between maternal haemoglobin level and the I.Q of the child, there are no significant correlations between the haemoglobin level and any of the last three variables discussed, the effects of which extend throughout pregnancy.

It might have been thought that a high level of maternal haemoglobin would improve placental efficiency, and that this would be reflected in a high relationship between the maternal haemoglobin level and the weight of the baby, but this is not so. This suggests that placental efficiency is dependent on factors, other than the maternal haemoglobin level but, given a certain level of efficiency of the balance between placenta and foetus, a high maternal haemoglobin level could be a protective factor against any threat of intermittent hypoxia, occasioned by excessive maternal exercise or placental mishap.

If this were so, and a high maternal haemoglobin level did protect the baby against hypoxia, then it should protect the baby to some extent against the hypoxia of labour. But again there is no simple correlation between maternal haemoglobin level and the condition of the child after delivery to support this.

The significance of the final analysis in Table 3 must be judged critically, since it is probable that in this analysis the "HB" variable has absorbed some of the influence of the social class variable which had not been included in the analysis.

However, in Table 4, the influence of the maternal haemoglobin level continues to be significant even at the 0.1% level of significance (a very high degree of significance), and Table 5 shows that the relative influence of maternal haemoglobin level accounts for 10% of the variation in the I.Q scoring of the population.

It could be suggested that maternal haemoglobin level is a measurement of the state of the mother's nutrition, and that the deficit in "iron-deficiency anaemia" is not just in iron but in other foetal requirements, such as the essential amino-acids, vitamins and trace elements. But this standard of nutrition is more likely to have been included in the variable "social class".

In 1962 Heyns showed that increased foetal oxygenation in pregnancy and labour, using the abdominal decompression technique, resulted in the birth of children who appeared to be above average in intelligence. It is now suggested that a high maternal haemoglobin level affects the foetus in a similar manner, bringing an increased oxygen supply to the foetus, and so protecting the developing neurones and their inter-connections.

CONCLUSION

Doubt has been cast (Douglas, 1960) on the utility of the method of controlled comparison in socio-medical studies. So often the findings of a survey are made suspect by a failure to match the controls in all respects.

By the use of multiple regression analysis it has been possible to determine the relative influence of the obstetric factors which might affect the eventual intelligence of the child. These factors are multiple and interacting and some of them, such as the age, gravidity and social class of the mother, continue to operate after the birth of the child, whilst others, such as placental efficiency, anoxia and maternal haemoglobin level, are effective only during the pregnancy.

Whether the constant factor in the regression equation represents basic inherited intelligence, is open to doubt, but the extension of the regression equation, to establish the relative influence of each variable on the variation of the I.Q scoring in the population, merits further study.

In an attempt to rationalise the findings it has been found useful to develop the theory that foetal anoxia is the major obstetric hazard, and that obstetric complications affect the child's potential intelligence mainly by reason of the degree and the duration of the hypoxia or anoxia which they cause.

The findings of the analysis suggest that of the variation in the I.Q scoring of the population

1. Inherited intelligence accounts for 27%;
2. Social class for 16%;
3. Gravidity of the mother for 13%;

4. Weight of the child at birth for 11½%;

5. Maternal haemoglobin level for 10%;

6. Age of mother at birth of child for 9½%;

7. Condition at birth (reference to anoxia) for 9%;

8. The presence or absence of pre-eclamptic toxaemia for 4%.

At the present stage of medical knowledge little control can be exerted over most of these factors, but if only the variation due to maternal haemoglobin level is considered, then in view of the incidence of iron-deficiency anaemia in pregnancy, it will be recognised that there is at present an enormous, unnecessary intellectual wastage in the community.

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APPENDIX I

THE CASE CARD

Haemoglobin/I.Q Survey

Surname ____________________

Other Names ____________________

M | F   D. of B. ____________

Address ____________________

____

Born at: Home / St. Mary's / Other

Hospitalisation: Social / Medical

Maturity	Length of 2nd stage
Age of Mother	Blood group
Parity of Mother	Hb 1-12 weeks
Condition at birth	Hb 13-28 weeks
Type of delivery	Hb 29-T
"At risk"	Hb Post-natal

Primary School ____________________

Secondary School ____________________

I.Q. ____________________

Length of 1st stage ____________________

Weight of baby ____________________

Occupation of Father ____________________

Social class ____________________

APPENDIX II

THE PRINT-OUT OF THE DATA

IN THE OTHER COPY