OCCASIONAL HUNDREDS 8

The formative years

Birth, family, and development in Newcastle upon Tyne

GERALD NELIGAN
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HANS STEINER

Published for the
Nuffield Provincial Hospitals Trust
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PREFACE

In this book we are reporting some of the results of two surveys which have been carried out with a minimum of paid research staff, using information supplied to us by a very large number of other people. We are aware that those who were in direct contact with the children at different stages (midwives, health visitors, school doctors and nurses, teachers) were often contributing a disproportionate amount of time and effort on top of an already heavy load of routine work, and we are particularly grateful to them, but they are far too numerous to be named individually (there are between three and four hundred of them) and we can only express our thanks through the administrative hierarchy, whose consent and co-operation formed the corner-stone upon which the whole structure was built.

When our Community Study began in 1960, Dr. R.C.M. Pearson was Medical Officer of Health for the City of Newcastle upon Tyne and Dr. Shirley Livingston was Child Welfare Medical Officer; Miss F.E. Hunt was Chief Nursing Officer and her Deputy was Miss A.Y. Sanderson. In 1964, when our children were starting to enter the schools, Mr. G. Squires was Director of Education for Newcastle upon Tyne and the Senior School Medical Officer was Dr. H.S.K. Sainsbury. In 1969, when we were planning our final round of data collection, Miss A.C. Emerson was Deputy Superintendent Health Visitor/School Nurse and Mr. C. L. Mellowes was Director of Education for the County of Northumberland and made it possible for us to test the children who had moved into his area. We are most grateful to all those named, both for their help as individuals and for the help so generously given to us by their successors (in some cases) and their staffs (at all levels and in all cases). The Heads of all the private schools in or near the City also helped us in a similar way and we wish to express our thanks to them.

Our postal follow-up of the children who moved away from our area during the first five years of life was

made possible in the first instance by the generous help we received from the Health Department of almost every Local Authority in the British Isles, and many consultant paediatricians. When more direct approaches failed, the tactful and enthusiastic co-operation of Miss H. Marshall and her staff in the Family Allowances Branch, Ministry of Social Security, enabled us to obtain useful information from the families of over 500 'lost' children.

The data of the Newcastle Maternity Survey were made freely available to us through Professor J.K. Russell and his colleagues, and the early stages of the analysis of our own data were made possible by the expertise and unstinted help of the late Professor D.G. Millar. The planning of our programme of objective tests was carried out by a small working party of psychologists (Dr. R.D. Savage, Mrs. Jane Nolan and Dr. L.F. Mills) and we were much helped over individual problems by colleagues in London (Professor J. Tizard, Dr. W. Yule and Professor M. Rutter in the case of the behaviour inventories). General epidemiological and statistical advice has been freely given by Dr. S. Brandon; by Professor A.M. Thomson and Mr. W.Z.Billewicz of the Medical Research Council's Reproduction and Growth Unit; and by the late Professor Herbert Birch, Dr. Stephen Richardson and Professor Ernest Gruenberg on pastoral visits from New York.

Financial provision for our small research staff and running expenses has come from a succession of different sources, to all of whom we are most grateful. They are:- The Research Committee of the Board of Governors, Royal Victoria Infirmary, and that of the Newcastle Regional Hospital Board; The Association for the Aid of Crippled Children, New York; and The Nuffield Provincial Hospitals Trust, whose contribution has been the major one, particularly in making possible the analysis of the data and its preparation for this publication. The machinery used for the analysis has been made available in the Computing Laboratory, University of Newcastle upon Tyne (Professor E.S. Page).

Our Hospital Study's perinatal data were derived from the routine clinical records made by a series of paediatric residents and midwives in the Princess Mary Maternity Hospital, to whom we are grateful for the high standard of this basic information. We would also like to thank Dr. E. Ellis and Mr. R.G. Chaytor for their help with the confirmation of the diagnosis in the cases of cerebral palsy and of deafness.

Too many individuals have helped with the different stages of data collection and processing during the past 14 years for us to name them all, but we feel particularly indebted to two of them. Mrs. Julia Stott was continuously involved in this work for a period of eight years; and Mrs. Morella Marr, MA, in addition to being respon-

sible for these aspects of the work over the past four and a half years, has done the final typing upon which the quality of the end product directly depends, and has also taken on the role of literary editor, to the great benefit of both text and tables.

INTRODUCTION

More than a century ago, Little (1843-4) described the whole range of handicapping conditions of the central nervous system which could be caused by adverse factors acting before, during, or soon after a child's birth. He ascribed certain cases of 'universal spasmodic contraction...an impairment of intellectual powers...even complete idiotcy...great irritability of temper...epilepsy' to the effects of 'premature birth' or of 'asphyxia following birth at the full period of gestation'. He also suggested the possible mechanisms, namely that the premature birth and the subsequent handicap might have a common origin ('derangement of the health of the parent...directly impaired the nutrition of the foetus... and the healthy development of the nervous system'), or that the one might cause the other ('...an infant prematurely born is, although in a normal stage of development, inadequately prepared to contend against the operation of external agents'). He even went so far as to suggest that, sometimes, associated social factors may be of overriding importance ('...in some instances the weakness of intellect has appeared to result less from permanent injury to the brain than from the want of sufficient training and education after its recovery from the severe physiological shock it had received').

Today, all these ideas are accepted as almost self-evident; they are of particular concern to all who are interested in studying or assisting the development of children, and insofar as the stated adverse factors are under the control of clinicians, at least in theory, the whole subject is of particular concern to those who are medically responsible for the care of pregnant women and newborn babies. These obstetricians and paediatricians are becoming increasingly concerned about the quality rather than the mere quantity of the survivors of the perinatal period (Report of the Expert Group on Special Care for Babies, 1971).

The concept of a 'continuum of reproductive casualty'

proposed by Lilienfeld and Pasamanick (1955) has added considerably to these anxieties. Their idea, which they proposed as 'a conceptual framework for further research', was described as follows:-

> 'The pattern of factors such as complications of pregnancy, prematurity, etc., which influence infant loss, seems to behave in a similar manner with regard to cerebral palsy. On the basis of these related patterns it was possible to postulate the existence of a continuum of reproductive casualty with a lethal component consisting of abortions, stillbirths, and neonatal deaths, and a sublethal component consisting of cerebral palsy and perhaps other related conditions.'

Not only does this seem possible, but, accepting the idea in the spirit in which it was proposed, we can extend it to cover the possibility that adverse factors may give rise to milder (and potentially much commoner) impairments of function, which could turn out to be even more important than the clinically recognisable forms of brain damage (Neligan, 1970).

If we try to look for the practical implications of these quotations for clinical, administrative and social policies, we clearly need to know the numerical facts concerning the relationships between various types of adverse antecedent factors and the quality of the survivors of the first month of life. Such numerical facts might also make it possible to identify antecedent factors whose effects upon the survivors are favourable. The tools which we can use to identify and analyse such facts, which were not available to Little and were only beginning to be available to Lilienfeld and Pasamanick, are the epidemiological and statistical methods of assessing the significance of numerical data, together with the more sensitive and objective methods of assessing the neurological, intellectual and emotional development of children which have been devised during the past half century. In this book we are reporting some of the results of two surveys, which we have carried out in Newcastle upon Tyne during the past 14 years with the purpose of making available relevant numerical facts on which practical policies can be based.

Other Surveys

Before describing our methods and results, we would like to refer briefly to previous work in the same field. In view of its potential practical importance, it is not surprising that this subject of the relationships between antecedent factors and the subsequent development of children has been studied by many observers, and many different approaches have been used. These fall

into two main groups, depending upon the type of criteria used to define the populations studied. The criteria may be medical, in terms of an adverse antecedent factor such as neonatal asphyxia or low birth weight, or in terms of an unfavourable outcome such as cerebral palsy or mental defect; or they may be geographical, in terms of residence within an administratively defined community such as a country or a city within which it is possible to study a whole birth cohort or age-group, or a representative sample.

Studies of medically defined populations have been much the more numerous, and include all the well-known reports of Pasamanick and his associates (see Pasamanick and Knobloch, 1960). They have the advantage in terms of economy of effort, since all the work of investigation and analysis can be concentrated upon children whose condition is directly relevant to the (relatively limited) questions being asked. It is practicable, therefore, to study large numbers of children in the defined abnormal groups by detailed and specialised techniques. However, the general applicability of studies of this type is, unfortunately, strictly limited, and the inherent weaknesses have been discussed by MacMahon and Sowa (1961).

Studies of geographically defined populations have been much less numerous, but they provide the only valid basis for drawing more generally applicable conclusions concerning the incidence and relative importance of the abnormalities identified, and for defining the fields in which preventive action is most likely to be rewarding. Valid comparison groups are also much easier to specify and identify. However, such studies pose a very difficult problem of deciding how many children to include in the population studied. The difficulty is particularly great in the case of a prospective, longitudinal study, since neither the number of children included, nor the depth and scope of the information collected during the earlier stages of their lives, can be stepped up if they are shown to be inadequate by the later findings. To ensure a reasonable chance of including an adequate number of children for purposes of detailed statistical analysis, the investigator would like to enroll a very large number of children. At the same time, to ensure a reasonable chance of detecting minor abnormalities, he would like to apply a large battery of sophisticated tests to each child. In practice, most investigators who have planned this type of study have clearly decided that it is beyond the effective capacity of available technical and intellectual resources to attempt to achieve these two objectives simultaneously, however desirable each may be individually. The compromises which have been adopted are the result of making one of three choices:-

(a) To study a very large number of children (over 50,000) using the relatively superficial data available through the routine medical and educational services of the community. Examples are the Birmingham studies of Knox and Mahon (1970) and McKeown and Record (1971).
(b) To study a very small number of children (under 1,000) using relatively sophisticated procedures carried out by research staff specifically for the purpose of the particular investigation. An example is the Kauai study reported by Werner, Bierman and French (1971).
(c) To study an intermediate number of children (between 5,000 and 20,000) using a combination of the two types of data. Examples are the British studies by Douglas (1964) and by Davie, Butler and Goldstein (1972), and the Aberdeen study by Birch et al (1970).

We cannot conclude this brief account of other surveys without mentioning one which does not fit into any of the above categories, but which has deployed resources on a scale which probably exceeds the sum of all the other surveys in this field. The investigators who planned the Collaborative Project on Cerebral Palsy, Mental Retardation and Other Neurological and Sensory Disorders of Infancy and Childhood decided to combine the two main approaches in one survey by covering a very large number of pregnancies and surviving children (upwards of 50,000) and studying them prospectively in very great depth and detail (so that each case produced 'several thousand bits of information', according to Weiss (1966)). They did not use overtly medical criteria for selecting their study population, but since all the children were delivered in one of 14 very heterogeneous collaborating institutions, any findings would require to be reviewed with critical caution before being accepted as more generally applicable to other populations. Moreover, the sheer mass and complexity of the data have caused predictable and almost insurmountable difficulties for those attempting to analyse and understand the findings (Berendes, 1966; Weiss, 1966).

Two other published surveys are so relevant to our own that they must be mentioned here, even though their primary objectives were quite different. Firstly, the longitudinal study of 'A Thousand Families in Newcastle upon Tyne' (Spence et al, 1954) was intended as 'a contribution towards understanding the needs of families in sickness and in health' (Miller et al, 1960). It had established the fully integrated approach to epidemiological research involving families in our City, combining the efforts of members of the University's Department of Child Health and of the City's Departments of Health and Education, which has formed the essential foundation of our main survey, begun nearly 13 years later. Secondly, in their cross-sectional study of all the approximately

3,500 children aged 9-11 years living on the Isle of Wight, Rutter, Tizard and Whitmore (1970) were primarily interested in assessing the prevalence and service implications of specific handicapping conditions, but interesting comparisons with our population are again possible, because the same three group tests of intellectual and educational performance were administered in the two studies to children in the same age range, and the same behaviour inventories were completed by teachers and parents.

Our Surveys

We have carried out two surveys:-

A. A Community Study of a geographically defined population, the Newcastle Survey of Child Development. This was planned as a longitudinal prospective study of a three-year birth cohort (1960-2) concerning which obstetric and perinatal information was available through the Newcastle Maternity Survey (Russell et al, 1963). Our study comes into the category described as choice (c) above; we planned to enroll the approximately 14,000 survivors of the first month of life and follow them through to school age, using the routine medical and educational services as our main source of information, but also obtaining some extra information through a very small research staff. This study is intended to give an overall picture of a superficial type, but suitable for comparison with other geographically defined populations, and for more general application. This picture is built up in Chapters 2-9 of this book: a brief account of the population studied and the methods used is included in Chapter 1.

This study also acts as a launching pad for much more detailed studies of specific problems, using samples of children selected by appropriate criteria based upon data available for the whole three-year cohort. Not only does this have the advantage of enabling the relationship of the sample to the whole population to be clearly specified, but it also means that data about the earlier stages of the children's development is available. The results of these more detailed, and more limited, studies are not included in this book, but the problems under investigation are:-

(a) The effects, at school age, of variations in the duration and rate of intrauterine growth, and
(b) Subsequent development of speech retarded children, and
(c) Normal dimensions of temperament in infant school children, and
(d) Normal dimensions of behaviour in infant school children, all by Dr. I Kolvin and colleagues in the Nuffield Child Psychiatry Unit.

(e) Medical and social factors in children with possible brain damage, a Special Study within our own Newcastle Survey of Child Development.
(f) Causes of short stature (Lacey and Parkin, 1974 a) and b)), and
(g) Causes of obesity in childhood by Dr. J.M. Parkin and colleagues in the University Department of Child Health.
(h) Physical growth through puberty by Professor A.M Thomson and colleagues in the Medical Research Council's Reproduction and Growth Unit.

B. A Hospital Study of a medically defined population. This was designed to enable us to study the sequelae of some of the severe perinatal clinical problems which involve obvious risks of brain damage and form an important part of the work of a Special Care Nursery, and concerning which we did not expect to obtain adequate information from our Community Study. The adverse factors in which we were interested were not sufficiently common, nor were they sufficiently uniformly recorded or treated, for this to be likely. We planned, therefore, to enroll all the survivors of these relatively rare adverse factors born in our hospital over a period of years, which eventually extended from January 1961 to May 1970, and to assess their neurological and intellectual development, if possible up to the age of about 8 years. The primary clinical assessment of each child, repeated at successive 'key' ages wherever possible, was carried out by one observer (H.S.), who obtained a second opinion or a specialised assessment from colleagues when indicated. For the purposes of this book, we have confined our attention to sequelae which could be described as moderate or severe handicaps. A brief account of the population studied and the methods used is included in Chapter 1: the results are summarised in Chapter 10.

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1

OUR CHILDREN AND METHODS

The children enrolled into our Community Study were all those born to mothers resident in the City of Newcastle upon Tyne between 1 January 1960 and 31 December 1962, wherever the birth occurred, who survived the first four weeks of life. For our present purposes, we have confined our attention to the 13,203 children in this three-year birth cohort who were singletons (in the sense that they were the only viable product of the pregnancy) and legitimate (in the sense that the mother was a married woman).

Basic information concerning each mother's social background, past obstetric history and pregnancy, and concerning each child's birth and condition during the first month of life, was made available by the Newcastle Maternity Survey. The methods used in that survey and the social and economic background of the population concerned, are briefly described by Russell et al (1963). The original observations were made by the midwives and doctors clinically responsible for each mother and child, but the recording of these observations was to some extent standardised in accordance with the requests of the small survey research staff. Our Newcastle Survey of Child Development, whose aim has been to follow as many as possible of these survivors of the neonatal period to an age when their quality could be reliably assessed, has relied on comparable methods for the basic epidemiological study whose results are reported in this book. Information has been supplied by the health visitors, school nurses and doctors, and teachers in direct contact with the children at successive stages, in a standardised form and in accordance with defined criteria wherever possible; the small Survey staff has collected, processed (including scoring where indicated) and analysed the data.

Number of Children

The numbers of children for whom the specified types of

Table 1.1

Numbers of children in Newcastle Survey of Child Development for whom information at different stages is available

Type of Information		Number
Singleton legitimate survivors of first month (born 1960-2)		13,203
First Year Health Visitor Proforma		12,259 (92.9%)
Second Year Health Visitor Proforma		11,632 (88.1%)
Third Year Health Visitor Proforma (1961-2 births)		7,212
Located in school at 5 years		9,626 (72.9%)
(Drawing Test available for	8,819	
School Entry Medical Proforma for	9,398)	
Located at age of 10 years		9,019 (68.3%)
(Non-verbal IQ test available for	8,413	
Behaviour inventory (teacher) for	7,900	
Height record for	6,426)	
Some follow-up information available from all sources (including postal)		12,906 (97.8%)

Table 1.2

Numbers of children in our Hospital Study for whom different types of information are available

Type of Information		Number
Survivors of one of our six very severe adverse factors (born January 1961-May 1970)		218
Adequate information at mean age of 4.0 years		211 (96.8%)
(personally examined by H.S.	181	
other information including postal -	30)	
Deaths before the age of 4 years		6
(4 quadriplegic and defective		
1 'cot' death at 9 months, 1 pneumonia at 2 months)		
Controls (birth weight >2.5 kg, vertex or Caesarean delivery)		200
(50 examined at 10 months, 50 at 18 months,		
50 at 4 years and 50 at 8 years)		

information are available at successive stages of our Survey are shown in Table 1.1. It is obvious that, even at the same stage of the Survey, there are differences between the numbers of children for whom different types of information are available. For instance, although we located 9,626 children in normal schools accessible to us when the children were 5 years of age, we received Drawing Tests for only 8,819, because some children were absent from school on the day the test was administered by their teacher and attempts to catch them on another occasion were unsuccessful. We received Medical Proformas for a larger number, and in the cases for which we had no Drawing Test (and so no intellectual assessment) it was helpful to know that the children were considered suitable for education in a normal school and were free of any clinically recognisable handicap, when calculating the prevalence of severe mental subnormality at the age of 5 years.

This problem of differences in the numbers of children for whom different types of information are available is a recurrent one in our data, and inevitable in a longitudinal prospective study of this kind. We would like, therefore, to describe our approach to two aspects of this problem which are relevant to a full understanding of much that we have written in later chapters.

Firstly, there is the question of which numbers of children to use when analysing and reporting the effects of a number of different antecedent factors upon a particular outcome, or those of a particular antecedent factor upon a number of outcomes. In the former situation, we have always used the total number of children for whom we have information concerning all the antecedent factors shown in the particular table or figure. Where there is only one antecedent factor, of course, we have used all the children for whom information is available concerning both this factor and the outcome. The number of children is not stated in most instances, to avoid confusion, but in that case it can be assumed to be a large number. The number is stated where it is small (less than 100 in any cell) or where there is a particular point to be made. An example of this is the direct comparison between the effects of birth weight and those of gestational age in Chapter 7. We have reliable information concerning the latter for only about 84 per cent of our population, as compared with nearly 100 per cent in the case of birth weight; since we are making a direct comparison, however, we have confined our analyses to the 3,603 boys for whom both types of information are available (Figures 7.2 and 7.3). When we come to look at the combined effect of social class and birth weight in Figure 7.4, however, the number available falls to 3,281 boys, because in 322 we did not know the father's occu-

pation, or the parents were separated or divorced by the time the child was 5 years old. For the analyses of variance in Chapter 9, we have used only those children for whom information is available concerning all of the factors used as independent variables in each of the analyses.

Where the purpose is to illustrate the effect of a single factor upon a number of different outcomes, however, as in Figures 7.6 and 8.2, we have used all the children for whom we have information concerning each of the reactions shown.

The second major problem concerning variations in the numbers of children available for different analyses arises from the inevitable losses from the original population at successive stages of the investigation. This problem is particularly severe if the investigation is planned in the interests of economy, as ours was, to test directly only those children who remain accessible in or near their community of origin at the relevant ages. Even though the same quality of information cannot be obtained concerning those who have moved away, it seemed to us to be of vital importance to obtain simple information about them, by postal follow-up if necessary, which would at least enable us to categorise them as presumably normal, definitely abnormal or of doubtful status. The fact that we were able to achieve this level of information (or better) for 97.8 per cent of our enrolled population seems to us to provide the justification for drawing meaningful conclusions concerning the children whom we were able to test in accordance with our programme.

Methods of Assessment

The methods used for obtaining the data specifically for the purposes of our Survey on which this book is based were as follows:-

(a) Delay in establishing regular active respiration after birth. The midwife or doctor was asked to record whether this was less than 1 minute, 1-4 minutes, or 5 minutes or longer. In the latter group, the actual time was recorded, together with any methods of resuscitation employed, and the original records were scrutinised by Survey staff in the case of hospital deliveries.

(b) Developmental milestones were recorded on the Health Visitor Proformas in accordance with written definitions (Neligan and Prudham, 1969). These proformas also included information about health and handicaps, and the Third Year Health Visitor Proforma (for 1961-2 births only) included certain extra social information.

(c) The Drawing Test at 5 years was administered as a

group test by the class teachers during the children's first term in school. It included the Goodenough Draw-a-Man Test, which was scored by Survey staff and an IQ calculated in accordance with the original instructions of Goodenough (1926); and the children were asked to copy a circle, a square and a diamond from which we derived a Figure Copying score with a range of 0-9 points, by our own modification of the Stanford-Binet procedure.

(d) The School Entry Medical Proforma, completed by the nurse or doctor at the routine medical examination in City schools (or by Survey staff in the case of private schools) included the father's occupation at that time, the number of immunisation procedures the child had had, and his height, in addition to routine medical information.

(e) The non-verbal IQ at 10 years was derived from the National Foundation for Educational Research's (1965) Test 5/BD, administered as a group test by the class teachers (or by Survey staff in private schools) and the scoring checked by Survey staff in accordance with the manual. We were able to identify and test 655 of our original population in schools in Northumberland County at this stage, to add to those still living in the City.

(f) The behaviour inventory at 10 years was the Behaviour Inventory Scale B(2), completed by the children's teachers for almost all the children for whom a non-verbal IQ score was available. The inventories were scored by Survey staff in accordance with the instructions of Rutter (1967).

(g) Height at 10 years was measured specifically for the purposes of our Survey by the City's school nurses.

Appendix

In the interests of clarity and simplicity, we have not included in this book much detailed information which would be a matter of concern for only the professional epidemiologist or statistician. This information is available in the form of an optional Appendix, on request to the senior author at the Princess Mary Maternity Hospital, Newcastle upon Tyne. It includes the proformas employed, the accompanying instructions, the raw data on which all the figures and tables in the book are based and a description of the statistical methods used in the various analyses, together with data concerning the changes in the social class structure of our population at successive stages due to selective migration.

The Hospital Study

The index cases were survivors of one or more of our six very severe adverse factors (cardiac arrest; delay

of more than 20 minutes in establishing regular respiration; birth weight of 1,360 g or less; apnoeic/cyanotic attacks; convulsions; cerebral irritation requiring sedation for more than 7 days). They (and the controls) were identified, and basic data were extracted, from the routine neonatal records of the Princess Mary Maternity Hospital. The index cases were examined, many of them repeatedly, at ages determined by clinical indications or by practicability. The tests used to obtain the results reported in Chapter 10 were:-

(a) Standard clinical/neurological examination.
(b) Assessment of the development of the gross motor system (Milani-Comparetti and Gidoni, 1967).
(c) Stycar Hearing Test (Sheridan, 1958).
(d) Stycar Vision Test (Sheridan, 1960).
(e) Denver Developmental Screening Test (Frankenburg and Dodds, 1967).

The controls were examined, each on only one occasion, at the ages stated in Table 1.2, by the tests appropriate to their age.

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2

SIGNIFICANT PERINATAL FACTORS

Since we are primarily interested in the practical benefits which can be achieved by identifying the factors which have an adverse effect upon the quality of our community's children, we are particularly interested in those factors which can most easily be modified by deliberate changes in policy. Clinical practice can be so modified far more easily and predictably than can biological or social factors. For this reason, and because our whole involvement in the subject has a clinical bias (two of us are paediatricians, the third a statistician), we would like to start by trying to identify some adverse factors which seem particularly likely to impair the quality of our community's children, and which could easily be modified by deliberate changes in clinical practice if indicated.

The extent and the critical nature of the physiological and anatomical changes which occur around the time of birth, and the obvious validity of the observations made by Little (1843-4) and his many successors, focus attention upon perinatal factors as the most likely causes of brain damage on a significant scale; and because, in our community, the processes associated with birth are almost invariably supervised by a midwife and a doctor, they are accessible to clinical intervention. We have, therefore, looked for perinatal factors which could cause brain damage, and which could be modified by deliberate changes in clinical practice.

In deciding which particular perinatal factors are most likely to cause brain damage on a significant scale among the survivors of the neonatal period we have been influenced by the 'continuum of reproductive casualty' concept of Lilienfeld and Pasamanick (1955). We have looked for factors associated with a high mortality rate in our population (and with pathological evidence of brain damage in those who die) as being the most likely to cause sublethal but permanent brain damage amongst the survivors. Our choice has been much assisted by the

fact that an analysis of the causes of the perinatal deaths in the population from which our Community Study population was derived was published by Fairweather et al (1966). We have focussed our attention upon the causes of neonatal death rather than stillbirth as being more likely to have relevance to the condition of the survivors. Their Table III shows that the commonest pathologically identified causes of the 253 neonatal deaths were:-malformation (16.2 per cent); hyaline membrane disease (15.0 per cent); trauma (8.3 per cent); and anoxia (8.3 per cent). The first of these offers little scope for improving the quality of the survivors of the neonatal period by improvements in perinatal clinical management at the present time. The second is basically a problem of pathophysiology of the lungs, without any specific threat to the brain; it is already under intensive study, therapeutic attack and subsequent surveillance of the survivors by sophisticated techniques which are outside the scope of our study. The two next commonest causes of death look much more promising from our point of view.

The deaths attributed to trauma were commonly associated with 'abnormal delivery', a factor which is potentially modifiable by a change in clinical practice, and since the trauma most commonly took the form of intracranial haemorrhage, the increased risk of brain damage among the survivors seems self-evident. The deaths attributed to anoxia were not associated with any particular obstetric factor, but the introduction of a presumably more effective method of neonatal resuscitation, by intermittent positive pressure ventilation (IPPV), at about the time our study population was being born, has focussed our interest upon this particular factor. The risk that perinatal anoxia may produce pathological changes in the brains of babies and of experimental animals who die, and may also produce neurological and psychological deficits among those who survive, was well documented before our study began (Windle, 1958). We are particularly concerned to determine how important was the contribution of perinatal anoxia to the total amount of handicap in our community, and whether this could be decreased by the routine use of IPPV when indicated; and whether, if some deaths were prevented by this means, this might be achieved only at the expense of an unacceptable risk of severe handicap among the survivors.

For these reasons, we have thought it right to focus our interest first upon these two adverse perinatal factors, abnormal delivery (potentially modifiable by the obstetrician) and perinatal anoxia (potentially modifiable by the paediatrician or other persons responsible for neonatal resuscitation). However, before

Table 2.1

Neonatal mortality rate per 1,000 singleton legitimate live births delivered by the four commonest methods

Birth Weight	METHOD OF DELIVERY				Number of Babies
	Spontaneous Vertex (11,528)	Breech (290)	Forceps (865)	Caesarean Section (497)	
Boys					
<=2.5 kg	148.9	303.0	200.0	212.1	388
>2.5 kg	5.6	40.0	9.1	24.4	6,374
All weights	12.5	94.9	25.0	46.6	6,762
Girls					
<=2.5 kg	73.4	160.0	179.5	161.3	422
>2.5 kg	4.3	28.0	11.6	10.7	5,996
All weights	8.3	53.0	28.6	32.1	6,418
Both					
All weights	10.4	75.9	26.6	40.2	13,180

Table 2.2

Neonatal mortality rate per 1,000 singleton legitimate live births by time taken to establish regular respiration

Birth Weight	TIME TAKEN TO ESTABLISH REGULAR RESPIRATION			Number of Babies
	<1 minute (10,601)	1-4 minutes (2,070)	>=5 minutes (449)	
Boys				
<=2.5 kg	76.0	121.6	639.4	385
>2.5 kg	4.5	4.9	82.1	6,347
All weights	7.8	12.7	209.0	6,732
Girls				
<=2.5 kg	39.2	67.6	595.2	422
>2.5 kg	2.6	4.5	100.7	5,966
All weights	4.8	9.3	215.5	6,388
Both				
All weights	6.3	11.1	211.6	13,120

we go on to look at their effects upon the quality of the survivors, it seems desirable to look a little more deeply into the patterns of neonatal mortality associated with these two factors.

The neonatal mortality rates associated with the four commonest methods of delivery in our population are set out in Table 2.1, where we have shown the rates for babies of low birth weight and for the sexes separately, since babies of low birth weight and boys have a higher mortality rate (as shown in the table) and a higher risk of abnormal delivery (as shown in Appendix Table 2.1).

Clearly, of the three abnormal methods of delivery, breech is the one associated with the highest mortality rate (7 times greater than spontaneous vertex, nearly 3 times as great as forceps and twice as great as Caesarean section). Furthermore, if he considers it desirable to do so, the clinician can always avoid breech delivery by performing a version or a Caesarean section, and the commonest causes of death associated with breech delivery are intracranial trauma and anoxia. This, therefore, seems to be an ideal perinatal factor for us to study in relation to the quality of the survivors, and there were 268 singleton legitimate babies who survived the first month of life following a breech delivery and were enrolled in our Community Study. The differences between the figures in the different cells of Table 2.1 suggest that it may always be desirable to subdivide our results by birth weight and by sex.

Our clinical definition of neonatal asphyxia was in terms of the time taken to establish regular respiration after birth. As mentioned in Chapter 1, the midwives and doctors responsible for recording the perinatal data were asked to state the time interval between birth and the establishment of regular respiration for each live birth in one of three groups - less than 1 minute, 1-4 minutes and 5 minutes or more (including those in whom regular respiration was never established). In the third group, constituting 'clinical asphyxia', they were asked to state the actual number of minutes which elapsed in those who did eventually establish regular respiration, and the details of any procedures employed for resuscitation. The neonatal mortality rates associated with these three degrees of delay in all the singleton legitimate live births for whom the relevant data were available are shown in Table 2.2, which is subdivided in the same way as Table 2.1. In order to avoid undue complexity, we have not subdivided the two low birth weight groups in the table, but the overwhelming effect of a very low birth weight of less than 1,360 g (3 lb) is worth emphasising at this point. The 64 babies in this category had an overall neonatal mortality rate of 765.6 per thousand, without any detectable effect either

of sex or of delay in establishing regular respiration. The higher incidence of delay in onset of regular respiration among babies of low birth weight and among boys is shown in Appendix Table 2.2. The figures in Table 2.2 leave no doubt about the high mortality rate associated with clinical asphyxia as we have defined it. Of the 95 neonatal deaths in this group, 42 babies never succeeded in establishing regular respirations at all. There were 354 survivors of the first month of life who had suffered from clinical asphyxia and who were enrolled in our Community Study.

Both in attempting to understand the causes and the effects of clinical asphyxia, and in organising effective treatment, it is important to recognise its association with abnormal methods of delivery. This is illustrated in Table 2.3. During the years when our children were born, inhalation anaesthesia and intravenous pentothal were routinely used for abnormal deliveries; no doubt the introduction of nerve block and epidural techniques has diminished the strength of this association. However, any abnormal method of delivery can still be regarded as a warning of an increased risk of clinical asphyxia.

The opportunity to study the effects of IPPV was provided by the pure chance that at the time when our Community Study population was born, this method of resuscitation had not yet been widely accepted and was routinely available in only one of the two major obstetric departments covered by the Maternity Survey. We have, therefore, been able to compare the immediate effectiveness of IPPV in preventing neonatal death with that of chemical stimulants and intragastric oxygen, which were the only methods of resuscitation routinely available in the other obstetric department; and we have also been able to investigate the possibility that, if IPPV were shown to be effective, too many of those successfully resuscitated might show evidence of some degree of brain damage.

To assess the immediate effect, we have looked at the early neonatal deaths (occurring in the first four days) associated with clinical asphyxia, where there seems to have been a reasonable chance that the baby's life might have been saved by a more effective method of resuscitation. We have excluded 20 cases of traumatic haemorrhage, 15 of severe malformation and 10 with a birth weight equal to or less than 908 g (2 lb), and are left with 22 deaths from 'uncomplicated clinical asphyxia' during the first four days after birth in one or other of the two major obstetric departments. These include six cases of hyaline membrane disease, because we have reason to believe that more effective resuscitation can materially reduce the risk of death from this

Table 2.3

Percentage distribution of time taken to establish regular respiration related to the four commonest methods of delivery

TIME TAKEN TO ESTABLISH REGULAR RESPIRATION	METHOD OF DELIVERY				Number of Babies
	Spontaneous Vertex (11,402)	Breech (281)	Forceps (843)	Caesarean Section (478)	
<1 minute	84.0	46.6	65.4	52.7	10,508
1-4 minutes	14.2	37.4	22.6	28.7	2,054
>=5 minutes	1.8	16.0	12.0	18.6	442
Total	100.0	100.0	100.0	100.0	13,004

Table 2.4

Early neonatal deaths attributable to 'uncomplicated clinical asphyxia' per 1,000 singleton legitimate live births in two major obstetric departments, related to the availability of IPPV

INTERMITTENT POSITIVE PRESSURE VENTILATION	Birth Weight				Number of Babies
	909-1,362g (55)	-2,500g (422)	>2,500g (4,862)	All >909g (5,339)	
Not available	166.7	24.3	1.5	5.3	3,576
Available	0.0	22.4	0.0	1.7	1,763

Table 2.5

Number of singleton legitimate live births and early neonatal mortality rate per 1,000 attributable to 'uncomplicated clinical asphyxia', related to social class and availability of IPPV

INTERMITTENT POSITIVE PRESSURE VENTILATION	SOCIAL CLASS							
	I+II		IIIA		IIIM		IV+V	
	No.	Rate	No.	Rate	No.	Rate	No.	Rate
Not available	454	2.2	411	9.7	1,726	4.6	916	6.5
Available	282	0	222	4.5	836	2.4	377	0

cause (Omer, Robson and Neligan, 1974). The results summarised in Table 2.4 show that the overall early neonatal mortality rate attributable to 'uncomplicated clinical asphyxia' is 3 times as great when IPPV was not routinely available; however, this difference is not statistically significant ($p>0.05$). The reason for this failure to achieve conventional levels of significance is that the absolute rates are too small even in populations of this size. For these rates to become significant, the number of children in each group would need to be one third greater. The ratio of boys to girls (1.11) is slightly higher among those live births for whom IPPV was available, and all three deaths occurred in boys; among those for whom it was not available, 13 of the 19 deaths occurred in boys.

The social class distribution of the live births in the two departments also differs, and in view of the possible effects of this difference both upon the neonatal mortality and the performance of the survivors the results are shown in Table 2.5. Clearly, there is a higher proportion of babies in Social Classes IV and V among the live births for whom IPPV was not available, but this does not explain the higher mortality attributable to 'uncomplicated clinical asphyxia' among them, since it is present in every social class grouping (though still not statistically significant, of course).

DISCUSSION

We have identified two adverse perinatal factors which appear to fulfil our two requirements of being associated with a high neonatal mortality rate attributable to causes which might involve an increased risk of brain damage among the survivors, and of being directly modifiable by alterations in clinical practice. These two adverse factors are breech delivery and clinical asphyxia (defined as a delay of 5 minutes or more before regular respiration is established after birth). Our longitudinal data concerning the quality of the survivors of these two adverse factors should make it possible to decide whether there is a significant '...continuum of reproductive casualty with a lethal component consisting of...neonatal deaths, and a sublethal component consisting of cerebral palsy and perhaps other related conditions.' (Lilienfeld and Pasamanick, 1955). If this concept is of real importance, we would expect to be able to identify amongst these survivors a definite excess of clinically recognisable handicaps, and more widespread impairment of intellectual or behavioural development to a degree only recognisable by systematic application of standardised tests.

Retrospective analysis of the same data, starting

with children later identified as suffering from impaired functioning of the nervous system, should make it possible to evaluate the relative importance of these severe perinatal factors compared with other antecedent factors which might plausibly be suggested as 'causes' of the handicapping condition.

In the case of clinical asphyxia, a small bonus is available to us through the chance that at the time when our children were born, the technique of intermittent positive pressure ventilation was available in one but not in the other of the two large specialist obstetric departments which we covered. It should, therefore, be possible to assess the results of a presumably more effective method of treatment, not only in terms of neonatal mortality (as in this chapter), but also in terms of the quality of the increased number of survivors.

In the next two chapters (3 and 4) we have attempted to assess the sequelae of the two severe perinatal factors whose relationships with neonatal mortality we have described in this chapter; in the following two chapters (5 and 6) we have attempted to identify antecedent factors which could plausibly be regarded as the cause of the cases of malfunction of the central nervous system, with or without localising signs of 'brain damage', which have been identified by clinical, administrative or epidemiological methods in our study population.

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3

THE SURVIVORS OF BREECH DELIVERY

Breech delivery is one of the two perinatal factors which we have identified in Chapter 2 as a particularly promising subject for study of the sublethal component of a hypothetical 'continuum of reproductive casualty', because it is associated with a high neonatal mortality rate and an obvious risk of organic brain damage amongst the survivors, and it is preventable by straightforward clinical intervention.

In Table 3.1 we have summarised the information available when they had reached school age concerning the 268 survivors of the first month of life who had experienced a breech delivery (Appendix Table 2.1). The information available for those we have classified as 'normal' is as follows:-52 had not been traced at school age but had passed at least two major milestones before the 90th centile age (Neligan and Prudham, 1969 a) and b)); 23 were known to have entered a normal Newcastle school at about the age of 5 years but had not completed our special drawing test; 14 were reported by correspondence to have entered a normal school elsewhere in Britain. The 6 children we have classified as 'probably normal' had passed at least one major milestone at a similar age. It seems pretty certain that the 52, and likely that the 6, would have been able to enter normal schools at an appropriate age, but we have no direct information on this. In the case of the 4 for whom we have 'no usable information available', the most we know, for example, is that 'He seemed a pleasant baby at four months - emigrated to Canada'. Our search for the sublethal component of a 'continuum' in the form of major or relatively minor abnormalities occurring as sequelae of breech delivery is confined to the 169 children concerning whom we have more precise information.

Major Abnormalities

Of the 6 deaths before the age of 5 years, 4 were clearly unrelated to the method of delivery (1 case of tracheo-

oesophageal fistula dying at 7 weeks, 1 of bronchiolitis at 3 months, 1 'cot death' at 3 months and 1 road accident at 2 years). The remaining 2 died with subdural haematomas at 10 and 11 months respectively; each was thought to have been recently injured in a fall and it is difficult to credit that there can have been any connection with the method of delivery.

The one child who was too grossly handicapped to be admitted to a normal school was a case of Down's syndrome, whose cause is clearly unrelated to the method of delivery.

It is clear that breech delivery made no contribution to the sum total of severe abnormalities among children born into our community over a period of three years.

Minor Abnormalities

The most sensitive way of using our data to test the hypothesis that there may be a continuum of minor abnormalities of function due to minimal brain damage extending into our community as a sequel of breech delivery, is to analyse the results of the standardised tests of performance administered at the ages of 5 and 10 years. However, before doing so we have looked for cases of clinically recognisable neurological abnormalities amongst the survivors of breech delivery who were admitted to normal schools at about the age of 5 years. There were only two such children, both presumed to be suffering from a mild form of cerebral palsy with ataxia and clumsiness as the only definite clinical abnormalities; both were still in normal schools at the age of 10 years and had a non-verbal IQ in the lower part of the normal range. One weighed 2,870 g at birth and established regular respiration after 1-4 minutes; the other weighed 3,720 g, was very shocked after delivery by an inexperienced doctor in a small hospital, and took 15 minutes to establish regular respiration. This is a clear example of the syndrome which Little (1843-4) recognised, and the occurrence of two cases of cerebral palsy, however mild, among the 185* children admitted to normal Newcastle schools at about the age of 5 years, giving an incidence of 1.1 per cent, represents a significant excess of such cases among the survivors of breech delivery ($p<0.01$), since the incidence in our whole Survey population of this age is only 1.8 per thousand (see Chapter 5). However, in absolute terms, the contribution made by breech delivery to the sum total of minor clinically recognisable abnormality of this type in our population is still not a big one.

The first standardised tests of performance whose

*Of the 185, 162 had completed a drawing test and 23 had not (see Table 3.1 and text above).

Table 3.1

Information available at school age concerning all the singleton legitimate survivors of the neonatal period who had experienced a breech delivery

Information	Number
Died before age of 5 years	6
Grossly handicapped: never in normal school	1
Considered normal	89
Considered probably normal	6
No usable information available	4 (1.5%)
School drawing test IQ score available at 5 years	162
Total	268

Table 3.2

Mean Goodenough IQ and Figure Copying scores at age of 5 years

	Method of Delivery	
	Spontaneous Vertex	Breech
Boys		
Goodenough IQ	110.1**	104.7**
Figure Copying score	5.0**	4.4**
Girls		
Goodenough IQ	114.8	114.2
Figure Copying score	5.1	5.3

**Difference between breech and vertex $p<0.01$

results we can use in this analysis are those which were administered as part of the special drawing test during the children's first term in school. In Table 3.2 we have compared the mean Goodenough IQ and Figure Copying scores of those who had been delivered by the breech as compared with spontaneous vertex, and we have shown the results for boys and girls separately. There is a statistically significant impairment in performance by both tests following breech delivery, but only among boys. The difference between the mean IQ results in the boys is due to a general downward displacement of the distribution curve of the breech scores, not to any localised distortion of its shape such as would have indicated the presence of a few relatively severe cases of brain damage.

This unexpected sex difference might be explained as a further, sublethal, manifestation of the greater vulnerability of boys to the adverse effects of breech delivery, which is demonstrated in terms of mortality in Table 2.1. There, the overall increase in the neonatal mortality rate following breech as compared with spontaneous vertex delivery is shown to be very much greater in boys (82.4 per thousand) than in girls (44.7 per thousand). We know of no complete explanation of this sex difference in mortality rates (which might also help to explain the differences in performance at the age of 5 years), but there are some adverse factors which are well known to be associated with breech delivery, whose possible contribution to the impairment of performance at 5 years we have studied by further analyses. These have been confined to the boys because of the absence of overall differences among the girls in Table 3.2.

In Table 2.3 it is shown that clinical asphyxia, which is itself associated with a high neonatal mortality rate, was 9 times commoner following breech than following spontaneous vertex delivery, and the adverse biological factor of low birth weight was 4 times commoner among boys delivered by the breech than among spontaneous vertex deliveries (Appendix Table 2.1). We have, therefore, compared the Goodenough IQ scores following spontaneous vertex and breech delivery among boys, subdivided into three groups by duration of delay in establishing regular respiration (Figure 3.1) and by birth weight (Figure 3.2). In each case, the performance of the boys in all three breech subgroups is inferior to that of the corresponding vertex subgroups, and in spite of small numbers of children the differences are statistically significant where indicated. The fact that the combination of breech delivery and clinical asphyxia appears to be particularly harmful is not surprising (the delay in establishing regular

Figure 3.1

Effect of mode of delivery and delay in establishing regular respiration upon Goodenough IQ at 5 years, in boys (delay not recorded in 1 breech birth)

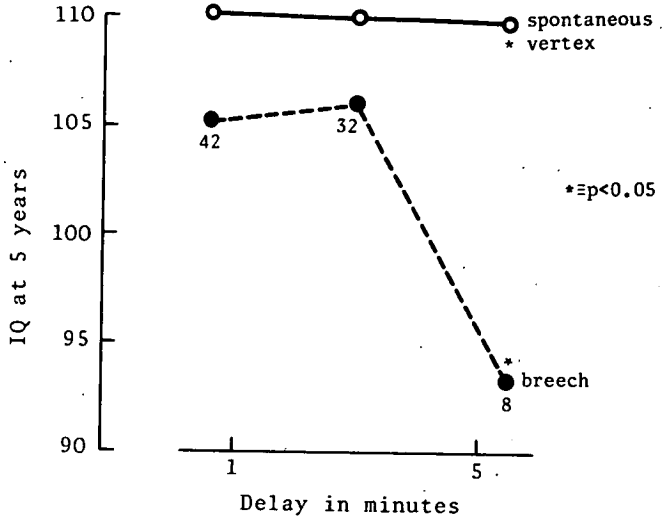
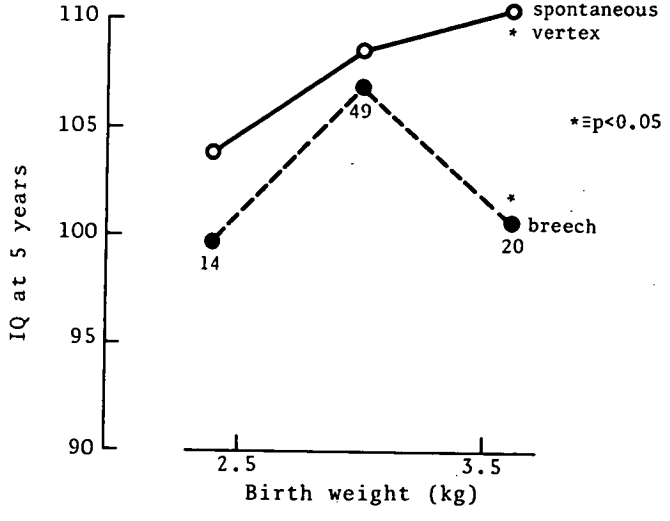


Figure 3.2

Effect of mode of delivery and birth weight upon Goodenough IQ at 5 years, in boys



respiration may be not only a potentially damaging factor in itself, but also an indicator of damage to the brain during the delivery). The adverse effect of high birth weight associated with breech in contrast to vertex delivery can most easily be explained as an indicator of damage to the relatively large aftercoming head of the heavy baby. The suggestion contained in both these figures, therefore, is that the inferior performance of the boys in the breech group is attributable to organic brain damage.

Before accepting this suggestion as proven, however, it is clearly necessary to consider the possibility that the differences illustrated so far might be due wholly or partly to other factors associated with the ones mentioned, either systematically or by chance. For instance, in the breech as compared with the vertex groups there is an excess of first births (40 per cent as compared with 23 per cent) and of births before 38 weeks (25 per cent as compared with 18 per cent), but no significant differences in social class distribution (13 per cent in Social Classes I or II as compared with 11 per cent). We have found that however they are subdivided in terms of these factors, the mean scores of the breech are lower than those of the corresponding vertex subgroups.

But even these results, however consistent, are not conclusive. The final answer can only be given by a more complex type of analysis in which the effects of these factors can be allowed for simultaneously and the significance of each can be assessed independently. We have carried out an analysis of this kind, and the results are reported in Chapter 9.

Meanwhile, we can learn more about the possible clinical significance of our findings at the age of 5 years by seeing if the differences are still present at the age of 10 years. In addition to the non-verbal IQ scores, we have looked at the proportion of the boys in each of the two delivery groups who were ascertained as in need of special education in a school for the educationally subnormal (ESN), and the proportion whose behaviour was rated as 'abnormal' on the evidence of the standardised behaviour inventory completed by the teacher. The results are summarised in Table 3.3. Although there is a difference in favour of the vertex groups in all three parameters, none of these differences is significant, either statistically or clinically; and when we look at the combination of breech delivery and clinical asphyxia, which appeared to have a particularly harmful effect upon the results at 5 years, we find that the 9 boys in the breech group who took more than 5 minutes to establish regular respiration have a mean non-verbal IQ score of 91.9, which does not differ significantly from

Table 3.3

Performance in boys assessed at the age of 10 years

	Method of Delivery	
	Spontaneous Vertex	Breech
Non-Verbal IQ by NFER test (mean)	93.8	92.5
Proportion in City ESN schools	1.8%	2.7%
Proportion with 'abnormal' behaviour reported by teacher	21.0%	25.3%

Table 3.4

Performance assessed at ages of 5 and 10 years

	Method of Delivery	
	Forceps	Caesarean Section
Boys		
Goodenough IQ at 5 years (mean)	111.5	110.4
Non-verbal IQ at 10 years (mean)	96.8	94.5
Proportion in City ESN schools	0.4%	3.2%
Proportion with 'abnormal' behaviour reported by teacher	15.1%	17.7%
Girls		
Goodenough IQ at 5 years (mean)	116.4	116.2
Non-verbal IQ at 10 years (mean)	98.0	95.0
Proportion in City ESN schools	1.1%	0.9%
Proportion with 'abnormal' behaviour reported by teacher	11.8%	11.2%

the spontaneous vertex mean score of 93.8.

On the basis of these findings, therefore, we must conclude that whatever differences there may have been between the performance of the boys in the vertex and breech groups at the age of 5 years, due to whatever causes, they are no longer demonstrable at the age of 10 years.

To complete the picture, and in case either of the other two common methods of delivery, though associated with a lower neonatal mortality rate than breech, might produce a greater impairment of performance among the survivors, we have analysed the data available for the children delivered by forceps or Caesarean section. The results are summarised in Table 3.4 and can be compared with those shown in Tables 3.2 and 3.3. The slight but consistent differences in favour of children delivered by forceps or Caesarean section are not significant, nor was there any excess of children delivered by forceps or Caesarean section among the cases of brain damage discussed in Chapter 5.

DISCUSSION

For many years, obstetricians have been extending their indications for Caesarean section in cases of breech presentation, because of their concern about the high perinatal mortality rate associated with breech delivery. More recently this trend seems to have been reinforced by increasing concern for the quality of the survivors. Rupek, Feldmann and Tenhaeff (1972), for instance, used 'the known high ratio of sublethal infantile brain damage', as well as the perinatal mortality, to justify a policy of performing a Caesarean section in all primiparae with a fully grown fetus presenting by the breech (but not in those with 'immature' fetuses, whose 'chances of survival...are definitely not enlarged through a Caesarean section'). Such statements suggest that it is an accepted fact that the increased risk of clinically recognisable brain damage among the survivors of breech delivery is sufficiently great to be of real clinical importance to the obstetrician. The existence of such a risk is not in doubt, and this was part of the evidence upon which the concept of a 'continuum of reproductive casualty' was based.

When we try to assess its magnitude on the basis of the published evidence, however, we find surprising differences between the findings of different workers. For instance, Lilienfeld and Parkhurst (1951), Lilienfeld and Pasamanick (1955) and Pasamanick and Lilienfeld (1955) reported an increased frequency of breech delivery in cases of cerebral palsy but not in cases of

epilepsy or mental retardation. Churchill (1957), on the other hand, reported a great increase in his cases of epilepsy but little in those of cerebral palsy. Asher and Schonell (1950) reported a probably normal rate of breech delivery in 221 cases of cerebral palsy with a birth weight of more than 2.5 kg. The methods used in some of these studies, based as they are upon medically defined abnormal populations with doubts about the validity of the comparison groups, have been criticised by MacMahon and Sowa (1961). The fact that this method of approach allows very large numbers of abnormal children to be studied does not compensate for its inherent weaknesses. On the other hand, when we look at the report by Birch et al (1970), who studied a geographically defined population with impeccable ascertainment of abnormal and control groups, we find that their evidence for a threefold increase of a history of breech delivery in cases of severe mental subnormality consists of only two children.

In terms of numbers, we are in the same vulnerable position as Birch et al (1970). Our only evidence of an increased incidence of clinically recognisable brain damage following breech delivery consists of 2 cases of cerebral palsy, as mentioned in Chapter 5. There are no instances of breech delivery among our 26 cases of severe non-specific mental subnormality, 7 of severe deafness and 3 of blindness, all thought to be prenatal or perinatal in origin, in our geographically defined population of singleton legitimate children followed prospectively to school age.

When we look at our objective test results at the ages of 5 and 10 years, however, the number of survivors of breech delivery in our population is quite sufficient for valid statistical comparisons to be made, either within the groups (e.g. boys cf. girls) or with other groups (e.g. breech cf. vertex). We do not know of any relevant published work, apart from a preliminary report of our own results (Russell, Neligan and Millar, 1970), and a hint in Chapter 16 of the book by Davie, Butler and Goldstein (1972), suggesting that breech delivery may produce an adverse effect measurable by objective tests. Our finding that the adverse effect is confined to boys (Table 3.2) has not been reported previously, as far as we know. There are many precedents for the overall superiority of the girls' test scores (Singer, Westphal and Niswander, 1968; Hutt, 1972). Indeed, Gruenberg (1964) has pointed out that the tests used systematically underestimate the degree of the girls' superiority, since they are 'purposely designed to avoid discrimination between boys and girls'. There are also many well-known instances of the greater vulnerability of the males to adverse perinatal factors. We have drawn

attention already to the much greater increase in the neonatal mortality rate associated with breech delivery among males, which is shown in Table 2.1. Another instance is the excess of males among the children who had suffered from neonatal clinical asphyxia, illustrated amongst the survivors in Table 4.2. Our finding that the male survivors of breech delivery performed less well in objective tests at the age of 5 years than the corresponding females, to a significantly greater extent even than the survivors of vertex delivery, therefore seems to us to be not so surprising and also to be consistent with the concept of a continuum. However, the actual magnitude of the effect is certainly not great.

When we pass on to consider the results of the assessment at the age of 10 years, in fact, there are no longer any significant differences at all between the breech and the vertex groups of the same sex. This may mean that the tests which we used at that age were not suitable for detecting residual evidence of minimal brain damage, or that the children had learnt to compensate for any minimal brain damage which they might have suffered as a result of breech delivery. We feel that the results of the simple analyses reported in this chapter enable us to say that there is a perceptible amount of handicap among the survivors of breech delivery, at least at the age of 5 years, in conformity with the ideas of Little (1843-4) and Lilienfeld and Pasamanick (1955), but that the magnitude of the effect is insufficient to be of real clinical importance. However, the multivariate analysis whose findings are reported in Chapter 9 may be expected to throw more light onto this question.

For the obstetrician, the question of whether the risks to the baby associated with breech delivery are too great to be accepted is better answered in terms of the risk of death than the risk of brain damage among the survivors. For the paediatrician, it does not look as if the overall picture of handicap in the community would be significantly altered by a change in his obstetric colleague's policy about the management of breech delivery.

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4

THE SURVIVORS OF CLINICAL ASPHYXIA

The second perinatal factor which we have identified in Chapter 2 as a particularly suitable subject for study in the same way as breech delivery is clinical asphyxia (defined as a delay of 5 minutes or more in establishing regular respiration after birth). It too is associated with a high neonatal mortality rate and an obvious risk of brain damage among the survivors, and it is accessible to direct clinical intervention by modern methods of resuscitation. Our study population included 354 children who had survived the first month of life after experiencing clinical asphyxia, and in Table 4.1 we have categorised them in terms of the information available about them up to school age.

The evidence which led us to consider 114 children as 'normal' or 'probably normal' is similar to that which we have specified in relation to the survivors of breech delivery in Table 3.1. Similar too are the steps we have taken to analyse the more precise information available about the 6 who had died, the 8 who had been excluded from normal school because of gross handicaps and the 221 for whom we had IQ scores.

Major Abnormalities

Of the 6 deaths before the age of 5 years, 3 were due to causes which could not be related to the clinical asphyxia (1 case each of Down's syndrome, congenital heart disease and head injury at 10 months*). In the other 3 cases we cannot be so sure, but it is difficult to see a causal connection between the clinical asphyxia and 'cot death' (2 cases, at 3 and 4 months) or a death from 'overlaying' in the parents' bed (at 3 months). As in the case of breech delivery, therefore, we have found no relationship between the perinatal factor of clinical asphyxia and postnatal death before the age of 5 years

*This is one of the two deaths from subdural haematoma described in Chapter 3.

in our population.

Among the 8 children categorised as grossly handicapped, there are again 3 in whom the cause was clearly unrelated to the perinatal adverse factor (1 case of Down's syndrome and 2 of postnatal illness - septicaemia with thrombocytopenia leading to mental subnormality, and retinoblastoma with radiation cataract leading to blindness). The other 5 cases are less easy to be sure about, but their clinical histories can be summarised briefly as follows:-

4 suffered only a short period of asphyxia of between 5½ and 8 minutes. 2 (1 hemiplegic and 1 mentally subnormal) were of normal birth weight at full term and had atraumatic deliveries; 1 (mentally subnormal) weighed 1,840 g at 31 weeks; 1 (severely deaf) weighed 1,610 g at 28 weeks and suffered from hypoglycaemia in the neonatal period and epilepsy later.

1 (severely deaf and epileptic) suffered a long period of clinical asphyxia in that he took 37 minutes to establish regular respiration after delivery as an uncorrected face presentation at a gestational age of 45 weeks by the mother's dates, but he was treated by IPPV through an endotracheal tube from the age of 3 minutes and was pink from the age of 4 minutes, though he remained limp until after active regular respiration was established.

The numbers are clearly too small for any detailed statistical analysis, but the impression of an excess of gross handicap among the survivors of clinical asphyxia is inescapable. However, the clinical summaries suggest very strongly that in few, if any, cases was the delayed onset of regular respiration the direct and sole cause of the neurological damage. The delay was of relatively short duration with one exception, and in that case the anoxaemia, which is the potentially damaging effect of the delay, was virtually eliminated by the method of resuscitation used, the baby's colour being pink from the age of 4 minutes. The clinical asphyxia seems rather to have been itself a symptom and an indicator of other adverse factors, perinatal or biological.

Minor Abnormalities

Of the survivors of clinical asphyxia who were admitted to a normal school at the age of 5 years, only 2 were known to be suffering from a clinically recognisable neurological abnormality in the shape of a mild cerebral palsy; one is the second of the two cases of this condition described in Chapter 2, and the other weighed 1,590 g at 29 weeks by dates, taking 2½ minutes to establish regular respiration after being intubated at 2½ minutes of age. We can draw no general conclusions

from this finding, except that the incidence of clinically recognisable minor abnormalities following clinical asphyxia cannot be high.

We can now proceed to consider the relationship between clinical asphyxia and the results of our tests of performance at the ages of 5 and 10 years. In Table 4.2 we have shown the mean Goodenough IQ scores for the three main groupings of delay in establishing regular respiration. The number of children in each of the groups is given to show the extent to which there is a progressive excess of boys in the groups where regular respiration was delayed (giving a male/female ratio of 1.1 for 1-4 minutes and 1.5 for 5 minutes or more). Although there is a progressive fall in the mean IQ with increasing delay, in both sexes, the differences are so slight as to be obviously not statistically significant.

We have, therefore, looked separately at the children in whom there was a more prolonged delay, of 20 minutes or more, in establishing regular respiration. There are 13 boys, and their mean Goodenough score is depressed to 103.9, more than 6 points below that of the less than 1 minute group; but this difference is not statistically significant with these small numbers in the abnormal group. There are only 7 girls with a similar degree of delay (giving a male/female ratio of 1.9), and their mean score is actually raised to 125.3 (but again, the difference, of 10.2 points compared with the 'less than 1 minute' girls, is not significant). We have not directly compared the boys and girls who experienced this prolonged delay, because we have noted that there is a marked distortion of the birth weight distributions in these two small groups; there is an excess of low birth weights among the boys (3 weighed less than 2.5 kg where one would have been expected) and of high birth weights among the girls (5 weighed more than 3.5 kg where two would have been expected). This distortion of an associated biological factor would be expected to make a considerable contribution to the observed difference between the boys' and girls' scores (see Chapter 7).

The apparently cumulative adverse effect of clinical asphyxia and breech delivery is noted in Chapter 3 (see Figure 3.1). To see whether there may be a comparable effect with other abnormal methods of delivery, we have looked at the Goodenough IQ scores of children with clinical asphyxia following delivery by forceps or Caesarean section. They were no different to the scores of children who had experienced a similar delay in establishing regular respiration following a spontaneous vertex delivery, and we have not thought it necessary to show the results in detail.

Table 4.1

Information available at school age concerning all the singleton legitimate survivors of the neonatal period who had suffered from clinical asphyxia

Information	Number
Died before age of 5 years	6
Grossly handicapped: never in normal school	8
Considered normal	105
Considered probably normal	9
No usable information available	7(2.0%)
School drawing test IQ score available at 5 years	219*
Total	354

*Two deaf children categorised as grossly handicapped completed the drawing test, bringing the total number available for analysis up to 221.

Table 4.2

Mean Goodenough IQ scores at age of 5 years

	Delay in Regular Respiration		
	<1 minute	1-4 minutes	>=5 minutes
Boys			
Goodenough IQ	110.1 (3,500)	109.9 (736)	109.1 (132)
Girls			
Goodenough IQ	115.1 (3,477)	114.1 (660)	112.5 (89)

Table 4.3

Performance assessed at age of 10 years

	Delay in Regular Respiration		
	<1 minute	1-4 minutes	>=5 minutes
Boys			
Non-Verbal IQ by NFER test (mean)	93.9	93.8	97.2
Proportion in City ESN schools	1.9%	1.5%	3.0%
Proportion with 'abnormal' behaviour reported by teacher	21.1%	19.3%	16.7%
Girls			
Non-Verbal IQ by NFER test (mean)	95.2	95.5	93.5
Proportion in City ESN schools	1.6%	1.7%	1.3%
Proportion with 'abnormal' behaviour reported by teacher	11.9%	12.1%	10.3%

As in the case of breech delivery, we have looked for effects of clinical asphyxia again at the age of 10 years, and the results are summarised in Table 4.3. This time there is not even a suggestion that the adverse perinatal factor is systematically associated with impaired performance.

The Later Effects of IPPV

In Chapter 2 we reported evidence of a beneficial effect of IPPV upon neonatal mortality, but mentioned our worry that '...if IPPV were shown to be effective, too many of those successfully resuscitated might show evidence of some degree of brain damage'. We have, therefore, sought to determine the size of this risk by analysing the information available when they reached school age about the children who had been born in each of the two major obstetric departments.

Before comparing the later performance of the two groups of children (those for whom resuscitation with IPPV was, or was not, routinely available when they were born), we have compared their birth weight and social class distributions, since these are factors which may be expected to affect the results. We have found no significant differences in either parameter. Of the 554 boys tested at the age of 5 years for whom IPPV was available, 7.2 per cent weighed 2.5 kg or less and 42.8 per cent weighed more than 3.5 kg; in the 1,180 for whom it was not available, the comparable proportions are 7.6 per cent and 40.9 per cent. The social class distribution of those for whom IPPV was available (22.1 per cent in I, II or IIIA and 23.8 per cent in IV or V) is also very similar to those for whom it was not (20.6 per cent and 25.8 per cent). We feel, therefore, that it is useful to start by comparing the mean Goodenough IQ scores of these two groups of children, as shown in Table 4.4.

We could not expect to see any effect of IPPV in those who started breathing regularly within 1 minute, since such treatment was seldom initiated, and could never have been effective in establishing regular respiration, within this time. It is also questionable whether there could have been any beneficial effect within the 1-4 minute subgroup, but it is possible that the duration of anoxaemia could have been reduced in some cases by the prompt use of intubation or bag and mask. It is in the clinical asphyxia subgroup that most benefit might be expected, either through stimulating the earlier onset of regular respiration, or through the ability to establish good oxygenation by ventilation in cases whose active response was very delayed; it is in this same group, however, that we would expect to see any harmful effects of the type about which we expressed our worry. In Table 4.4 the only statistically

Table 4.4

Mean Goodenough IQ scores related to routine availability of IPPV for neonatal resuscitation

IPPV	Delay in Regular Respiration		
	<1 minute	1-4 minutes	>=5 minutes
Boys			
Available	109.8	111.4*	107.6 (39)
Not available	108.9	107.1*	109.3 (44)
Girls			
Available	115.3	115.2	109.7 (27)
Not available	115.4	112.0	114.0 (26)

*Difference between these two subgroups significant ($p<0.05$)
Absolute numbers shown in parentheses where they are less than 100

Table 4.5

Mean Goodenough IQ scores of children actually resuscitated by IPPV compared with those of all births in the same obstetric department

	Duration of IPPV			All Births
	<5 minutes	5-30 minutes	>30 minutes	
Boys	107.9 (22)	104.4 (11)	-	110.1
Girls	112.3 (14)	100.3* (7)	83.0 (1)	115.0*

*Difference between these two scores is significant ($p<0.05$)
Absolute numbers of IPPV children in parentheses

significant difference between relevant subgroups, the 4.3 points advantage of the boys who took 1-4 minutes to start breathing for whom IPPV was available at birth over those for whom it was not, is of doubtful practical significance.

There is internal evidence of systematic differences between the data from the two departments, reflecting differences in obstetric management or recording of observation. We have, therefore, not carried out any further comparisons between these two groups of children. Instead, we have looked more closely at the later information about the children born in the department where IPPV was available, who actually were resuscitated by this method.

There are 98 such children, of whom 32 were considered normal and 3 probably normal at school age, 4 had died or were grossly handicapped by conditions clearly unrelated to any perinatal factor, and 1 was lost to follow-up. Of the remaining 58, 3 were grossly handicapped by conditions which could have been related to perinatal causes. One of them was deaf (he took 37 minutes to establish regular respiration) and 1 was mentally subnormal (a pre-term baby of low birth weight); both are mentioned earlier under 'Major Abnormalities'. The third was mildly hemiplegic (another pre-term baby of low birth weight) and is mentioned under 'Minor Abnormalities'. This leaves 55 children (33 boys and 22 girls) for whom Goodenough IQ scores are available: 22 boys and 14 girls were ventilated for less than 5 minutes, 11 boys and 7 girls for 5-30 minutes and 1 girl for 35 minutes. The mean Goodenough IQ scores of these subgroups and of the total of 554 boys and 534 girls who had been born in the same obstetric department are given in Table 4.5. The progressive depression of mean scores with increasing duration of IPPV, in both sexes, is obvious and large enough in some subgroups to be of practical importance, but in view of the relatively small numbers in some of the cells, only one of the relevant differences is statistically significant. There is a general downward displacement of the scores in the IPPV groups, with a distinct excess of scores below 90 (e.g. 4 of the 11 boys who were ventilated for 5-30 minutes). We have also looked at the clinical histories of the individual children for evidence of associated, presumably primary, adverse factors, and found plenty of them. For instance, of these same 11 boys, 5 were of low birth weight and only 1 had a spontaneous vertex delivery (6 were delivered by Caesarean section, 3 by forceps extraction and 1 by the breech). There were also 2 instances of placenta praevia and 2 of clinical fetal distress. In these children, as in the case of the major abnormalities, it looks very much as if the duration of

clinical asphyxia is usually an indicator of the severity of other adverse perinatal factors, rather than being directly harmful in itself. Prolonged delay in establishing active regular respiration, in spite of stimulation by IPPV, presumably indicates severe depression of the respiratory centre and possibly more widespread changes in the brain, which could be permanent. We have come to regard this as a particularly worrying prognostic sign in cases of cardiac arrest (see Chapter 10).

DISCUSSION

The findings which we have reported in this chapter suggest that among children who have experienced neonatal clinical asphyxia there is an excess of handicapping conditions, at two levels of severity. There appears to be an increased incidence of clinically recognisable brain damage when they reach school age (although the relatively small numbers in our study population preclude statistical confirmation of this suggestion); and there is evidence of some impairment of intellectual performance, though not at a statistically significant level, in the results of our objective testing of the whole population during the first term in school. The findings, in fact, resemble those reported in Chapter 3 for the survivors of breech delivery, and the same reservation applies here, that confirmation of the statistical conclusions depends upon the results of the more complex analysis to be reported in Chapter 9.

In spite of the similarity of these results, however, the actual roles which these two adverse perinatal factors play differ so much that the clinical problems which they pose look very different. Breech delivery can be regarded as a 'primary' factor in that it can be avoided by the deliberate choice of the obstetrician if he considers that the balance of advantage is in favour of doing so, and it is an important cause of clinical asphyxia, which is, therefore, a 'secondary' factor (see Table 2.3). When we find that the apparently harmful effects of breech delivery upon the quality of the survivors is most marked in boys who have also experienced the secondary factor of clinical asphyxia, the most obvious explanation is that anoxaemia resulting from the baby's delay in establishing effective active respiration has aggravated any organic brain damage which may have been caused by trauma or anoxaemia during the delivery itself. This is also the most hopeful explanation, since the neonatal anoxaemia can theoretically be virtually eliminated by early and efficient use of IPPV. There are too few cases of breech delivery complicated by clinical asphyxia in the department where

IPPV was available, for us to be able to test this explanation directly by statistical analysis of the results of resuscitation. We can note in passing, however, that the girl who took 35 minutes to establish regular respiration and later had an IQ of 83 (Table 4.5) had been intubated at 1 minute and was pink before 10 minutes, whereas another, who took 20 minutes to establish regular respiration and later had an IQ of 132, had been born in the department where IPPV was not available; and that a boy with the same duration of clinical asphyxia, who later had an IQ of 112, had been born at home.

Another explanation, which seems more probable in the light of our general findings, is that the combination of breech delivery and clinical asphyxia is particularly harmful because the delay in establishing regular respiration is an indicator of severe damage to the vital centres in the brain stem caused by other factors operating before or during the birth itself. Prolonged delay according to this explanation carries more sinister implications in cases where prompt and efficient IPPV has been available, because failure to respond promptly to this most effective method of resuscitation implies relatively severe (and so possibly permanent) damage to the vital centres and presumably to other parts of the brain. An exception to this generalisation would be the case in which the baby's behaviour is explained purely by the depressant effect of a drug administered to the mother which has crossed the placental barrier; maintenance of adequate ventilation until the drug's effect has been neutralised or has worn off should protect the baby from any ill effects, in spite of prolonged clinical asphyxia (as judged by delay in establishing satisfactory active respiration). However, exceptions of this kind are relatively few in our material.

Before leaving the subject, however, we wish to compare our findings with those of similar studies reported in the literature. We only know of three which are prospective studies of clinically diagnosed cases of neonatal asphyxia using objective tests at school age, with appropriate control groups in two of the three studies. Campbell, Cheeseman and Kilpatrick (1950) examined 61 index cases who had suffered from 'asphyxia pallida, or asphyxia livida lasting for a stated period of over 2 minutes', and 134 controls, at the age of 6-9 years, using a variety of methods of assessment including testing by Raven's matrices. They found no significant differences in performance between the two groups of children, but two of the index cases, not available for examination, were known to be mentally defective. Fraser and Wilks (1959), confining their attention to singleton legitimate children with a birth weight of more than 2.5 kg, examined 100 index cases (60 of whom

had taken 3-5 minutes, and 40 more than 5 minutes, to establish regular respiration), and 100 controls at the age of 7-11 years. They found one case of cerebral palsy and five of convulsions among their index cases, and were able to demonstrate minimal impairment of motor function and perception, but no reduction in IQ, reading, arithmetic or personality scores. Schachter and Apgar (1959) examined 156 clinically diagnosed cases of asphyxia at the age of 7-9 years and found no neurological evidence of brain damage, but nearly 5 points depression of IQ. The methods of resuscitation available in the three institutions where these children were born are not specified, but it is safe to assume that IPPV was not one of them. The fact that all three studies report, if anything, less evidence of brain damage among children who had suffered neonatal asphyxia than we have found, may be accounted for partly by the relatively mild degrees of asphyxia included in their clinical definitions, and partly by the relatively late age at which they examined their children, when their performance may have been modified by several years of schooling; but the main defect of all three studies, which has made it impossible to know how much weight could be placed on their encouraging findings, is the high proportion of losses from their index cases between the time of birth and the single follow-up examination - no information whatever is available concerning 27 per cent, 20 per cent and 60 per cent of their original cases. If only a small proportion of these missing children were grossly handicapped, this would be enough to invalidate all their published conclusions. With the support of our own findings, however, based as they are upon information concerning 98 per cent of our 354 index cases who took 5 minutes or more to establish regular respiration after delivery, we feel able to conclude that there are surprisingly few sequelae of neonatal clinical asphyxia, but that there are some. The effect of IPPV upon the quality, as distinct from the quantity, of the survivors of the neonatal period has caused us some perplexity and clearly merits further study. The sequelae of severe neonatal asphyxia (defined as a delay of more than 20 minutes in establishing regular respiration) among babies born in a hospital where IPPV is routinely available, are discussed in Chapter 10, as are their clinical implications.

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5

CLINICALLY RECOGNISABLE BRAIN DAMAGE

We now want to reverse our procedure, by looking for plausible causes of the handicaps about which we have information in our community. Impaired performance which might be caused by organic damage to the brain or special senses was identified for us in a variety of ways - by the community's routine health services at a clinical level, by its routine educational services at an administrative level, and by the special tests carried out specifically for the purposes of our Survey at an epidemiological level. This chapter is concerned only with the clinically recognisable handicaps, of a severity which usually precluded admission to a normal school.

The number of children suffering from such handicaps, which any practicable, prospective survey of a geographically defined population is likely to include, is frustratingly small. This point was made in the Introduction, and is amply confirmed by our experience. Among the 9,626 Survey children who were still living in the City of Newcastle when they reached school age, there were only 76 known to be suffering from a disability of this type. There were 14 with pure cerebral palsy, 11 severely deaf, 4 blind and 47 severely mentally subnormal. (Two of the deaf and 1 of the mentally subnormal also suffered from cerebral palsy, bringing the total number of cases of this condition up to 17.) Nevertheless, because our data were recorded prospectively and systematically for a whole geographically defined population, they may make it possible for us to learn something about the size of the problem which each of these conditions presents in our community, and about their causes.

If we want to calculate the prevalence of each of these conditions in a prospectively studied population such as ours, we have to decide what age-groups to use for our calculations. The number of singleton legitimate survivors of the neonatal period originally enrolled in our Survey was 13,203; by the age of 5 years, the number

Table 5.1

Cerebral Palsy

19 cases diagnosed during first 5 years of life
Categories of clinically plausible causes of brain damage

a) Postneonatal illness	3 cases	2 of meningitis (at 3 months and 2 years) 1 of cerebral thrombosis complicating congenital heart disease (at 10 months)
b) Perinatal factor	3 cases (2 doubtful)	2 of breech delivery (1 weighed 2,800 g and took 1-4 minutes to establish regular respiration; the other 3,720 g and 15 minutes) 1 of clinical asphyxia (of 6 minutes duration after elective Caesarean section)
c) Biological factor	5 cases	All 5 of low birth weight (1,130 g; 1,190 g; 1,500 g; 1,700 g; 1,820 g)
d) No plausible factor	8 cases	

Table 5.2

Severe Deafness

11 cases diagnosed during first 5 years of life
Categories of clinically plausible causes of brain damage

a) Postneonatal illness	1 case	Meningitis at age of 9 months
b) Family history	1 case	Had two deaf first degree relatives
c) Damage to embryo	2 cases	1 mother had rubella in first trimester 1 child had microtia and facial palsy - ?due to thalidomide
d) Perinatal factor	1 case	Severe neonatal clinical asphyxia (of 37 minutes duration following face presentation - see Chapter 4)
e) Biological factor	3 cases	All 3 of low birth weight (1,590 g; 1,815 g; 1,875 g)
f) No plausible factor	3 cases	

known to be still living in the City had fallen to 9,626. It seems best to calculate the prevalence at this age, since all cases of the conditions with which we are here concerned should have been identified by then. However, we recognise that in some instances the resultant figure may have been distorted by selective migration; for instance, the presence of an excellent School for the Deaf may have induced certain families to stay in the City who would otherwise have moved away. We will, therefore, state the whole picture as far as we know it for each condition in turn, including known deaths and removals before the age of 5 years.

Cerebral Palsy

There were 17 cases of cerebral palsy still living in the City at the age of 5 years (2 of whom were also deaf and 1 severely mentally subnormal), giving a prevalence of 1.8 per thousand singleton legitimate children of that age. Two further cases had been diagnosed before the age of 2 years, but had moved away from the City before the age of 5. There are, therefore, 19 cases in whose records we can look for plausible causes of their brain damage, using simple clinical judgment to reach our decisions. The results are summarised in Table 5.1.

The two cases which followed breech delivery were mild, with ataxia and clumsiness the main abnormality; there were 2 cases of spastic paraplegia (weighing 1,130 and 1,820 g respectively); the remainder were asymmetrical cases of spastic cerebral palsy (hemiplegia or monoplegia). Of the 17 who were still living in the City at 5 years, 7 were in normal schools and 10 in special schools (7 for the physically handicapped, 2 for the deaf and 1 for the severely mentally subnormal). Of the 16 cases not thought to be due to postnatal illness, 9 were boys and 7 girls. Only in the two instances of breech delivery does there appear to have been any opportunity for prevention by direct clinical intervention (if we assume that most cases of low birth weight are still not preventable).

Severe Deafness

There were 11 children who had been admitted to the local special School for the Deaf by the age of 5 years, giving a prevalence of 1.1 per thousand. No severely deaf children were known to have moved away from the City by that age. We have attempted to find clinically plausible causes of their disability by examining their records, and the results are summarised in Table 5.2.

Of the 7 not thought to be due to categories a)-c), 5 were boys and 2 were girls. There were opportunities for prevention by improved clinical management, by present standards, in three cases. Exposure to thalidomide

Table 5.3

Severe Mental Subnormality
35 non-specific cases diagnosed during first 5 years of life
Categories of clinically plausible causes of brain damage

a) Postneonatal illness	3 cases	1 of gastroenteritis and thrombocytopenia at 6 weeks 1 of encephalitis at 9 months 1 of cerebral abscess at 9 months
b) Family history	3 cases	All had close relatives similarly affected
c) Perinatal factor	2 doubtful cases of clinical asphyxia (see Chapter 4)	
d) Biological factor	5 cases	1 of low birth weight (1,760 g) with vaginal bleeding before 28 weeks 2 of low birth weight (1,930 g and 2,070 g) born at home without any attendant 2 of low birth weight without any specific complication (1,220 g and 1,500 g)
e) No plausible factor	22 cases	

and other teratogenic drugs is avoidable, and systematic immunisation of susceptible women should eliminate the risk of rubella embryopathy. The case of neonatal clinical asphyxia followed a gestation of 44 weeks by dates and there had been clinical signs of fetal distress for two hours before delivery; the baby's blood chemistry was not investigated, so that any metabolic acidosis which may have been present remained uncorrected; he had a number of fits on the second day of life. Both obstetric and paediatric management left room for improvement, by present standards, but, again, it looks as if, in the majority of cases, no such straightforward opportunity for prevention of the handicap can be identified.

Blindness

There were 4 blind children known to be still living in the City at the age of 5 years, giving a prevalence of 0.4 per thousand. Another two, both grossly mentally subnormal, were known to have died before school age, and a third, also mentally subnormal, had moved away. Only in two cases was there a plausible clinical cause of the blindness - one was due to retinoblastoma and radiation cataract, and one to familial cataracts (three first degree relatives affected). In none was there a clear opportunity for prevention by improved perinatal management.

Severe Mental Subnormality

There were 47 severely mentally subnormal singleton legitimate children excluded from normal school, whose families were known to be still living in the City when they reached the age of 5 years, giving a prevalence of 4.8 per thousand. The epidemiology of all degrees of mental subnormality is discussed in Chapter 6; at this point we wish to give only the same type of clinical data as we have given for the other three clinically recognisable types of handicap covered in this chapter. In addition to the 47 still in the City, we know that another 8 cases of Down's syndrome had been diagnosed (of whom 4 had died before the age of 6 months, and 4 had moved away from the City before the age of 5 years), and 5 cases of non-specific severe mental subnormality had died (between the ages of 1 and 4½ years) and 3 had moved away from the City. This makes a total of 63 cases in which we could look for a plausible clinical cause. However, as the cause is self-evident in cases due to specific syndromes, we have excluded the 25 cases of Down's syndrome, 2 of cretinism and 1 of Hurler's syndrome from Table 5.3.

DISCUSSION

Our findings point to the general conclusion that there is little scope for reducing the amount of gross handicap in our community by improved perinatal clinical management, at least in terms of avoiding breech delivery and treating neonatal asphyxia. As mentioned in the introductory portion of this chapter, the absolute number of children in each of the four clinical groups is frustratingly small and only in the case of cerebral palsy and severe mental subnormality is it useful to make any comparisons with previous publications.

In a retrospective study of 370 cases of cerebral palsy, Skatvedt (1958) reported that 58 per cent had had an abnormal neonatal experience (including 'protracted initial apnoea' in 25 per cent and 'asphyxial attacks after initial respirations' in 18 per cent). However, the conclusions appear to be based upon unreliable evidence (e.g. 'The mother declared that the child had been worked on for a long time before it started to cry.'). The mother's story was also relied upon by Asher and Schonell (1950), who reported that of 221 cases of congenital cerebral palsy with a birth weight of more than 2.5 kg, 38 per cent had a history of perinatal 'injury' or 'asphyxia'. Griffiths and Barrett (1967) also relied primarily on the mother's story, and '...where the information was equivocal or suspect the obstetric records were obtained where available'. Among 310 cases of cerebral palsy, they obtained a history of asphyxia and/or birth injury in 53 per cent of those who weighed more than 2.5 kg and in 33 per cent of those who weighed less. However, their criteria of asphyxia included 'delay in establishing respiration' (of unspecified duration), and their criteria of trauma included 'other evidence of injury such as cephalhaematoma'. Even Ingram (1964), in his most detailed analysis of possible antecedent factors, had access to clinical records of perinatal events in less than half of his 208 Edinburgh cases of cerebral palsy born between 1938 and 1952. Among 160 cases not thought to have been postneonatal in onset, he found evidence of 'insults' (anoxic, toxic or traumatic) in 81.2 per cent. The one factor which features most prominently and consistently in most of these reports of possible causes of cerebral palsy (but not in Ingram's) is low birth weight. It has been pointed out by Nelson (1968) that retrospective histories taken from mothers tend to exaggerate perinatal abnormalities.

In their historic paper, Lilienfeld and Parkhurst (1951) derived their data about antecedent factors from birth certificates. They found mention of one or more complications of pregnancy or delivery in the certificates of 37.8 per cent of 517 children who were later

found to be suffering from cerebral palsy, as compared with 62.4 per cent of the perinatal deaths and 20.9 per cent of the total survivors of the neonatal period in the same population. One of the specific complications which they noted separately was breech delivery, in 5.8 per cent of cases of cerebral palsy, 8.0 per cent of perinatal deaths and 2.0 per cent of survivors. They also noted the excess of low birth weights in the two abnormal groups.

Because the absolute number of cases of cerebral palsy in our population is so small, comparison with the findings of these large retrospective studies can only be impressionistic. It does look as if our findings are congruous, however, if we allow for the fact that there had already been marked improvements in obstetric practice (reflected in reduced perinatal mortality rates) during the 20-30 years prior to 1960-2 when our cohort was born. These, and the fact that we have prospective, professionally recorded data concerning perinatal events, may account for our much lower rate of 'trauma' or 'asphyxia'.

We have particularly focussed our attention upon plausible perinatal causes of cerebral palsy (and other forms of brain damage) which could easily be prevented by straightforward clinical intervention. There is no doubt that our findings are disappointing when considered from the point of view of the opportunities for effective action in the future. We have not, so far, looked at the role of all possible adverse factors in the pregnancy and the perinatal period, since this approach appears to offer little prospect of leading to effective preventive action at the present time. It seems clear that the biggest single technical step forward would be the prevention of low birth weight.

Our clinical findings concerning plausible causes of severe mental subnormality are similar to those of previous studies insofar as valid comparisons are possible. Drillien (1968) did not find complications of delivery to be at all frequent, and Kushlick and Cox (1973) reported probable birth injury in only 1.0 per cent (but possible birth injury in another 12.8 per cent) of 508 severely subnormal adolescents aged 15-19 years. The scope for straightforward perinatal preventive action again looks to be small. More may be learnt about the likely causes of severe mental subnormality by the epidemiological approach used in the next chapter.

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6

MENTAL SUBNORMALITY

The other handicap which we wish to consider at this stage is the whole range of intellectual subnormality. Unlike the individual categories of clinically recognisable brain damage discussed in the previous chapter, we can anticipate that the number of children identified as suffering from this handicap, even in our prospectively studied Survey population, will be adequate for simple statistical analysis. The problems of defining mental subnormality have recently been helpfully discussed by Birch et al (1970) and by Kushlick and Cox (1973); the marked differences between reported prevalence rates in different age-groups have been emphasised by Gruenberg (1964). Although no single nomenclature or definition of categories is universally accepted, it is legitimate to make comparisons between populations in which the cases of mental subnormality have been identified at comparable ages and using comparable criteria, including educational and administrative definitions of children requiring special education, and evaluation of all children in the specified age-groups by objective 'intelligence tests'.

Our Survey population is comparable to the Aberdeen population aged 8-10 years which was studied by Birch et al (1970), and we have deliberately adopted the same criteria for subdividing our population, but have, perforce, used our own nomenclature to describe the four main categories:-

(a) Severely subnormal. Clinically identifiable, never admitted to a normal school, mostly attending Training Centre or in residential institution; IQ less than 50.
(b) Educationally subnormal. Administratively ascertained and transferred to special school for the educationally subnormal (ESN) at about the age of 7 years; IQ in the range 50-75, with few exceptions.
(c) Borderline subnormal. Children in normal schools whose IQ is assessed as less than 75 at the age of

Table 6.1

Prevalence of severe mental subnormality (IQ<50) among 9,626 Newcastle singleton legitimate 5 year-olds and 8,274 Aberdeen legitimate 8-10 year-olds

Clinical Category	RATE PER 1,000 (and no. of children) Newcastle	Aberdeen
Total severely subnormal	4.9 (47)	3.7 (31)
Specific syndromes:-		
Down's	1.9 (18)	0.6 (5)
Cretinism, Hurler's	0.3 (3)	-
Postnatal illness	0.3 (3)*	0.1 (1)
Non-specific	2.4 (23)	3.0 (25)

*For details see Table 5.3

Table 6.2

Prevalence of lesser degrees of mental subnormality among 7,168 Newcastle singleton legitimate 9-10 year-olds known to be still resident in the City and 8,274 Aberdeen legitimate 8-10 year-olds

Category	RATE PER 1,000 (and no. of children) Newcastle	Aberdeen
Educationally subnormal:-		
IQ<60	2.4 (17)	2.5 (21)
IQ>=60	14.9 (107)	6.3 (52)
Borderline subnormal	*100.1 (653)	14.9 (123)

*This rate was calculated after excluding 647 children whose test papers were administered incorrectly

9-10 years.

(d) Normal children. The remainder of the children in normal schools at a comparable age.

Our severely subnormal children were identified by the age of 5 years, and for purposes of calculating prevalence, we have taken those known to be still alive and resident in the City at that age as the numerator, and all other Survey children known to be still living in the City as the denominator. The other three groups were identified at the age of 9-10 years when our programme of objective testing was extended outside the City to cover Survey children known to be in normal schools in the County of Northumberland. However, our information about children in ESN schools only covers those still resident in the City, so the prevalence rates have been derived from the Survey children known to be still resident in the City at the age of 9-10 years. The general approach to the problem of administrative ascertainment of ESN children appears to have been much the same in Newcastle and Aberdeen, but the fact that there was no surplus accommodation in the two Newcastle ESN schools might suggest that there would have been less incentive for teachers to recommend children for investigation with a view to ascertainment than in Aberdeen, where 'both the procedures for diagnosis and the facilities for special education...are exceptionally good and make it unlikely that many cases have been missed' (Birch et al, 1970). The Aberdeen procedures for diagnosis included routine objective testing of virtually the whole school population at the ages of 7, 9 and 11 years, using the Moray House Picture Intelligence Test, and the result of this test was the one used by Birch et al (1970) in their investigation. In Newcastle there was no comparable routine testing of the whole population; the Non-Verbal Intelligence Test 5/BD was administered specifically for the purposes of our Survey. Both tests are designed to be administered to groups of children in school, have been validated on large representative populations and are regarded as satisfactory screening tests for mental subnormality.

Prevalence of Mental Subnormality

We need to subdivide the cases of severe mental subnormality into a number of clinically distinct groups before we can make any meaningful comparisons of prevalence rates or assessments of possible causation. We have used the same subdivisions as Birch et al (1970) in Table 6.1, where our findings are compared with theirs. The higher overall rate in our Newcastle population is accounted for almost entirely by our excess of cases of Down's syndrome. Possible explanations are discussed later in this chapter. In our non-specific group there

Table 6.3

Birth weight distribution of Newcastle singleton legitimate children still living in the City at the age of 10 years (expressed as percentages)

BIRTH WEIGHT	Normal (5,740)	Mentally Subnormal Borderline (653)	Educationally (124)	Severely (23)
<=2.5 kg	5.6	8.0	19.4***	21.8**
-4.0 kg	85.0	84.2	75.0	65.2
>4.0 kg	9.4	7.8	5.6	13.0

**p<0.01, ***p<0.001, significant excess over normal

Table 6.4

Social class distribution of Newcastle singleton legitimate children still living in the City at the age of 10 years (expressed as percentages)

SOCIAL CLASS	Normal (5,248)	Mentally Subnormal Borderline (553)	Educationally (102)	Severely (23)
I+II	11.4	1.8	-	13.0
IIIA	11.4	4.7	4.0	17.4
IIIM	52.7	51.2	40.2	43.5
IV+V	22.3	38.2***	52.9***	26.1
Sep. + div.	2.2	4.2	2.9	-

***p<0.001, significant excess over normal

are 14 boys and 9 girls, giving a male/female ratio of 1.6.

The Aberdeen investigation, which included a detailed neurological and psychiatric examination of all index cases, showed the value of subdividing the educationally subnormal group into two subgroups; three-quarters of those with an IQ below 60 had associated neurological or psychiatric abnormalities, while three-quarters of those with an IQ of 60 or more had neither. This practice has, therefore, been adopted in Table 6.2. Within our educationally subnormal group the IQ scores were obtained as a result of individual testing by the City's educational psychologists, using whichever test seemed most appropriate (usually the Stanford-Binet test). Two children scored less than 50, and 3 scored more than 75; the remainder were all within the range 50-75. Sixty-six are boys and 58 girls (giving a male/female ratio of 1.1). The borderline subnormal group was identified by group tests in both cities, as has been mentioned above; its sex ratio in our population is 1.2. There is clearly a progressively larger excess of cases of non-specific mental subnormality in Newcastle as compared with Aberdeen, as we pass from the more severe (where there is no excess) to the milder degrees of handicap. Possible explanations for this are included in the discussion at the end of this chapter.

Incidence of Pregnancy Complications

In view of the possibility that various complications of the pregnancy may cause organic damage to the fetus, we have compared the incidence of certain complications, concerning which information was available in the Maternity Survey data, in four groups of children. In the severely subnormal (non-specific) group we have found an apparently marked excess of severe (but not mild) pre-eclamptic toxaemia and of bleeding before 28 weeks, with a slight excess of antepartum haemorrhage but none of clinical fetal distress. However, in view of the very small total number of children in this group, none of the rates differs significantly from those found among the normal children. There is no suggestion of any comparable excess among the educationally or borderline subnormal children.

Deviations from the normal distribution of birth weight can also be considered a complication of pregnancy, implying disturbances of either the duration or the rate of growth of the babies within a deviant group. The results of a simple analysis, summarised in Table 6.3, suggest that some such deviations are present in some of the mentally subnormal groups in our population. We have excluded from this analysis those subgroups in which the handicap is clearly attributable to genetic

or other specific causes. The obvious excess of babies of low birth weight in the severely and the educationally subnormal groups as compared with the normal group is highly significant, but the excess of heavy babies in the severely subnormal group is not statistically significant in view of the small numbers in the group. The excess of babies of low birth weight appears to be progressive, with increasing severity of mental subnormality. The fact that the educationally subnormal group differs very significantly from the borderline group in this respect ($p<0.001$) is of particular importance as a possible indicator of an element of organic deficiency in the educationally as well as in the severely subnormal group. The relative importance of variations in the duration and the rate of intrauterine growth will be considered in more detail in Chapter 7.

Social Class and Mental Subnormality

The social environment in which the child grows, both before and after birth, is the other factor which could most obviously be related to the causation of mental subnormality. We have, therefore, taken a preliminary look at this problem at this point by comparing the social class distribution (by father's occupation) of those children in each of the same four groups, for whom the relevant information is available. The results are summarised in Table 6.4. The severely subnormal group clearly does not differ significantly from the normal, but there is a striking and highly significant excess of children in Social Classes IV and V in the other two subnormal groups, and a corresponding deficiency of children in Social Classes I, II and IIIA. The deviation from the distribution among normal children is most marked in the group of children who had been transferred to ESN schools. The relationships between social factors and intellectual performance in our population are examined in more detail in Chapter 8.

DISCUSSION

The prevalence rates of nearly all the forms of mental subnormality shown in Tables 6.1 and 6.2 are higher in Newcastle than in Aberdeen, to a degree which is quite startling in some instances. It is clearly of interest, and could throw some light on possible causes of mental subnormality, to try to explain these differences.

In the severely subnormal group (Table 6.1), the higher overall prevalence in Newcastle is entirely accounted for by the subgroups which are attributable to specific causes, particularly Down's syndrome. Our rate for this condition, of 1.9 per thousand singleton legitimate children aged 5 years, is much higher than almost

all other published rates, usually in somewhat older age-groups, as summarised by Kushlick and Cox (1973). The one rate which approaches ours is that of 1.7 per thousand children aged 7½-14½ years reported from Edinburgh by Drillien (1966); her overall rate of 5.0 per thousand for all children with an IQ of less than 50 is also almost identical with ours. The much lower figures for the prevalence of Down's syndrome quoted from elsewhere could theoretically be explained by a lower incidence of the condition amongst all live births, by a higher mortality rate in early childhood, by losses through selective migration away from the community studied, or by less complete ascertainment. We have no means of deciding between these explanations, but it is possible that our higher prevalence is at least partly explained by a continuation of the trend towards increased survival of cases of Down's syndrome noted by Carter (1958). By adding the 8 Newcastle cases of Down's syndrome known to have died or moved away from the City by the age of 5 years to the 18 known to be still in the City at that age, we find that there were 26 known cases among the 13,203 singleton legitimate Newcastle children enrolled in our Survey at the age of 1 month, giving a rate of 2.0 per thousand at that age.

If we turn our attention to the non-specific subgroup of cases of severe mental subnormality, we find that our Newcastle prevalence is actually lower than that reported from Aberdeen. This difference may be explained by the fact that our rate is for cases identified by the age of 5 years, compared with 8-10 years in Aberdeen, (and we know that some Newcastle children were transferred from normal schools into this category after the age of 5 years). We do not think that the truly comparable rates for the two cities would differ at all. As mentioned in the text, investigation of this subgroup in Newcastle revealed an excess of males and of complications of pregnancy, and also a gross excess of low birth weight babies, as shown in Table 6.3, but no distortion of social class distribution, as shown in Table 6.4. In all these respects our findings agree closely with those reported from Aberdeen by Birch et al (1970), and strongly support the suggestion that this type of mental subnormality is due to structural defects of the brain. Unfortunately, the data reported in Chapters 3, 4 and 5 do not suggest that any significant proportion could be prevented by improvements in perinatal clinical management which would be relatively easy to achieve.

When we turn to the lesser degrees of mental subnormality (Table 6.2), we find that the actual prevalence rates for children in the educationally subnormal subgroup with an IQ of less than 60 are almost identical in the two cities. In this subgroup, as well as in the

severely subnormal group, the Aberdeen finding of associated neurological abnormalities in the majority of children strongly suggests that their handicap is due to structural defects. No comparable detailed and formal neurological examination was carried out in our Survey, but there is strong support for the organic basis of the handicap in a further analysis of the birth weight distribution. The highly significant distortion which is shown for the whole educationally subnormal group in Table 6.3 reaches extreme proportions in this subgroup, where we find that 7 of the 17 children (41 per cent) weighed less than 2.5 kg at birth.

In the educationally subnormal subgroup with an IQ of 60 or over, however, our Newcastle prevalence (14.9 per thousand) is more than twice as high as that reported from Aberdeen (6.3 per thousand). This is all the more surprising in view of the suggestion made near the beginning of this chapter that there may have been a greater incentive to achieve ascertainment of such children in Aberdeen than in Newcastle. We have not been able to find any strictly comparable data from other communities which would enable us to decide whether the Newcastle rate is unusually high or the Aberdeen rate unusually low, but it is possible to make some deductions from the data of Drillien (1966). She calculated a true rate of 6.3 per thousand for Edinburgh children aged 7½-14½ years, ascertained as educationally subnormal and with an IQ in the range 50-69. This is identical with the Aberdeen rate for children in the same educational category, but with an IQ range extending up to 75. Clearly, the Edinburgh rate for children in this extended range would be much higher than the Aberdeen rate, but we cannot say by how much. In this subgroup, too, we have found an excess of low birth weight babies (15.9 per cent) and a marked distortion of the social class distribution equivalent to that shown for the whole group in Table 6.4. The Aberdeen findings and our own suggest that there is a less marked element of structural defect in this subgroup, but that social factors play an overwhelmingly important part in the ascertainment of both the educationally subnormal subgroups. The mechanisms involved may include the availability of alternative, private forms of education for children in the upper social classes whose intellectual limitations would otherwise lead to ascertainment. However, it is also clear that the proportion of children with an IQ in the educationally subnormal range is much lower in the upper social classes.

The really startling difference between the figures for Newcastle and those for Aberdeen is in the rates for children in normal schools with an IQ of 75 or less - the Newcastle rate of 100.1 per thousand is actually more than six times higher than the Aberdeen rate. In seeking

to explain this really alarming apparent excess of intellectually inferior Newcastle children, it seems right to start by looking for purely technical differences which could be important between the two sets of data, and since social factors appear to be involved (Table 6.4), we have looked there first. The Aberdeen figures are based upon the total population of the city in the age-group 8-10 years at the time of the study. It was known that there had been a decrease of about 10 per cent in this population since the children were born and that the nett loss (due to a higher rate of migration out than in) had been greater among families in the upper social classes. The Newcastle population upon which our figures are based, consisting of children still living in the City 10 years after they were born, has, of course, been subject to much greater nett losses (since we have not included in-migrants in our Survey); but the resultant distortion of our social class distribution may actually be less, because our losses have been of two kinds which have tended to balance each other. The families which moved right away from the area were, as in Aberdeen, drawn largely from the upper social classes, but the families which moved just outside the City boundary as a result of rehousing by the Local Authority were drawn predominantly from the lower social classes. We cannot, unfortunately, strictly compare the overall social class distributions of the resultant populations of the two cities because of the different way in which Birch et al (1970) subdivided their Social Class III and merged part of it with Classes I and II. However, we can directly compare the percentages in Social Classes IV and V and we find that the Aberdeen figure is 4 per cent higher than our 22.3 per cent. We are less confident of the comparability of their Social Classes I-IIIA, covering 29.7 per cent of their population, with our Classes I+II+IIIA, covering 22.8 per cent of our population, but these comparisons are enough to establish the fact that, in terms of social class derived from the father's occupation, there are no gross differences between the two populations. Both surveys exclude children born to unmarried mothers; we have also excluded the products of multiple birth, which should, if anything, tend to reduce the risk of mental subnormality in our Survey population.

The fact that different objective tests of general intelligence were used to test all children in the relevant age-groups in the two cities is an important technical difference between the two sets of data. The originator of the Moray House Picture Intelligence Test has stated (Pilliner, personal communication, 1972) that he knows of no direct comparison between its results and those of the Non-Verbal Test 5/BD in the same group of

children. Although both tests were standardised on a mean of 100 with a standard deviation of 15, we cannot be sure that their results are directly comparable in practice. However, we can compare our Newcastle results with those obtained in the large (over 16,000 children) population which was considered 'representative of the country as a whole' on which the Non-Verbal Test 5/BD was standardised (NFER, 1965). The mean score in our population is 94.6, 5.4 points below that of the standardisation sample; our standard deviation is 14.7, which is consistent with correct administration and scoring of the test. The downward displacement of the mean by more than a third of a standard deviation clearly indicates a definite impairment of general intelligence in our population as compared with the standardisation sample when measured by this method. The same test was administered to over 3,000 children living on the Isle of Wight by Rutter, Tizard and Whitmore (1970), and their mean scores are 103.4 at the age of 9 years and 102.0 at 10 years, on average more than 8 points above our Newcastle figure. The different social class distribution in the Isle of Wight children may explain some of this difference - 36.0 per cent were in Classes I, II and IIIA (as compared with 22.8 per cent in Newcastle), and 18.4 per cent in Social Classes IV and V (as compared with 22.3 per cent in Newcastle).

We can summarise the situation as follows:-

(1) The prevalence of relatively severe degrees of mental handicap, including all children with an IQ of less than 60, is approximately the same in our population as that reported from Aberdeen by Birch et al (1970) and from Edinburgh by Drillien (1966).

(2) The prevalence of children ascertained as educationally subnormal and transferred to ESN schools, whose IQ is in the range 60-75, is more than twice as high in Newcastle as in Aberdeen, but probably not so much higher than in Edinburgh.

(3) The presence of an alarming excess of Newcastle children in normal schools, whose IQ is less than 75, more than six times the corresponding figure for Aberdeen, is supported by comparing our mean score in the Non-Verbal Intelligence Test 5/BD with the national standardisation sample and that for the population of the Isle of Wight (Rutter et al, 1970).

(4) Biological factors and structural defects are clearly implicated in the causation of the relatively severe degrees of mental handicap; social factors also play a very significant part in the ascertainment of children as educationally subnormal and are of overwhelming importance in determining the prevalence of low general intelligence among children in normal schools. Creak (1965) reported similar findings among children born in our City in 1947.

(5) Our findings are in complete agreement with those reported from Aberdeen by Birch et al (1970), which were an extension of the original observations of Fairweather and Illsley (1960).

(6) We cannot compare our results so fully with those reported from Birmingham by Barker (1966 a) and b)) for a very much larger number of mentally subnormal children, because he gives no information concerning their social background. However, there are many points of agreement over clinical antecedent factors associated with mental subnormality, including the importance of low birth weight and delivery in the absence of any qualified attendant.

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7

VARIATIONS IN INTRAUTERINE GROWTH

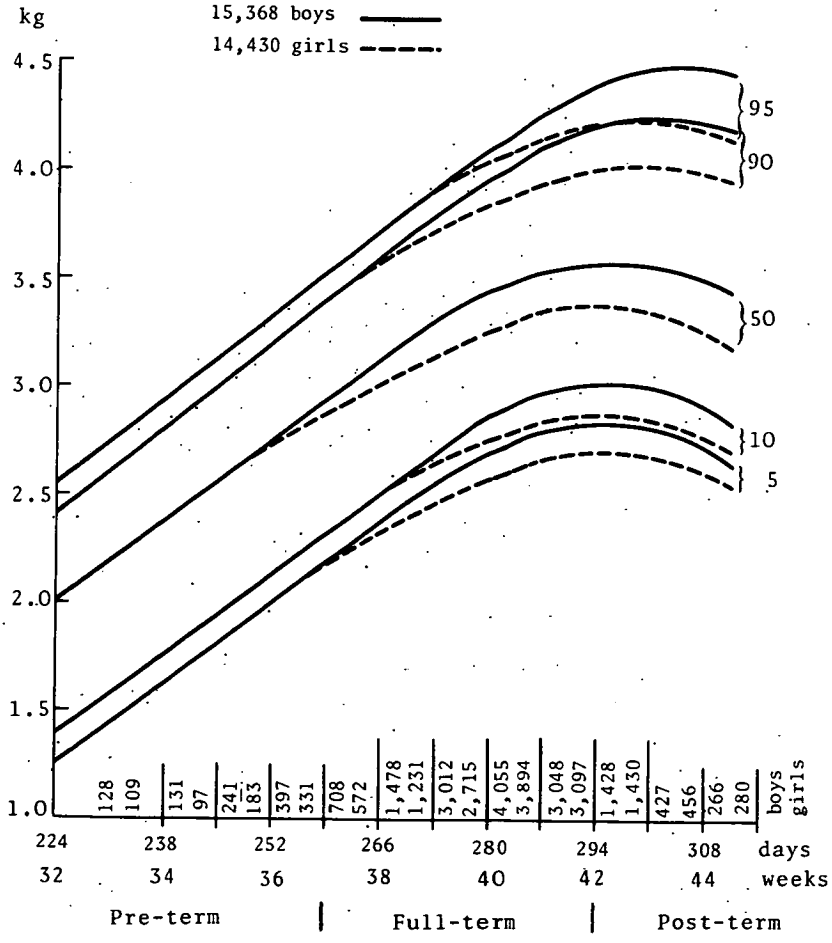
So far we seem to have seen only one adverse antecedent factor behaving in a manner really consistent with the concept of the continuum postulated by Lilienfeld and Pasamanick (1955), and that is low birth weight. This factor is seen to be associated with a high neonatal mortality rate in Chapter 2 and with the various clinical syndromes of brain damage amongst the survivors in Chapters 3-6. The cause of low birth weight must logically be either shortening of the duration or slowing of the overall rate of the intrauterine phase of growth. In the latter case, growth may be slow throughout (resulting in the birth of a stunted baby), or it may be normal up to a point, but giving way to a period of actual weight loss before delivery (resulting in the birth of a baby with a well grown skeleton but wasted subcutaneous tissues). There is an excess of congenital malformations among cases of intrauterine growth retardation (Warkany, Monroe and Sutherland, 1961), but they form a very small proportion of the epidemiologically defined group of 'light-for-dates' babies.

The possibility that low birth weight due to shortening and to slowing of the intrauterine phase of growth may be followed by different sorts of sequelae is being investigated in depth and in detail in the course of the major Special Study of a segment of our Survey population, the results of which are being prepared for publication by Kolvin, Scott and Neligan (1974). It is our purpose in this chapter to deal with this subject relatively briefly, looking at some general effects of variations in the duration and rate of intrauterine growth in our whole Survey population, to enable comparisons to be made with the effects of other types of factor. We are particularly interested in the possibility that if poor intrauterine growth is disadvantageous in terms of later performance, the obverse may also be true and good growth may be advantageous.

Figure 7.1

Centile curves of birth weight/gestational age

Smoothed and corrected from raw data for all singleton legitimate live births to Newcastle residents with 'sure' dates, 1960-8



Intrauterine Growth Chart

The basic tool necessary for epidemiological studies of intrauterine growth is an appropriate chart relating birth weight to gestational age, comparable to the charts used for studying growth in later childhood, which relate weight, height, etc. to postnatal age. Many sets of data and charts purporting to be appropriate for our purpose have been published, relating to different populations selected according to different criteria. Ideally, such data should fulfil two basic requirements. They should relate to a whole cohort of children born to residents in a geographically defined community for which census and other demographic data are available, so that comparisons with other communities can readily be made, and they should be based upon accurate and reliable measurements of both birth weight and gestational age. So far, the only available data which come anywhere near to fulfilling these ideal requirements are those for the population of Aberdeen published by Thomson, Billewicz and Hytten (1968). The raw data for all other large populations studied so far, regardless of their suitability on other grounds, have been marred by a systematic error which is common to them all. This error was precisely defined and a rational explanation for it worked out on data from the first three years' births in the Newcastle Maternity Survey (Neligan, 1965); Gruenwald (1966) reached the same conclusion. The fact that the distribution of recorded birth weights in the gestational age-groups before about 36 weeks is not normal, but grossly skewed or even bimodal, appears to be the result of contamination of the data for truly pre-term births by varying proportions of weights of full-term babies whose gestational ages have been misreported, in all good faith, by their mothers. Such misplaced weights account for up to one third of the raw data in some of the early gestational age-groups. We have used charts based upon the Newcastle Maternity Survey data as they became available for both clinical and research purposes, using the method proposed by Neligan (1965) for correcting the raw data before 36 weeks; this was necessary in the early stages, because no better data were available (Neligan, 1971). However, since the publication of the Aberdeen data (Thomson et al, 1968), we have continued to use our own charts, as illustrated in Figure 7.1, because they seem more appropriate in that they are derived from our own community, and we regard it as the main vindication of the method of correction used, that the resultant smoothed curves correspond closely with the Aberdeen ones. We are still, of course, faced with the problem of how to eliminate the individual misreported 'pre-term' babies which contaminate our raw data, and we have chosen to do so by excluding from the results reported in this chap-

Figure 7.2

Effect of a) birth weight in kg
b) birth weight/gestational age centile
upon Goodenough IQ at 5 years in 3,603 boys

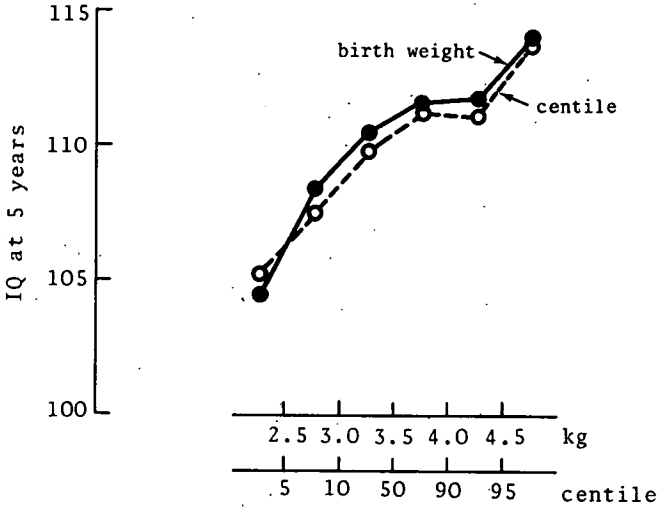
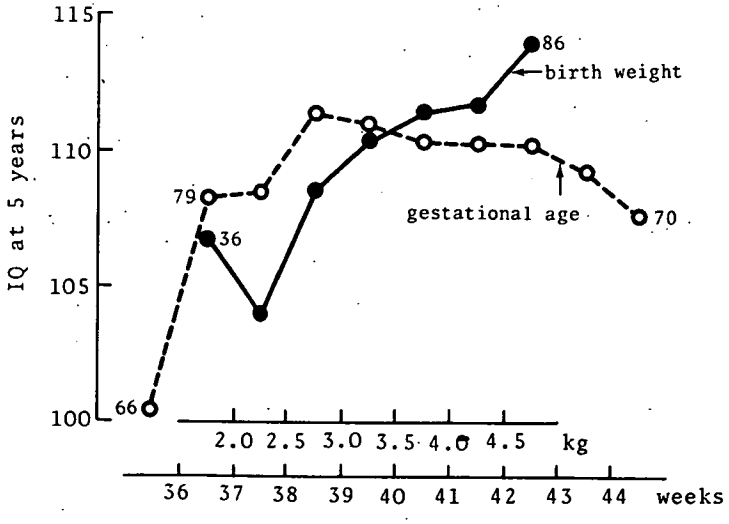


Figure 7.3

Effect of a) birth weight in kg
b) gestational age in weeks
upon Goodenough IQ at 5 years in 3,603 boys



ter all babies with a gestational age of less than 37 weeks whose birth weight falls above the corrected 90th centile curve. By this procedure we have presumably also excluded some of the heaviest truly pre-term babies.

Gestational Age and Birth Weight

Babies who are born too soon may presumably be at increased risk of brain damage (either through associated adverse factors such as placenta praevia or pre-eclamptic toxaemia in the mother, or as a consequence of complications such as hyperbilirubinaemia or apnoeic attacks in the baby), but we would expect their subsequent growth to be normal, unless they are also light-for-dates at birth or are allowed to undergo a period of postnatal malnutrition equivalent to the intrauterine malnutrition of the light-for-dates baby (Davies and Davis, 1970). Those who are born light-for-dates, however, may presumably be at increased risk of impaired physical as well as cerebral development, if their period of intrauterine malnutrition should produce permanent effects upon the growth of their bodies and their brains in any way comparable to those experimentally produced in newborn rats by Widdowson and McCance (1963) and those discussed by Dobbing (1970). The results of a preliminary analysis of a small proportion of our Survey data appeared to be consistent with these possibilities (Neligan, 1967); we have now attempted to explore these ideas further by looking at all the singleton legitimate Survey children born during 1960-2 for whom we have acceptable data concerning the two parameters which are essential for the study of intrauterine growth.

Birth weight is no problem; it was recorded for 99.9 per cent of our children and we have no reason to doubt that these recordings were accurate. Gestational age as calculated from the mother's menstrual dates, however, is another matter altogether. No less than 16 per cent of our Maternity Survey mothers had to be excluded because they did not have 'sure' dates to within five days either way (Neligan, 1965). Even among those who did, we have good reasons for doubting that they were correct in up to one third of those reporting a very short period of gestation, and so have had to exclude all babies with a gestational age of less than 37 weeks whose birth weight falls above the 90th centile curve, for reasons described above. Unfortunately, the birth weight alone tells us very little about the intrauterine growth of the individual baby; the only thing we can be sure about is that if the weight is high, the overall growth rate must have been rapid, but even then, if we do not know the corresponding gestational age, we do not know just how rapid. If the birth weight is either average or low, only by relating it to gestational age can we tell if the rate

was rapid, average or slow. We have confined all the analyses reported in this chapter to those children for whom we have acceptable data concerning both birth weight and gestational age, because we wish to explore the effects of these two parameters both individually and in relation to each other. Clearly, any such comparisons require to be made in the same children. We have continued to treat the data for boys and girls separately, as in previous chapters, (although it is a theoretical advantage of birth weight/gestational age centiles derived from sex-specific curves that they eliminate the sex differences in birth weight as measured). All children who were in normal schools at the relevant ages and for whom we have appropriate measurements of performance have been included.

In spite of the advantages of using birth weight/gestational age centiles when dealing clinically with individual babies or when studying the effects of intrauterine growth in depth, this may be less important in more general epidemiological studies because of the fact that the great majority of babies are born at a stage of gestation (from 38 weeks onwards) when the nett slope of the curves is almost horizontal, so that weight as measured is roughly proportional to growth. Therefore, since it seems likely that accurate and reliable birth weight measurements will continue to be available in a much higher proportion of babies than comparable data concerning gestational age for the foreseeable future (in fact, until some new method of measuring the latter is worked out), we have attempted to compare the effects of birth weight as measured, and as expressed in terms of birth weight/gestational age centiles, upon the subsequent performance of our Survey children. We have found the effects to be so similar that it seems justifiable to use simple birth weight for our present purposes, so that any conclusions which we reach can have the widest possible application. An example of the relationships we have found is illustrated in Figure 7.2, where the difference between the two curves for Goodenough IQ at the age of 5 years is nowhere as much as one IQ point. (The results of a more sophisticated analysis of these relationships are reported in Chapter 9.)

We have directly compared the effect of birth weight with that of gestational age upon the Goodenough IQ at the age of 5 years. The result for boys is shown graphically in Figure 7.3. It can now be seen that the progressive fall in mean score with diminishing birth weight shown in Figure 7.2 does not continue into the weight-group below 2.0 kg (which we have shown separately here to illustrate this point), whereas the progressive fall in mean score with diminishing gestational age below 38 weeks does continue into the group below 36 weeks. At

the other end of the range, the progressive rise in mean score with increasing birth weight continues into the heaviest weight-group, suggesting that the beneficial effect of good intrauterine growth upon later performance is continuous; in contrast, the fall in mean score with increasing gestational age beyond full term, though slight, suggests that prolongation of pregnancy beyond a certain point begins to have a harmful effect upon later performance, reminiscent of the effect upon birth weight illustrated in Figure 7.1.

Social Factors

It was mentioned earlier that adverse sequelae of low birth weight may be due directly to the biological effects of the disturbance of intrauterine growth or they may be due to associated prenatal or postnatal medical complications. We must now consider the possibility that some of the effects shown in Figure 7.3, whether adverse or beneficial, may be due to associated social factors. To see whether this is likely, we have looked first for a systematic association between the child's social class at the time of the school entry medical examination and the two parameters of intrauterine growth included in Figure 7.3. Table 7.1 makes it clear that there are some associations, of a kind which might go some way towards explaining the effects, both of birth weight and of gestational age, shown in the figure. There is a higher proportion of children in Classes I+II and IIIA, and a lower proportion in Classes IV+V, in those weight-groups and gestation groups whose mean IQ is highest, and vice versa. However, a simple breakdown by social class of the data from Figure 7.3 shows that the biological factor's effect persists, to some extent at least, within social class groupings. The results for birth weight are illustrated in Figure 7.4, where the harmful effect of low birth weight is clearly shown in all groupings, but the beneficial effect of high birth weight is seen only in Social Classes I+II. In general, the results for gestational age groupings show similar effects, and so do the results for girls, except that the magnitude of the effect is less than for the boys. For instance, while the difference between the mean IQ of low birth weight boys in Social Classes IV+V and that of high birth weight boys in Social Classes I+II is 25 points (Figure 7.4), the corresponding difference in the girls is only 11 points.

Postnatal Growth

We are also interested in the possibility that variations in intrauterine growth may have an effect upon postnatal growth, at least in the statistical sense of the term, in which case there may be an underlying bio-

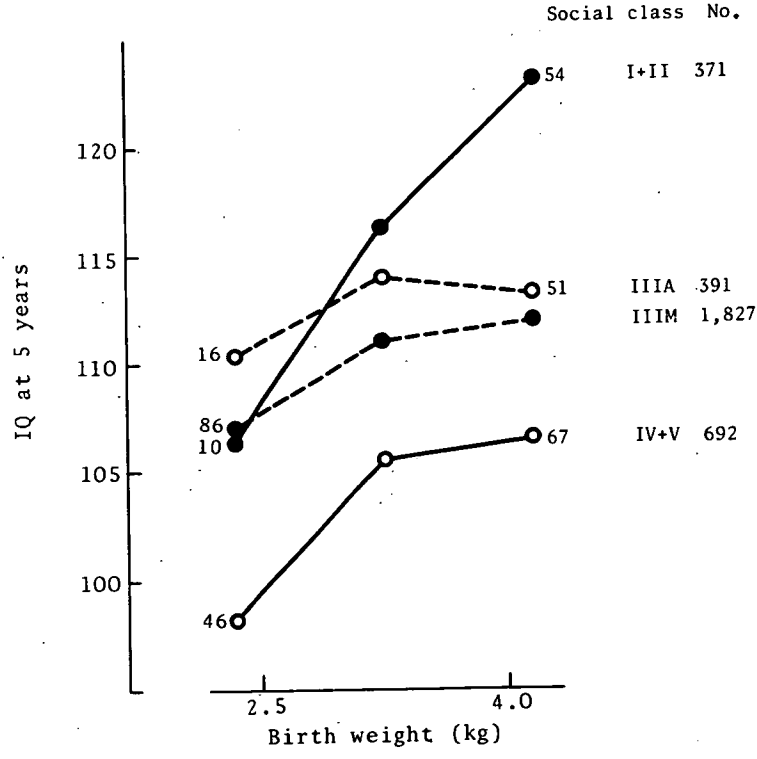
Table 7.1

Percentage distribution of social class at school entry related to birth weight and gestational age groups among the boys illustrated in Figure 7.3 for whom the relevant information is available

	SOCIAL CLASS			
	I+II	IIIA	IIIM	IV+V
Birth Weight				
<2.5 kg	6.3	10.1	54.4	29.1
2.5-3.9 kg	11.2	11.9	55.7	21.2
>=4.0 kg	13.9	13.1	55.8	17.2
Gestational Age				
<37 weeks	9.4	3.9	56.2	30.5
37-41 weeks	11.7	12.5	55.7	20.1
>=42 weeks	9.3	10.9	55.1	24.7

Figure 7.4

Effect of birth weight and social class at 5 years upon Goodenough IQ in 3,281 boys (322 of those included in previous two figures excluded because father's occupation not known, or parents separated or divorced by this stage)



logical effect as well.

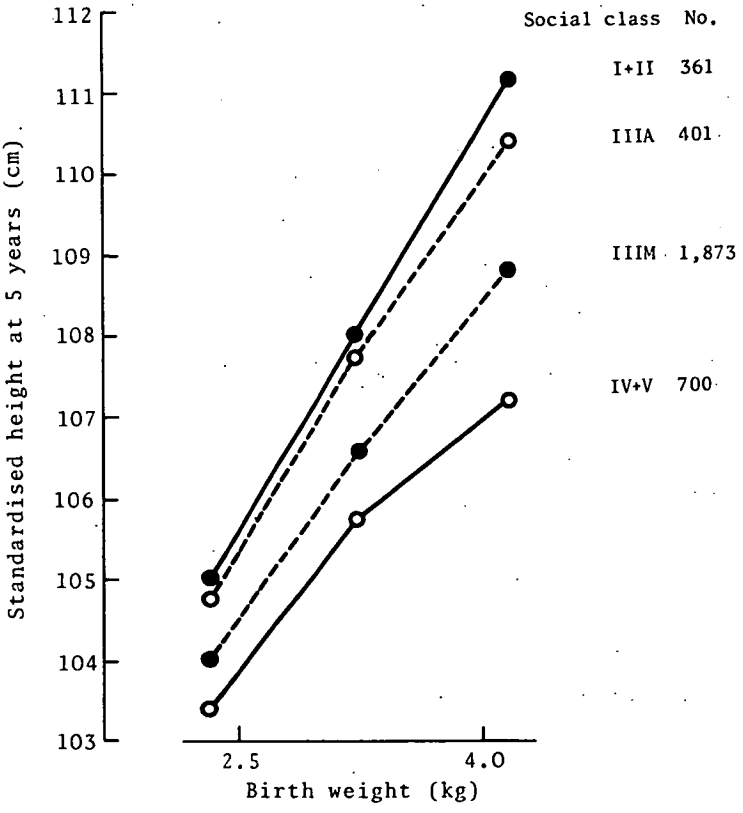
We predicted earlier that variations in the rate of intrauterine growth would be found to produce much greater effects upon the rate of postnatal growth than variations in its duration, and we accepted the idea that, for our present purposes, it is reasonable to use simple birth weight as an indicator of the rate of intrauterine growth. Our prediction is readily confirmed by an analysis similar to the one whose results are shown in Figure 7.3, but this time comparing the effects of birth weight with those of duration of gestation upon standardised height at the age of 5 years. (We have taken height at 5 and 10 years to be a better qualitative indicator of growth than weight measured at the same ages, particularly if we accept the popular value judgment that 'bigger is better'; and height is less liable to transient fluctuations.) The progressive rise in mean height from the lowest to the highest birth weight group is 5.5 cm, whereas the maximum rise with increasing gestational age (reaching its peak at 41-2 weeks) is 2.25 cm. However, in view of the association between both these parameters and social class distribution, which we demonstrated in Table 7.1, we have proceeded directly to a further analysis by social class groupings, whose results are illustrated in Figure 7.5. In this case, it looks as if the beneficial effect of good intrauterine growth is as marked as the harmful effect of poor intrauterine growth. Of course, some of the height difference between the heaviest and the lightest groups will be accounted for by differences in their mean length at birth, but we do not have any data concerning this parameter in our Survey population, and are thus not able to make any accurate correction to allow for this factor. The effect of variations in the duration of intrauterine growth almost disappears when tested by a type of analysis similar to that illustrated for birth weight in Figure 7.5; the differences between the mean heights of boys in the pre-term (shortest) and the post-term (tallest) sub-groups in the four social class groups range from 1.3 cm to only 2.4 cm.

Results at 5 and 10 Years

Another way of throwing some light upon the relative magnitude and practical importance of the effects of these biological and social factors is to see how they change between the ages of 5 and 10 years. We would expect differences due to the effects of social factors to increase with increasing duration of exposure to different environments, whereas those due to the effects of prenatal or perinatal biological factors might be expected to become blurred with the passage of time under the modifying influence of different environments, unless very fundamental biological processes are involved. The

Figure 7.5

Effect of birth weight and social class at 5 years upon standardised height of 3,335 boys (all those for whom height data were available and whose parents were not separated or divorced by that stage)



apparent effects of differences in birth weight upon IQ and standardised height at 5 and 10 years are summarised in Table 7.2, and those of differences in gestational age in the same children are summarised in Table 7.3. The numbers of boys covered by each of the four measurements range from 2,635 (height at 10 years) to 3,603 (IQ at 5 years); the corresponding numbers of girls range from 2,602 to 3,487. The relatively small number of children for whom we have height measurements at the age of 10 years is explained by the fact that at that age no height measurements were made in children who were in County schools and some private schools.

By comparing the results in Tables 7.2 and 7.3, we can readily confirm that decreasing birth weight is associated with impaired performance in both sexes, as measured by both IQ and height at the age of 5 years, and that the differences persist at approximately the same magnitude at the age of 10 years; we can also confirm that gestational age differences produce less marked effects, partly due to the fact (illustrated in Figure 7.2) that the best performance is found in the middle, full-term, group in almost all instances, because prolongation of gestation to 42 weeks or longer produces some deterioration in performance, though less marked than that produced by shortening to less than 37 weeks. Again the differences in performance at the age of 10 years are of the same order of magnitude as at 5 years. Bearing in mind the associations demonstrated in Table 7.1, the most likely explanation of these findings would appear to be that they are the result of interaction between biological and social factors.

Direct visual comparison of these results is made difficult by the fact that different units are used for measuring IQ and height, and that the ranges are different at 5 and 10 years even when the same units are used. We have, therefore, converted all measurements to a common scale, of standard deviations above or below the mean for the population, to enable meaningful direct visual comparisons to be made. The results for the boys in Table 7.2 are shown in this form in Figure 7.6. It is clear that birth weight differences have more effect upon height than upon IQ at both 5 and 10 years, but that the apparent effect upon IQ tends to increase between 5 and 10 years.

DISCUSSION

The adverse effects of low birth weight upon the quality of the survivors of the neonatal period have long been recognised and extensively documented. To be clinically useful, any further investigation of this subject requires to separate the effects of shortening from those

Table 7.2

Effect of differences in birth weight upon IQ and standardised height at 5 and 10 years

	BIRTH WEIGHT			
	<2.5 kg	2.5-3.9 kg	>=4.0 kg	All
Boys				
Goodenough IQ at 5 years	104.5	110.5	112.2	110.4
Height at 5 years (cm)	103.9	106.6	108.9	106.7
Non-verbal IQ at 10 years	90.6	94.0	97.6	94.3
Height at 10 years (cm)	132.5	135.5	138.6	135.8
Girls				
Goodenough IQ at 5 years	111.1	115.4	117.7	115.4
Height at 5 years (cm)	103.1	105.7	108.9	105.8
Non-verbal IQ at 10 years	90.8	96.0	98.2	95.8
Height at 10 years (cm)	131.8	134.8	138.6	134.9

Table 7.3

Effect of differences in gestational age upon IQ and standardised height at 5 and 10 years

	GESTATIONAL AGE			
	<37 weeks	37-41 weeks	>=42 weeks	All
Boys				
Goodenough IQ at 5 years	105.1	110.7	110.2	110.4
Height at 5 years (cm)	105.0	106.8	107.0	106.7
Non-verbal IQ at 10 years	91.5	94.4	94.1	94.3
Height at 10 years (cm)	134.6	135.8	135.8	135.8
Girls				
Goodenough IQ at 5 years	113.7	115.7	114.2	115.4
Height at 5 years (cm)	104.6	105.9	105.6	105.8
Non-verbal IQ at 10 years	91.2	96.2	94.9	95.8
Height at 10 years (cm)	133.3	135.1	134.4	134.9

of slowing of the intrauterine phase of growth, and we are attempting this elsewhere in reporting the results of a study which is designed to measure the quality of only 400 survivors over a wider range of performance and in much greater depth (Kolvin, Scott and Neligan, 1974).

Our first objective in this chapter has been to compare the effects of differences in duration of gestation with those of differences in birth weight (which we have accepted as an adequate indicator of intrauterine growth rate for our present, very general, purposes). We have looked at both over the whole of their distribution, subdividing them in such a way as to leave adequate numbers of children within each birth weight or gestational age interval.

The adverse effect of both shortening and prolongation of the duration of gestation, which we have illustrated in terms of the Goodenough IQ score of boys aged 5 years (Figure 7.2), has been reported previously in terms of a verbal reasoning test at 11 years (Barker and Edwards, 1967; Record, McKeown and Edwards, 1969), and of both reading ability and social adjustment at 7 years (Davie, Butler and Goldstein, 1972). The adverse effects of both factors upon perinatal mortality rates have long been known, and the progressive change in the slope of the curves of birth weight from about 38 weeks of gestation onwards, as illustrated in Figure 7.1, has given strong support and precision to the idea that the placenta's aging beyond a certain optimal point, which must vary greatly in different women, impairs its effectiveness in its nutritional as well as its more acutely vital functions. Long-term adverse effects could, therefore, be due to the biological effects of nutritional impairment at a critical stage of development as well as to the effects of complications such as hypoxia or hypoglycaemia in the neonatal period. However, the social associations illustrated in Table 7.1 obviously complicate the picture and need to be allowed for before reaching any firm conclusions about the long-term biological effects of variations in the duration of gestation.

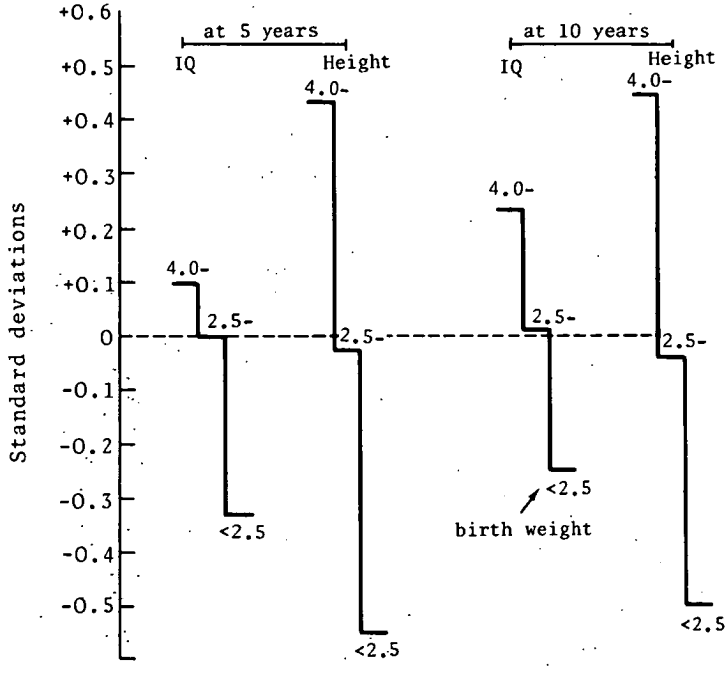
The continuously progressive beneficial effect of increasing birth weight upon Goodenough IQ score at the age of 5 years is clearly a reflection of good intrauterine growth (Figure 7.2), which in turn must logically be a reflection of good placental function in the absence of rare complications such as maternal diabetes mellitus. A very similar finding was reported by Record et al (1969) in terms of a verbal reasoning test at the age of 11 years.

The fact that physical growth (as reflected by measured height) and mental development (as reflected by measured general intelligence) are affected in just the

Figure 7.6

Effect of birth weight (< 2.5, 2.5-, 4.0- kg) upon IQ and standardised height at 5 and 10 years in boys. Results from Table 7.2 converted into comparable units (standard deviations)

Results expressed as $\frac{\text{(mean for birth weight group)-(mean for all boys)}}{\text{standard deviation for all boys}}$



	at 5 years		at 10 years	
	IQ	Height	IQ	Height
Standard deviations	17.5	5.1	14.7	6.4

same way, as is apparent from inspection of Tables 7.2 and 7.3, could be explained in two different ways. Either the biological factors associated with differences in birth weight or duration of gestation could be affecting the subsequent growth of the body and of the brain of the children concerned, or the associated social factors (as illustrated in Table 7.1) could again be producing at least part of the observed effects.

Our first measurement of the performance of our children was carried out during their first term in school when they were about 5 years old in order to minimise the modifying effects of differences in their social and educational environments upon their biological potential. The fact that the same, or similar, measurements were repeated when they were about 10 years old gives us a chance of comparing the magnitude of the effects after more prolonged exposure to these environmental differences. The findings illustrated in Figure 7.6 show that the effects of differences in birth weight upon physical growth are almost identical at the age of 10 to those at the age of 5 years, suggesting that the biological influences are of paramount importance. The effects upon IQ, however, are slightly greater at 10 than at 5, suggesting that associated environmental factors tend to enhance the effects of birth weight.

If we look back to where we started this chapter by examining how well our findings fit in with the concept of the 'continuum of reproductive casualty' (Lilienfeld and Pasamanick, 1955), we are bound to conclude that the fit is remarkably good. Variations in both birth weight and gestational age, which have been shown to increase the risk of perinatal mortality or mental subnormality, also produce an adverse effect upon the quality of the survivors, which is only demonstrable by large-scale epidemiological studies. But there are also associations with adverse social factors, which represent an extension of the continuum in a different direction, and we aim to examine their effects in the next chapter.

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8

SOCIAL FACTORS

That a child's social class background can have a major effect upon various aspects of his performance has long been recognised, and few would doubt that, in this context, the word 'effect' can legitimately be used in its causal as well as its purely statistical sense. Social factors may presumably modify or exaggerate the effects of associated clinical or biological factors, and these associations may be either coincidental or systematic (Illsley, 1967). We have already illustrated these effects in relation to perinatal mortality (Table 2.5), to mental subnormality (Table 6.5) and to the performance of children in normal schools (Chapter 7). We have so far defined the child's background simply in terms of social class derived from the father's occupation, which was described by Birch et al (1970) as being 'perhaps the most effective single criterion available and the one most frequently used in socio-medical research'. We will continue to rely primarily upon this simple criterion for our present purpose of exploring the relationships between social factors and performance a little more deeply, but we also intend to assess the effects of differences in the quality of child care within a single social class later in this chapter.

The age at which the children are tested seems to us to be one of the most important variables to be taken into account when attempting to evaluate the relative strength of the effects of various social and biological factors upon different aspects of their performance. As mentioned in the previous chapter, the older they are, the more opportunity will there have been for differences in environmental factors to modify the development of their innate biological potential; and differences in quality of schooling may be expected to have a particularly marked effect upon intellectual performance. As far as we know, no other large, geographically defined population of children has been objectively assessed, taking into account the effects of biological and social factors,

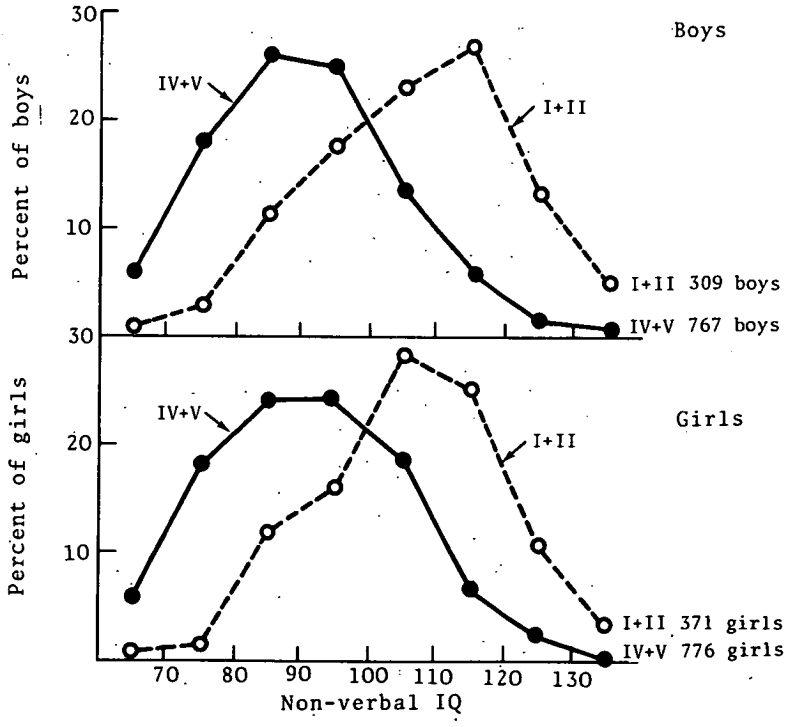
Table 8.1

Effect of social class upon IQ at 5 years (Goodenough Draw-a-Man Test) and 10 years (Non-Verbal Test 5/BD)

SOCIAL CLASS	MEAN IQ AT 5 YEARS		MEAN IQ AT 10 YEARS	
	Boys	Girls	Boys	Girls
I+II	116.3	120.3	105.8	105.3
IIIA	113.5	118.2	98.9	99.6
IIIM	110.7	115.1	93.6	94.5
IV+V	105.5	111.2	89.0	90.6
All	110.5	115.2	94.2	95.3

Figure 8.1

Effect of social class upon non-verbal IQ at 10 years: distribution in social class group I+II compared with IV+V



until after at least two years in school. The British cohort of 1946 was first tested at the age of 8 years (Douglas, 1964), and the 1958 cohort at 7 years (Davie, Butler and Goldstein, 1972), as were the Aberdeen cohorts of 1950-5 (Illsley, 1967) and 1950-4 (Birch et al, 1970). The Isle of Wight population tested by Rutter, Tizard and Whitmore (1970) was aged 8-10 years.

We carried out our first round of objective testing during our children's first term at school, at a mean age of approximately 5 years. This was the first time at which they were conveniently accessible for group tests to be administered (through the invaluable co-operation of all the teachers concerned), and we hoped that their performance would, as yet, be distorted relatively little by the social, and hardly at all by the educational, differences in their environments. When we retested them after an interval of about five years we expected to see an increase in the magnitude of any differences in performance which might have been caused by such differences in their environments.

The two measures of performance which we attempted to record at both 5 and 10 years were general intelligence and physical growth. Some of the results for the former are summarised in Table 8.1, where the children have been subdivided into four social class groups by combining Classes I and II and Classes IV and V. This has been done to ensure that each group contains enough children for purposes of further statistical analysis. As a result, the smallest number of children in any one group is 309 (boys aged 10 years in Classes I+II). It is clear that the effects of social class differences are already very obvious by the age of 5 years; the mean scores of the Goodenough Draw-a-Man test show a steadily progressive fall of 10.8 points in the boys and 9.1 points in the girls as we pass from Social Classes I+II to IV+V. By the age of 10 years, when the Non-Verbal Intelligence Test 5/BD was used, the corresponding differences between the highest and lowest mean scores can be seen to have increased to 16.7 and 14.7 points respectively for boys and girls. At both ages and in both sexes, these differences between the mean scores of the different social class groups are due to a shift in the whole distribution of the individual scores rather than to any noticeable change in the shape of the curves. This point is illustrated in Figure 8.1, where only the two extreme social class groups in each sex are shown, in the interest of clarity. The curves for intermediate classes lie in intermediate positions.

Turning to physical growth, we have confined our present analysis to standardised height at 5 and 10 years, rather than weight, for reasons described in the previous chapter. Our findings are summarised in Table 8.2. This

Table 8.2

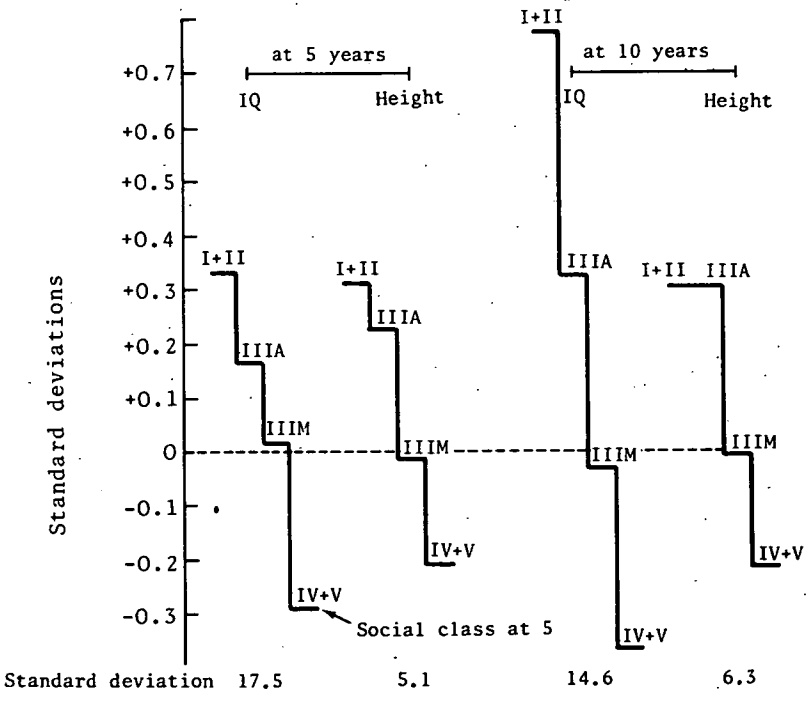
Effect of social class upon standardised height at 5 and 10 years

SOCIAL CLASS	MEAN HEIGHT AT 5 YRS (cm) Boys	Girls	MEAN HEIGHT AT 10 YRS (cm) Boys	Girls
I+II	108.3	107.4	137.7	137.1
IIIA	107.9	106.9	137.7	136.8
IIIM	106.6	105.5	135.6	134.6
IV+V	105.6	104.6	134.3	133.5
All	106.7	105.7	135.7	134.9

Figure 8.2

Effect of social class upon IQ and standardised height at 5 and 10 years in boys. Results from Tables 8.1 and 8.2 converted into comparable units (standard deviations)

Results expressed as $\frac{\text{(mean for social class group)-(mean for all boys)}}{\text{standard deviation for all boys}}$



time the progressive fall in values is less marked between the upper two groups in each column, and there is no real difference between boys in group I+II and group IIIA at 10 years. This suggests that social factors modify the innate biological potential for growth less than they modify innate general intelligence as we have measured it, at least in the upper part of the social scale.

In order to allow valid visual comparison between different variables and different ages, we have again converted the results (from Tables 8.1 and 8.2) into comparable units in the shape of standard deviations, as shown in Figure 8.2. It is obvious at a glance that the effects of social class differ markedly from those of birth weight as illustrated in Figure 7.6 (in the same units and to the same scale). The effect of social class upon IQ at 5 years is somewhat greater than upon height at the same age, but the most striking feature is the increase in the strength of the effect upon IQ by the age of 10 years, whereas the effect upon height is the same as it was at 5. The spread of the mean IQ scores for our four social class groups can be seen to have increased from 0.62 standard deviations at 5 years to 1.1 standard deviations at 10 years. The corresponding spread of mean standardised heights is about 0.5 standard deviations at both ages. Between the three birth weight groups shown in Figure 7.6, the spread of mean IQ score is about 0.45 standard deviations and of mean standardised height 1.0 standard deviations at both ages.

The other main type of performance which we attempted to measure in all our children who were still accessible at the age of 10 years was behaviour. The Behaviour Inventory Scale B(2) was completed by their teachers for all the children who did the objective group tests in normal schools at that age, and scored by Survey staff according to the instructions of Rutter (1967). This enabled us to categorise each child's behaviour as 'normal' or as 'abnormal' in a 'neurotic', 'antisocial' or 'undifferentiated' way, depending upon the scoring of specified items. The two perinatal factors, method of delivery and delay in onset of regular respiration, do not show any consistent relationship with abnormalities of behaviour. Differences in birth weight do show such a relationship, though it is small; among the boys, the proportion whose behaviour was scored as normal rises steadily from 75.3 per cent of those with a birth weight of 2.5 kg or less to 81.5 per cent of those who weighed more than 4.0 kg; the corresponding rise among girls is from 85.0 per cent to 89.1 per cent. However, in view of the associations between birth weight and social class summarised in Table 7.1, we need to know if there are any consistent relationships between class

Table 8.3

Behavioural diagnosis by Teacher's Inventory at 10 years expressed as a percentage of each class/sex group assigned to the specified diagnostic categories

SOCIAL CLASS	BEHAVIOURAL DIAGNOSIS				No. in Class
	Normal	Neurotic	Antisocial	Undifferentiated	
Boys					
I+II	90.2	2.4	5.7	1.7	296
IIIA	85.9	3.9	9.7	0.5	362
IIIM	80.7	4.6	13.0	1.7	1,907
IV+V	72.4	4.4	20.9	2.3	803
Girls					
I+II	95.1	1.6	3.0	0.3	368
IIIA	92.0	2.4	5.1	0.5	374
IIIM	89.5	4.3	5.2	1.0	1,744
IV+V	82.1	6.6	9.3	2.0	804

Table 8.4

Percentage of families whose 'Care of Child' was graded as 'Good', 'Average' or 'Poor' in Third Year Health Visitor's Proforma

SOCIAL CLASS	'CARE OF CHILD' GRADE			No. in Class
	Good	Average	Poor	
I+II	75.8	23.3	0.9	881
IIIA	60.8	38.6	0.6	830
IIIM	45.4	51.0	3.6	3,323
IV+V	29.3	64.1	6.7	1,366

and behaviour before accepting this finding at its face value.

The results summarised in Table 8.3, in fact, show that the associations between social class and behaviour are stronger by far than those mentioned above. Among both boys and girls, there is a steadily progressive fall in the proportion of 'normal' children as we pass from Classes I+II to IV+V, the total spread being 17.8 per cent in the boys and 13.0 per cent in the girls. There is also a progressively greater excess of 'abnormals' among the boys compared with the girls in each class-group, and it is no surprise to find that this excess is in the 'antisocial' rather than the 'neurotic' type; but even in the girls, there is a threefold increase in the proportion categorised as 'antisocial', as well as a fourfold increase in the 'neurotic', as we pass from Classes I+II to IV+V.

'Care of Child'

So far we have been comparing the effects of differences across the whole range, including the extremes, of social environment in our community, and we have been using a very indirect and inferential criterion for categorising these differences between individual children, namely their father's occupation. We now want to make use of some of the additional social information which we have available through the Third Year Health Visitor's Proforma for children born in 1961-2, which is more directly focussed upon the quality of the environment provided by the family as it might be expected to affect the well-being of the child.

The particular item with which we are concerned here was entitled 'Care of Child', and the health visitor was requested to grade the family as A (good), B (average) or C (poor). The written instructions stated:-

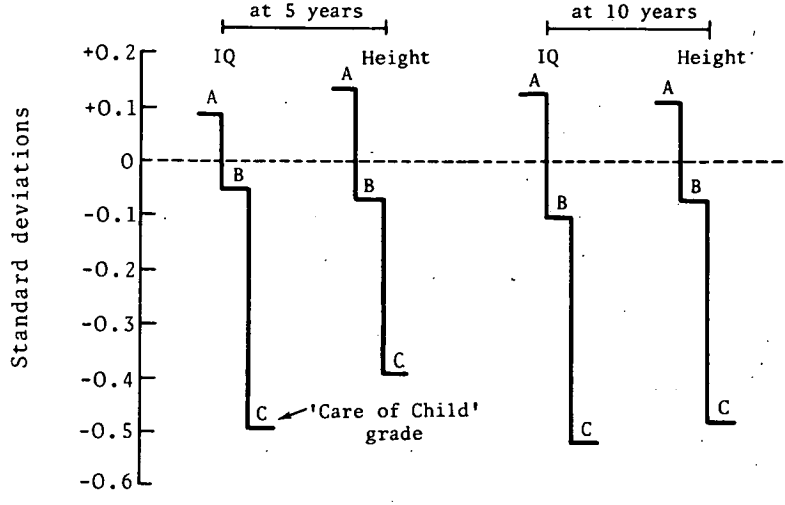
> 'This is intended to include adequacy of food and clothing and of supervision by a responsible adult or older child: cleanliness: seeking appropriate help in case of illness: evidence of affectionate parental interest in the child. If good on all counts, ring A (?top 20% of total community in City), if poor, ring C (?bottom 20%), if average, ring B (?middle 60%).'

In retrospect, we regret having included such diverse aspects of 'care' in the one item; as a result, we cannot identify the effects of differences in each of these aspects. It is also clear from our results that we should have used a more stringent definition of A and a less restrictive definition of C if we wanted each to include about 20 per cent of our families. This point is obvious from Table 8.4, which shows the grading of the families of the children for whom a third year proforma was avail-

Figure 8.3

Effect of 'Care of Child' grade upon IQ and standardised height at 5 and 10 years in Social Class IIIM (boys and girls)

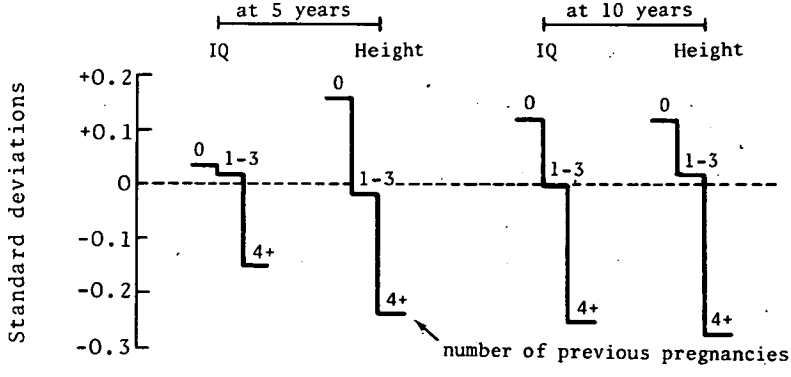
Results expressed as $\frac{\text{(mean of grade)-(mean of Class IIIM)}}{\text{standard deviation for Class IIIM}}$



	IQ (5 yrs)	Height (5 yrs)	IQ (10 yrs)	Height (10 yrs)
Standard deviation	17.2	5.2	14.0	6.4

Figure 8.4

Effect of number of previous pregnancies in mother's history upon IQ and standardised height at 5 and 10 years in Social Class IIIM (boys and girls)



	IQ (5 yrs)	Height (5 yrs)	IQ (10 yrs)	Height (10 yrs)
Standard deviation	17.1	5.2	13.9	6.3

able, in each social class group; but the main purpose of this table is to show the progressive decrease in the proportion of families graded A, and the corresponding increase in the proportion graded C, as we pass from Classes I+II to IV+V.

However, some of these differences could be reflecting limitations imposed by extreme economic and domestic inequalities, or social disorganisation, and we think it should be possible to learn more about the specific effects of differences in the quality of 'care of child' if we confine our attention to a single social class group. Class IIIM looks to be suitable for our purposes, since it includes a large number of families (over half the total) and excludes both social extremes. Within the general discription of 'skilled manual workers' it includes, to quote examples taken at random, miners working at the coal face, dental mechanics, masons, fitters and electricians and drivers of commercial vehicles. Since the grading of the families for our present purpose depended upon information available from the third year proforma, we have based our social classification for the purposes of this analysis upon the father's occupation as reported in the same proforma.

The effect of the health visitor's grading for each child in Social Class IIIM at the age of 3 years upon IQ and height at the ages of 5 and 10 years is shown diagrammatically in Figure 8.3. The procedure and scale are both identical with those of Figure 8.2, so that direct visual comparisons are possible, with the one difference that the results for boys and girls have been combined in Figure 8.3 to minimise the unreliability which could be a consequence of the comparatively small number of families in grade C.

It is surprising, and of great interest, to find that in certain variables (IQ at 5 years and height at 10) there is as great a spread between children in grade A and grade C families within Social Class IIIM as between the extremes (Social Classes I+II and IV+V) in Figure 8.2. In each case, the spread is about 0.5-0.6 standard deviations. The spread of height at 5 years between grades A and C is, however, less (just under 0.5 standard deviations), which implies that the spread of height at 10 years is not due primarily to differences in mean length at birth; this implication is supported by the finding that there is no appreciable difference between the mean birth weights of the children in grades A, B and C. The spread of IQ at 10 years between grades A and C is more than 0.6 standard deviations, but not nearly as great as the spread of 1.1 standard deviations between Classes I+II and IV+V. It is also noteworthy that this quite exceptional spread in IQ between the extremes of social class is accounted for largely by a unique ad-

vantage of children in Classes I+II over those in IIIA, whereas the greater part of the spread between the grades in Figure 8.3 is accounted for by the disadvantage of children in grade C compared with those in grade B.

Pregnancy Number

The number of previous pregnancies which a mother has had, regardless of the outcome, has been shown repeatedly, in national cohort studies (Douglas, 1964; Davie et al, 1972) and in a community study (Illsley, 1967), to affect the performance of her child. The implications are clearly complex, both biological (with respect to reproductive performance and maternal age, for instance) and social (with respect to the status and degree of organisation of the family, and the number of older children, in the environment). We do not intend to try to unravel all these complexities here, but will seek to determine whether, in our population, the mother's pregnancy number affected the children's performance, as assessed at 5 and 10 years, in a manner which could not be explained by the known association of family size with social class.

In our population, this association is not apparent until we consider the children of mothers who had had four or more previous pregnancies, of whom only 13 per cent were in Classes I, II or IIIA and no less than 37 per cent in Classes IV or V (compared with the corresponding figures of 25 per cent and 21 per cent respectively for those whose mothers had had three or less previous pregnancies). It is not surprising, therefore, in view of the results shown in Figures 8.1 and 8.2, to find that the performance of children whose mothers had had four or more previous pregnancies is inferior to that of the rest of the population. Moreoever, we have found that the effect persists when the children are compared with each other within the four main social class groups, and the results for children in Class IIIM are shown in Figure 8.4. The spread is clearly not as great as that produced by differences in the health visitor's grading of 'Care of Child' (Figure 8.3), and in most variables the greater part of the spread is accounted for by the impairment of performance among the children whose mothers had had four or more previous pregnancies. Pregnancy number does appear to have an effect, even after allowing for social class, and we feel justified in including it among the factors to be studied by analysis of variance in Chapter 9.

Use of Services

The immunisation state of their children has been used as an indicator of the extent to which families make use of the services freely available to them for their

children's benefit (Davie et al, 1972). We recorded at the time of the school entry medical examination the number of immunisation procedures which each of our children had undergone. The optimum number was 7 or more 'immunisations', depending upon the type of poliomyelitis protection employed.

Table 8.5 shows the relationship between the health visitor's grading of 'Care of Child' and the number of immunisations recorded by the school nurse two years later. Presumably the health visitors may have taken the child's immunisation state into account when making their grading, but it is clear that they were not narrow-minded about it, since 5 per cent of those whose families were graded A had had no immunisation. It is the striking feature of this table that there is a steep increase in the proportion of children whose immunisation state was suboptimal as we move from grade A to grade C, the increase being greatest (fivefold) in those who had not been immunised at all. There is a corresponding, approximately threefold, fall in the proportion whose immunisation state was optimal. In other words, it is a characteristic of the families whose care of their children overall is regarded by their health visitor as average or poor that they make progressively less use of the facilities and services which are freely available to them.

'Care' and Number of Children

We have looked for a relationship between our families' grading in respect of 'Care of Child' and the number of previous liveborn children which the mother had had. This appears to us to be potentially important from two points of view. It is possible that a mother's ability to care for her children may become less adequate as their actual number increases, so increasing the demands made upon her; and such an effect, if it exists, will tend to increase the actual proportion of children in the population who are cared for inadequately.

Since both the health visitor's grading and the number of children in families are distributed differently in different social class groups, we have studied this inter-relationship within these groups, as shown in Table 8.6. It is clear that these results confirm the possibility mentioned above, since there is an increase in the proportion of mothers who had had four or more previous liveborn children as we pass from families whose 'Care of Child' was graded A to those graded B or C, within each social class group. The full range of this effect is not apparent in groups I+II and IIIA, because too few children were graded C for percentages to be calculated, but even in these socially favoured groups there is a threefold increase in the proportion of large

Table 8.5

Percentage of children in each 'Care of Child' grade at the age of 3 years who had received stated number of immunisations by the age of 5 years

'CARE OF CHILD' GRADE	NUMBER OF IMMUNISATIONS 0	1-6	>=7	No.in Grade
Good	5.3	18.2	76.5	2,268
Average	15.5	30.9	53.6	2,849
Poor	31.2	44.7	24.1	266

Table 8.6

Percentage of children in each 'Care of Child' grade within each social class group at the age of 3 years whose mothers had had stated number of previous liveborn children

SOCIAL CLASS	GRADE	NUMBER OF PREVIOUS LIVEBORN CHILDREN 0	1-3	>=4	No.in Grade
I+II	Good	43.6	53.1	3.3	668
	Average	30.5	60.1	9.4	203
	Poor	(4)	(2)	(2)	8
IIIA	Good	39.4	57.6	3.0	505
	Average	29.7	61.1	9.2	316
	Poor	(0)	(3)	(1)	4
IIIM	Good	38.1	57.6	4.3	1,504
	Average	27.9	60.4	11.7	1,686
	Poor	20.3	56.8	22.9	118
IV+V	Good	31.5	56.7	11.8	400
	Average	25.9	59.1	15.0	869
	Poor	19.3	54.5	26.1	88

The figures in parentheses refer to the absolute number of children in cells where they are too few for percentages to be calculated.

families between grades A and B. The nature of this relationship between standards of maternal care and size of family means that the proportion of children in our community who experienced less than optimal care is greater than a glance at Table 8.6 would suggest. This table refers only to our Survey children. By definition, the number of siblings of those in the first column, with the relatively favourable grading of care, was 0, while that in the third column, with the relatively unfavourable grading, was at least 4. These siblings were presumably receiving the same standard of care as the Survey child, at least when the health visitor decided her grading.

DISCUSSION

Our findings are fully consistent with previous reports of the relationship between the performance of school-age children and their social class of origin as determined by their father's occupation. However measured, the average quality of this performance in children from 'lower' social classes is inferior to that of children from 'higher' social classes, if we accept the value judgments of class and of performance that are current in our society (Douglas, 1964; Illsley, 1967; Davie et al, 1972). The types of performance which we have reported in this chapter are general IQ and growth (as reflected by attained height) at 5 and 10 years of age and behaviour (as reported by the child's teacher) at 10 years. We feel that we can contribute something new to the findings concerning these effects because of the ages at which our measurements were made. The fact that social class gradients in performance were well developed by the age of 5 years, during the children's first term in school, means that the observed effect must have been produced almost wholly by the pre-school environment; and comparisons between the results of comparable measurements in the same children at the ages of 5 and 10 years afford an opportunity of differentiating between the effects of environmental factors (which are likely to increase with the passage of time) and those of innate or perinatally determined factors (which are likely to diminish under the modifying influence of environmental differences).

An example of this former process would appear to be the striking increase in the spread of IQ scores in boys between the ages of 5 and 10 years, largely accounted for by the marked gain in the performance of boys from Social Classes I and II (see Figure 8.2). At both ages, the test used was a 'non-verbal' one, intended to discount the modifying effects of differences in educational experience; but it is tempting to ascribe the obser-

ved effect to just such differences, because the most obvious systematic difference between the environments of boys in Social Classes I and II and Class IIIA between the ages of 5 and 10 is that private schooling is virtually confined to the former. A similar increase in the spread of the results of non-verbal test scores, between the ages of 8 and 11 years, was reported by Douglas, Ross and Simpson (1968). The effect in our population was certainly not due to any difference in nutritional state or other factor associated with differences in social class which would also affect growth, since the spread of heights is no greater at 10 than at 5 years of age.

Our finding that as the number of previous pregnancies in the mother's history increases the child's performance deteriorates, even when the analysis is confined to a single social class (see Figure 8.4), agrees with the reports by Illsley (1967) and Douglas (1964) and by Davie et al (1972) concerning family size. The fact that the effect in our population is, if anything, greater at the age of 10 years than at 5 suggests that it is environmentally determined, possibly through the larger number of older children competing for the parents' attention and so diluting its beneficial effect upon the individual child.

Our attempt to evaluate the effects of a health visitor's grading of the quality of a family's care of its children when the index child was 3 years old upon that child's performance at the ages of 5 and 10 years is without precedent, as far as we know. The magnitude of the effect which we have found, even within a single social class, has surprised us (see Figure 8.3). It cannot have been due to any bias on the part of the health visitors who did the original grading, since they had no idea to what use their observations would later be put. Systematic application of such observations would have great clinical importance, if we could find an acceptable way of modifying the environment of children who are suffering from poor parental care in such a way as to protect them from the long-term consequences of this handicap.

Apart from the obvious social class gradient, our description of the associated characteristics of families whose care of their children was graded less than optimal includes an increasing number of previous live-born children and a decreasing use of the services freely available for the benefit of the children, most marked in the families whose grading was 'poor'. There are obvious implications in these findings for ways of attempting to interrupt the 'cycle of deprivation' by which inadequate care of the children of one generation appears to repeat itself in the next.

One explanation which can be assumed to underlie the finding of uniform gradients of performance of the type reported here is that children in the 'lower' social classes perform relatively poorly partly because they have been deprived of the environmental encouragement and opportunity to develop their innate potential. Support for this assumption is provided by our finding of a social gradient in the opposite direction when assessing the performance of pre-school children by their rate of acquiring a natural skill. We reported then that children in our population learned to walk unsupported significantly earlier if they were born into Social Classes III, IV or V than Classes I and II (Neligan and Prudham, 1969). The likely explanation would appear to be that those who walked earlier had more opportunity to learn this particular skill because they were less protected.

However, regardless of the direction of the social gradients, causation is clearly complex, and obviously related to the associations between social and biological factors which are emphasised in Chapter 7. Some form of multivariate analysis is clearly necessary, if we are to attempt to assess the relative contribution of different factors, and the results of our work along these lines are reported in the next chapter.

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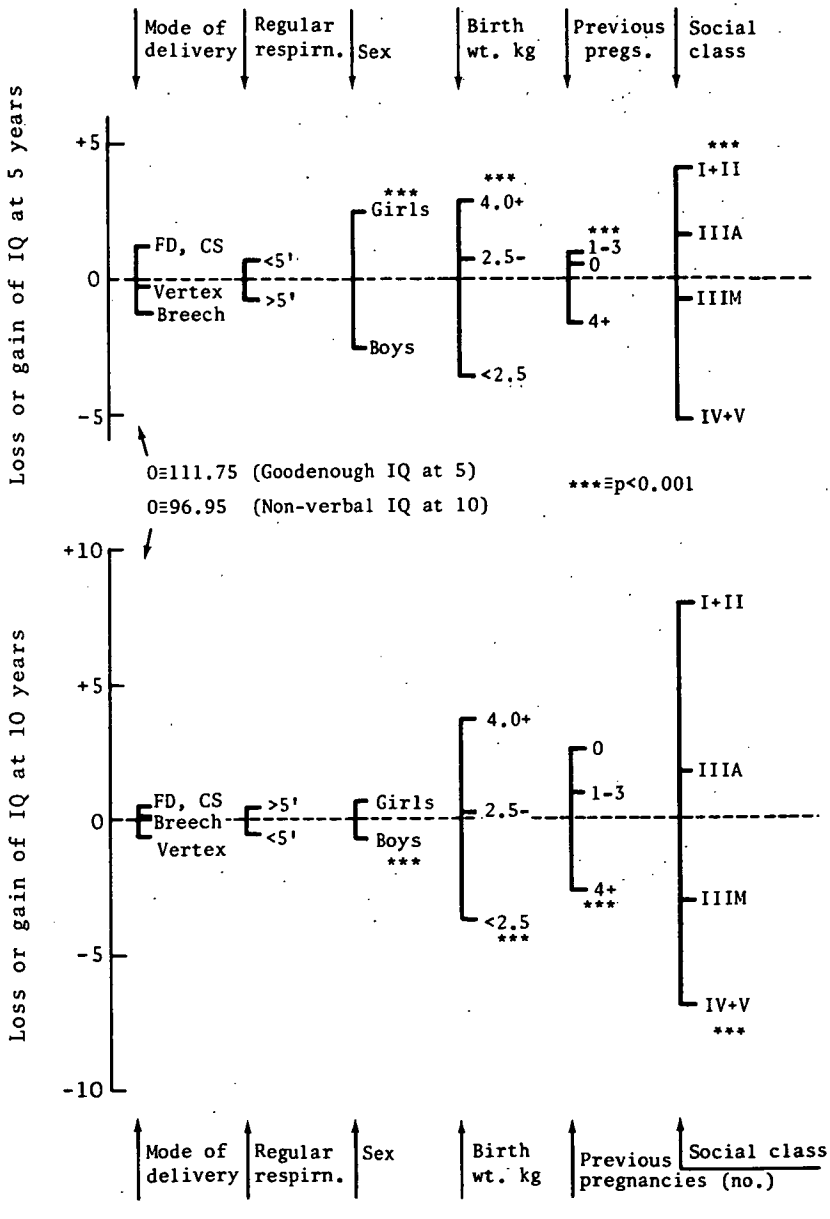
9

MULTIVARIATE ANALYSIS

In studying our epidemiological data prospectively, we have, so far, used only simple procedures, in most cases reporting the effect of one single variable (an antecedent factor) upon another (a measure of performance). We have used standard methods for testing the statistical 'significance' of these effects, but we have also drawn attention to the fact that certain antecedent factors tend to be systematically associated with each other, and have reported the effects of some such associations (e.g. of breech delivery and delay in establishing regular respiration in Figure 3.1, and of birth weight and social class in Figures 7.4 and 7.5). Such methods have enabled us to identify the maximum harmful effect which could be attributed to the individual adverse factors which we have studied, and also to identify certain favourable effects of other factors, such as that of high birth weight (in the absence of breech delivery) upon general intelligence measured at the ages of 5 and 10 years. However, the chain of association, even between the few variables whose effects we have reported in previous chapters, is far too complex to be adequately assessed by such simple types of analysis. If we want to identify the direct effects of any individual factor, so that we can judge the maximum benefit which could be expected from preventive measures designed to correct that one factor, we must be able to separate these direct effects from those of associated factors. From this point of view, it does not matter whether the associations are systematic (e.g. between abnormal modes of delivery and delay in establishing regular respiration, as shown in Table 2.3) or the result of chance in our particular set of data. In other words, we need to use some form of multivariate analysis which will enable us to estimate the effect of each of the individual antecedent factors in which we are interested, after allowing for the effects of other obviously relevant factors.

Figure 9.1

Effect upon Goodenough IQ at 5 years and non-verbal IQ at 10 years of 6 factors (2 perinatal, 2 biological, 2 social) studied by analysis of variance, so that in each case the effect of all the other 5 factors is allowed for



Analysis of Variance

In deciding which of the available statistical techniques would be most suitable for this purpose, we have taken into account the characteristics of the variables which we wish to assess, namely those between which our simpler analyses suggest that there may be important associations. Of the independent variables (antecedent factors), some cannot be measured continuously (e.g. sex, social class) and some, though they can be so measured, may be studied more profitably from the clinical point of view by grouping values so as to demonstrate the effects of extreme categories (e.g. high and low birth weight). The dependent variables (measures of performance at school age) which we wish to study further at this stage are general intelligence and growth, both of which can be measured continuously, in the form of intelligence test scores and of height standardised for age. Analysis of variance appears to us to be the technique best suited to this situation, and it has been used for all the calculations whose results are reported in this chapter. The statistical principles according to which a suitable programme was written for analysing our data by the IBM 360/67 computer are described in the Appendix (before Appendix Table 9.1). Where the results of measures of performance are expressed in terms of proportions (e.g. of children placed in different diagnostic categories by their behaviour inventory scores), they required to be analysed by a different procedure, and it is our intention to report the results of such analyses elsewhere.

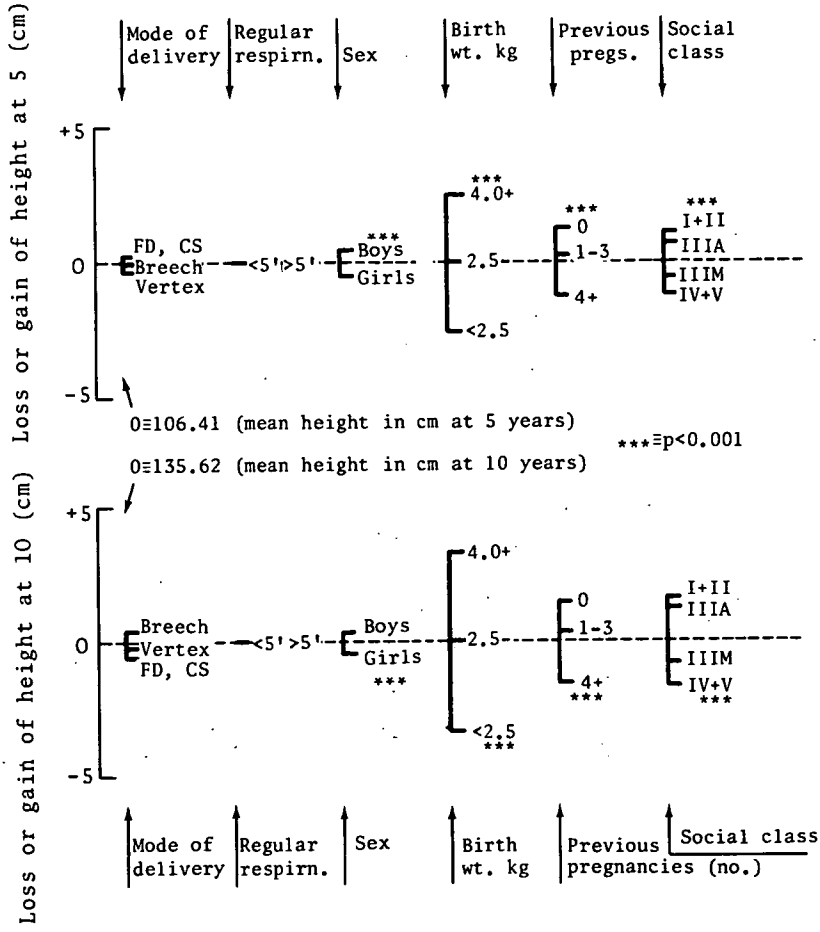
The antecedent factors whose effects we have assessed by our programme for analysis of variance (where they are treated as independent variables) include the majority of those which appear to us to be of practical interest and which our simpler analyses have shown to be probably or possibly statistically significant. They fall into two groups, which have been examined separately. The first includes six variables concerning which we have satisfactory data for virtually all the children tested at the ages of 5 and 10 years; two are 'perinatal' factors (mode of delivery and delay in establishing regular respiration), two are 'biological' (sex and birth weight) and two are 'social' (social class and number of previous pregnancies). The second group includes only three variables and is designed, after allowing for the effects of sex, to contrast the effects of birth weight with those of gestational age (concerning which we have reliable data for only about three quarters of our population).

Perinatal, Biological and Social Factors

In Figure 9.1 we have illustrated the results of examining the effects of the individual factors in the first

Figure 9.2

Effect upon height in centimetres at 5 and 10 years of the same 6 factors as in Figure 9.1, studied by analysis of variance, so that in each case the effect of all the other 5 factors is allowed for



group upon the dependent variable of IQ as we measured it at the age of 5 years (in 7,692 children) and again at the age of 10 years (in 6,496 children). We have used conventional methods of showing which factors are statistically significant (*≡p<0.05, **≡p<0.01, ***≡p<0.001). It is clear that, when the effects of the other five factors are allowed for in each case, neither of the perinatal factors has any significant effect upon IQ at either age. This point is emphasised by the fact that the relative effects of certain categories of both factors are actually reversed at 10 years as compared with 5 years of age. In view of the fact that, by simple analyses of the type reported in Chapter 3, the adverse effect of breech delivery is confined to boys, we have repeated the analysis illustrated in Figure 9.1, but in boys alone, and there is still no significant effect of breech delivery upon IQ.

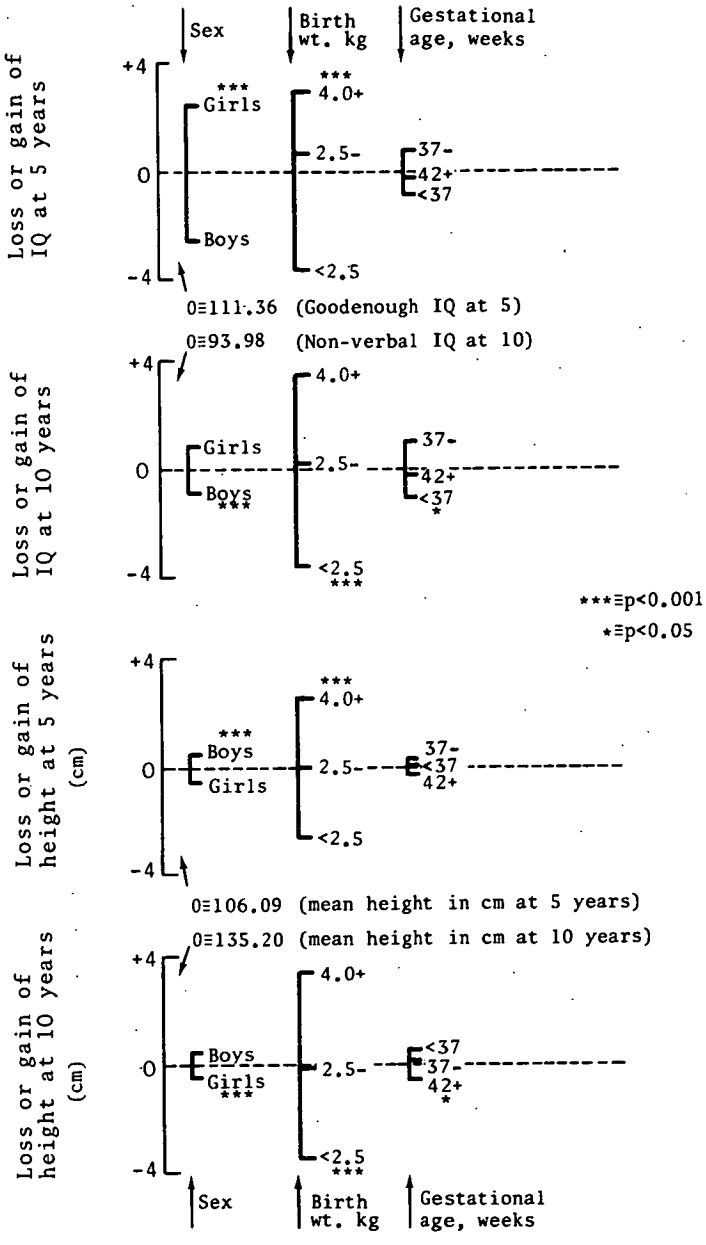
The greatest effects are again produced by differences in birth weight and social class, confirming the impressions gained earlier by comparing Figures 7.6 and 8.2. Social class has a greater effect upon IQ at 5 years than does birth weight, even when the five other factors have been allowed for in each case, and by the age of 10 years the social class effect has markedly increased, while that of birth weight is unchanged.

When height is the dependent variable, however, the results illustrated in Figure 9.2 show that birth weight has rather more effect than does social class, and both effects look to be about as great at 10 as at 5 years of age. However, since the units on the scales are centimetres, and the absolute measurements are more than 25 per cent higher at 10 than at 5 years, the effect is proportionately less at the later age. Not surprisingly, neither of the perinatal factors has any effect upon height, and the effect of sex is comparatively small, as it is upon IQ.

The remaining factor, which clearly has a significant effect upon both IQ and height at both ages, is the number of previous pregnancies the mother has had. The effect upon IQ is distinctly greater in absolute terms at the later age, and since the mean score is about 15 per cent less than at 5 years (97 as compared with 112), the effect is proportionately even greater than it looks. This finding is suggestive of an environmental effect. It could possibly be due to the number of older siblings with which our child has had to compete; but another possibility is the relationship between this factor and the 'Care of Child' grade, illustrated in Table 8.6, where there is an obviously progressive increase in the health visitor's grading of 'poor' with an increase in the number of previous liveborn children. Clearly, the effects of these and other associations need to be

Figure 9.3

Effect of sex, birth weight and gestational age upon IQ at 5 and 10 years, and height at 5 and 10 years, by analysis of variance



explored further, and we plan to do so in due course.

Birth Weight and Gestational Age

The second group of factors whose effects we have studied by analysis of variance consists of only three variables, namely sex, birth weight and gestational age. Our aim in carrying out this analysis is to compare the effect of the two most commonly quoted variables related to intrauterine growth, after allowing for the effect of sex.

The comparative effects upon both IQ and height at 5 and 10 years of age are illustrated in Figure 9.3. It is clear that both sex and birth weight have highly significant effects upon both dependent variables at both ages. The direction of the effect of sex is, of course, reversed when we move from IQ to height, and we do not know the reason for either effect - in the present state of knowledge, we simply have to accept, and allow for, both. The progressive advantage conferred by increasing birth weight upon both IQ and height at both 5 and 10 years, to much the same extent as is shown in Figures 9.1 and 9.2, strongly suggests that the recognised association between postnatal physical growth and general intelligence is at least partly explained by a physical process which is initiated during the intrauterine phase of growth, and reflected in birth weight acting as a crude indicator of the rate of intrauterine growth. The extent to which low birth weight (<2.5 kg) implies either shortening or slowing of the intrauterine phase of growth can be roughly assessed by a glance at Figure 7.1, but the results illustrated in Figure 9.3 strongly suggest that the effective component is the slowing, since the effect of duration of gestation has been allowed for in this type of analysis. Also, the effect of variation in gestational age at birth is barely significant, after the effects of sex and birth weight have been allowed for. In any case, and regardless of the duration of gestation, the beneficial effect of high birth weight (>4.0 kg in our analysis) must be a reflection of good intrauterine growth.

DISCUSSION

Analysis of variance shows that, if we first allow for the effect of a number of relevant associated variables, neither of the adverse perinatal factors which we studied earlier by simpler methods has any significant effect upon general intelligence as we have measured it in our normal school population. Even the adverse effect of breech delivery in boys reported in Chapter 3 is no longer significant when other associated factors are allowed for.

The highly significant effects of sex, birth weight,

number of previous pregnancies and social class, illustrated in Figures 9.1 and 9.2, confirm the results of the simpler analyses which we have discussed in previous chapters. Our findings are broadly similar to those reported by Davie, Butler and Goldstein (1972) using a similar method of analysis. The four effects shown are independent of each other, and so can be cumulated, with the result that in the Goodenough IQ test at 5 years we might expect a girl with a birth weight of more than 4.0 kg whose mother has had no previous pregnancies and whose father's occupation places her in Social Class I or II to score 5+6+2+9=22 points more than a boy with a birth weight of less than 2.5 kg whose mother has had four or more previous pregnancies and who is in Social Class IV or V. In the non-verbal IQ test at 10 years, the corresponding expected difference is 1+7+4+15=27 points. In the case of height, the (relatively slight) sex advantage would favour a boy, but, when associated with the same other favourable factors as in the case of the girl above, he would be expected to be 10 cm taller at 5 years and 13 cm taller at 10 years than a girl with the corresponding unfavourable factors.

Multivariate analysis has enabled us to strengthen the impressions conveyed by the simpler analyses reported in earlier chapters that the effects of biological and social factors upon the intelligence and growth of children in normal schools at the ages of 5 and 10 years are far stronger than those of the two perinatal factors which we have chosen to study. In fact, the latter produce no significant effect at all when due allowance is made for the effects of other adverse factors with which they are systematically associated. Clearly, the nature of the biological and social factors requires to be studied in greater depth to learn more about their practical implications.

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10

THE VIEW FROM THE NURSERY

On the broad canvas of our Community Study, perinatal adverse factors appear to contribute so little to the sombre shadows of handicapping conditions and impaired performance that it would be easy to dismiss them from further consideration; but those who are able to view the scene from the vantage point of the Special Care Nursery of a maternity hospital see a different picture. We see, as though in a vivid painting in miniature, a small number of babies whose misadventures during their passage through the 'valley of the shadow of birth' (Smith, 1951) confront them with the stark alternatives of sudden death on the one hand, and survival, but with an obvious risk of severe and irreversible brain damage, on the other. The outcome may be beyond our control, or it may depend directly upon the results of urgent clinical decisions. If we are to make the right decisions, we need the right information on which to base them, and although the disasters are so relatively infrequent that they do not materially affect the overall picture of our population of less than 10,000 school entrants born in 1960-2, we think that several other considerations need to be borne in mind. Firstly, the position we have described has been reached, at least partly, as a result of improvements in obstetric and perinatal clinical practice during the previous decades (see Discussion of Chapter 5). Continuous clinical vigilance is required if we are to maintain this position, let alone improve upon it. Secondly, although the disasters are relatively infrequent, their clinical manifestations can be very severe, since they involve any possible combination and degree of mental defect, cerebral palsy and convulsions. Finally, there is obvious room for improvement in perinatal mortality rates, and it is important to know whether, and if so how, such improvements can be achieved without paying an unacceptable price in terms of handicapped survivors.

The nature of the dilemma was high-lighted by Drillien (1967). She reported that, between two non-consecutive

five-years periods, the survival rate of Edinburgh babies with a birth weight of 1,360 g or less had improved from 17 per cent to 30 per cent as a result of 'advances in antenatal care of mothers, in management of late pregnancy and delivery, and in the techniques of premature baby care', but virtually all the extra survivors had later been found to be moderately or severely handicapped. In other words, the increase in the survival rate had been bought at the cost of an increase in the proportion of moderately or severely handicapped children among the survivors from 32 per cent to 56 per cent. Such results seriously suggest that some technical 'advances' may do more harm than good. Similar risks are obviously involved in undertaking to treat certain other perinatal problems, such as severe clinical asphyxia, especially if complicated by cardiac arrest. It is the main purpose of our Hospital Study to assess the magnitude of this risk in the case of those severe adverse factors which are sufficiently common to call for an agreed and soundly based policy of paediatric management. Our secondary purpose is to learn more about the relationships between this risk and the details of clinical management, so that we can take practical steps to reduce the proportion of handicapped survivors of these severe adverse factors.

The General Picture

Among the 20,793 babies who were born in our hospital between 1 January 1961 and 31 May 1970, we identified 218 (1.05 per cent) who survived and went home after suffering from one or more of the six adverse factors in which we are interested (and which are listed in Table 10.1). We were able to obtain sufficient information for our present purposes concerning 211 (96.8 per cent), of whom 181 were examined personally, and information from colleagues or parents was available for the remaining 30 children. Some of the latter were not accessible to us as a direct consequence of their handicap (e.g. if they were away in a residential institution) and they included a significantly higher proportion of handicapped children than did those who were accessible. This finding emphasises the importance of achieving as complete coverage as possible in follow-up studies of this kind (and of interpreting the results with great caution if coverage is very incomplete). It also explains why our final results, as described in this chapter, are less favourable than the preliminary results published in the Report of the Expert Group on Special Care for Babies (1971).

The handicaps whose incidence we are reporting here are only those of moderate or severe degree, by which we mean that there was a clear-cut neurological or intel-

lectual deficit, usually precluding admission to a normal school at the age of 5 years. There were 33 children who suffered from one or more such handicaps; they have each been assigned to only one category, that of their most severe handicap, as follows:-

cerebral palsy-23 children (11 quadriplegic
3 diplegic
8 hemiplegic
1 dyskinetic)

severe deafness-6 children

severe mental subnormality (IQ?<50)-4 children

Only 14 of these handicapped children were of school age when assessed; 10 were cases of cerebral palsy, of whom 5 were in normal schools - the remainder were in special schools or institutions.

It is obvious from a glance at Table 10.1 that there is a great excess of handicaps in the survivors of each of the six adverse factors and in the 211 index children as compared with the 200 controls. We need to look into the effects of each adverse factor separately, if we are to learn anything of clinical value, but before doing so, we wish to make two general points.

Firstly, since 288 adverse factors occurred in 211 children, who constituted only 1 per cent of our population, the factors clearly have a tendency to cluster, so that where one occurs, the likelihood of another is increased. In fact, there may be an obvious cause-and-effect relationship between certain pairs of factors, even though we may not understand the actual mechanisms involved. Where this is so, it is helpful, when looking for the possibility of prevention, to classify the causal factor as 'primary' and the consequence as 'secondary'. For instance, apnoeic/cyanotic attacks are much commoner in babies of low birth weight, and both cardiac arrest and severe clinical asphyxia may be followed by severe cerebral irritation or convulsions. We therefore classify very low birth weight, cardiac arrest and clinical asphyxia as 'primary' factors and apnoeic/cyanotic attacks, severe cerebral irritation and convulsions as 'secondary' factors (which pose less stressful clinical questions and require less detailed consideration here).

Secondly, in view of the possibility that the harmful effects of certain factors may be cumulative, we think it right to evaluate the survivors of each primary factor in two separate groups, the first uncomplicated, the second complicated by one or more of our other severe adverse factors.

Very Low Birth Weight (<=1,360 g)

There is no real difference between the incidence of handicaps in the uncomplicated group and that of the complicated group of survivors of very low birth weight,

Table 10.1

Proportion of handicapped children among the survivors of 6 severe adverse factors, 288 cases of which occurred among 211 index children, compared with 200 controls: mean age at time of last information is 4.0 years

Clinical Category	Number of survivors	Percent handicapped
Very low birth weight (<=1,360 g)	63	7.9
Cardiac arrest	22	22.7
Severe clinical asphyxia (>20 minutes)	30	30.0
Apnoeic/cyanotic attacks	83	19.3
Convulsions	74	20.3
Severe cerebral irritation*	16	25.0
Total cases of severe adverse factors	288	-
Index children	211	15.6
Control children	200	0.5

*requiring treatment by sedation for 7 days or longer

Table 10.2

Proportion of handicapped children among survivors of very low birth weight (<=1,360 g) related to occurrence of complications

	No. of Survivors	Handicapped		
		No.	Percent	Clinical
Uncomplicated	39	3	7.7	2 hemiplegic, 1 deaf
Complicated*	24	2	8.3	2 diplegic

*21 complicated by apnoeic/cyanotic attacks, 2 by severe asphyxia, 1 by cardiac arrest

Table 10.3

Proportion of handicapped children among survivors of cardiac arrest related to clinical category

Clinical Category	No. of Survivors	Handicapped	
		No.	Clinical
Fresh stillbirth	6	2	both quadriplegic, defective
Arrest <15 minutes after birth	8	2	both quadriplegic, defective
Arrest at 72 hours, ?cause	1	0	
Arrest during exchange transfusion	7	1	deaf (?due to hyperbilirubinaemia)
Total	22	5	

to our surprise (Table 10.2). The number of cases is, of course, very small, but it is interesting to note that the two cases of diplegia occurred in cases complicated by apnoeic/cyanotic attacks, as described by McDonald (1963). Her overall incidence of cerebral palsy, of 6.5 per cent in children with a birth weight of 1,800 g or less born in 1951-53 is almost identical to ours of 6.5 per cent in those with a birth weight of 1,360 g or less. Rawlings et al (1971) reported 7.4 per cent definitely abnormal among children who weighed 1,500 g or less when they were born in 1966-69; but the great importance of this report is that the survival rate in the weight-group 1,001-1,500 g was 73 per cent. In other words, the fear expressed by Drillien (1967) that increased survival of up to half of all the babies of very low birth weight '...might lead to the survival of an increasing number of defective children' was shown to be groundless. Among the possible explanations for the improved quality of the survivors in recent years is the increasing interest in attempting to achieve adequate nutritional intake as soon as possible after birth, either by stomach (Smallpeice and Davies, 1964) or by vein (Shaw, 1973). Perhaps a combination of the two routes, determined by clinical judgment in the individual baby, may be preferable to relying on either alone as a matter of principle. It may be relevant that all our 4 cases of cerebral palsy occurred among the 30 very low birth weight babies born before 1965, and none among the 33 born during 1965-70, when intravenous feeding was being increasingly frequently used to anticipate and to supplement gastric feeding; this difference is just statistically significant ($p<0.05$). It has been shown that the baby whose very low birth weight is due to intrauterine growth retardation may be less capable of attaining a normal skull circumference in spite of postnatal nutrition than the baby who is simply born too soon (Davies and Davis, 1970), but the neurological and intellectual implications of this difference are not yet clear.

Cardiac Arrest

Our 22 index cases of survivors of cardiac arrest are the product of 39 serious attempts to resuscitate fresh stillbirths or early neonatal deaths during the period covered by our Hospital Study. Of those in whom we were unsuccessful, 6 failed to respond at all and 11 died during the first week after an initial response. The absence of a heart-beat was determined by auscultation in all cases, usually by two observers. Routine treatment consisted of ventilation with 100 per cent oxygen by endotracheal tube, and closed chest cardiac massage. If the heart-beat did not return within 4 minutes, an intracardiac injection was given of 0.5-1.0 ml niketha-

Table 10.4

Proportion of handicapped children among survivors of cardiac arrest related to occurrence of complications

	No. of Survivors	Handicapped		
		No.	Percent	Clinical
Uncomplicated	9	1	11.1	deaf
Complicated*	13	4	30.8	all quadriplegic, defective

*Commonest complications were severe clinical asphyxia and convulsions (each in 7 cases); also apnoeic/cyanotic attacks (2 cases) and very low birth weight (1 case)

Table 10.5

Proportion of handicapped children among survivors of severe clinical asphyxia (>20 minutes) related to occurrence of complications

	No. of Survivors	Handicapped		
		No.	Percent	Clinical
Uncomplicated	10	2	20.0	1 hemiplegic, 1 defective
Complicated*	20	7	35.0	4 quadriplegic, defective
				1 hemiplegic, 1 defective
				1 deaf

*7 cardiac arrest, 12 convulsions, 4 apnoeic/cyanotic attacks, 3 severe cerebral irritation, 3 very low birth weight

mide in the earlier years, later replaced by a similar volume of 8.4 per cent sodium bicarbonate. If necessary, massage was continued up to 15 minutes after the arrest was thought to have occurred (and one of the neurologically and developmentally normal survivors had no audible heart-beat for this length of time). During the later years, an umbilical venous catheter was inserted as soon as practicable and a bolus of 8.4 per cent sodium bicarbonate was injected (5 ml for small babies, 10 ml for large ones or following prolonged arrest) followed by further doses if indicated by the results of blood gas studies. The same catheter was used for an infusion of 10 per cent dextrose solution 120 ml/kg during the next 24 hours. If renal function appeared satisfactory and the baby was not yet tolerating adequate feeds, an infusion was continued by scalp vein until this was achieved.

We would expect the prognosis to vary with the cause of cardiac arrest, and there is some evidence of this in the clinical categories of Table 10.3, since none of the 7 cases where the arrest occurred during exchange transfusion showed evidence of anoxic brain damage (the one case of deafness being probably due to hyperbilirubinaemia). Also, within the group of fresh stillbirths, 4 were caused by shoulder dystocia (difficulty in delivering the shoulders) in babies with a mean birth weight of 4.5 kg, and all 4 later developed quite normally. The remaining 2 fresh stillbirths, attributable to cord prolapse and difficult breech delivery respectively, developed the handicaps recorded in the Table.

The most worrying feature of the sequelae of this particular adverse factor is the severity of the brain damage in the children who did not survive unscathed; they were all 4 grossly mentally defective, as well as being quadriplegic. The results summarised in Table 10.4 indicate that the risk of this type of brain damage is confined to the cases which were complicated by one or more of our severe adverse factors. These were often multiple (for instance, one case was complicated by all our three 'secondary' factors) and the relationships are too complex for statistical analysis to be helpful in this small number of cases.

However, straightforward inspection of the raw data has enabled us to make one clear distinction between the 4 survivors who later developed evidence of severe, diffuse brain damage and the 18 who did not, namely that all of the former and none of the latter took more than 30 minutes to establish active regular respirations after the heart-beat had returned. This finding seems to us to justify a policy of desisting from further attempts at resuscitation after this interval - or sooner in cases like the two instances of stillbirth with a poor prognosis, mentioned above. Such a policy should materially

reduce the proportion of severely handicapped among the survivors of perinatal cardiac arrest.

We know of no comparable series of cases of survivors of cardiac arrest with which we can compare our findings.

Severe Clinical Asphyxia

We adopted our definition, a delay of 20 minutes or longer before establishing active, regular respiration, because we already had information from our Community Study suggesting that shorter periods of delay were unlikely to cause significant sequelae (see Chapter 4). In fact, we reported that remarkably little brain damage could be attributed to asphyxia of any degree, and there were only two mild cases of cerebral palsy (attending normal schools) among the 19 children who had taken more than 20 minutes to establish regular respiration.

The data summarised in Table 10.1, and in a different form in Table 10.4, reveal at a glance that the results in our Hospital Study are very different. One obvious reason for the higher incidence of severe handicap is that 7 of our Hospital Study cases were complicated by cardiac arrest (and there were no such survivors in our Community Study). All the 4 most severely brain-damaged children in Table 10.5 were the result of this complication, and a policy designed to prevent such cases in the future is described in the section on cardiac arrest above. Another, more disquieting, possible reason is that intermittent positive pressure ventilation (IPPV) was available as a routine method of resuscitation throughout our Hospital Study. It has already been suggested in Chapter 4 that the effectiveness of this procedure may prevent death from respiratory failure in some children who have sustained diffuse and irreversible brain damage as a result of adverse factors affecting them during pregnancy, labour or delivery. Instances of such a possibility are not difficult to find in our Hospital Study cases. For instance, all 4 survivors of severe concealed antepartum haemorrhage (treated by IPPV for 25-40 minutes after birth) developed cerebral palsy; and 5 of the 9 handicapped children in Table 10.5 were the product of a pregnancy of more than 42 weeks' duration. On the other hand, very prolonged clinical asphyxia in a pre-term baby is compatible with undamaged survival, as illustrated by two of our cases. They weighed 879 g at 29 weeks and 1,290 g at 32 weeks and required to be ventilated for 13 hours and 8 hours respectively after birth before establishing active regular respiration. Neither developed any neurological abnormality; the first had doubtful developmental delay when last seen at 31 months, the second had an IQ of 92 at 5 years.

The results of our Hospital Study, therefore, tend to

support the view that prolonged clinical asphyxia following delivery, particularly if it fails to respond quickly to the effective stimulus to Head's reflex provided by IPPV (Cross et al, 1960), is liable to be followed by evidence of brain damage amongst the survivors; but when adequate ventilation has been achieved within minutes of birth, this brain damage is likely to be the result of the same adverse factor as caused the asphyxia rather than a result of the asphyxia itself. Our discussion of the relevant literature in Chapter 4 suggests that it is difficult to evaluate the results of earlier reports because of the unsatisfactory nature of much of the perinatal information, and of more recent ones because of the high proportion of children lost to follow-up. We know of no comparable investigation of the sequelae of the use of IPPV for resuscitation.

'Secondary' Adverse Factors

Of the 173 cases of apnoeic/cyanotic attacks, convulsions or severe cerebral irritation (Table 10.1), 58 were clearly a complication of one of our three 'primary' factors. Others were equally clearly caused by other factors; for instance, apnoeic/cyanotic attacks often occurred in babies whose birth weight was low, though it did not fall below our arbitrary upper limit of 1,360 g; convulsions were in some cases a complication of hypoglycaemia or hypocalcaemia; severe cerebral irritation was in some cases a symptom of birth trauma or of withdrawal of a drug (narcotic or barbiturate) taken by the mother. The problems of investigation and treatment, whether specific or symptomatic, are relatively straightforward, and will not be discussed further here.

DISCUSSION

Individual reactions to the results reported in this chapter will no doubt vary in accordance with individual temperament. Some may be pleasantly surprised to find that the great majority of the survivors of such very severe adverse factors have turned out to be free of any moderate or severe handicap. In fact, of the 83 survivors who had reached school age 75 (90.4 per cent) were in normal schools. Moreover, the great majority of the 75, when assessed in terms of school performance or IQ, or both, were performing normally. Only among the survivors of very low birth weight is there an obvious, and statistically significant, depression of the distribution of school performance or IQ (confirming the results of our Community Study). Other readers may be so disturbed to find that such an effective and satisfying procedure as resuscitation by IPPV appears to have produced an increase in the proportion of handicapped children among

the survivors of severe clinical asphyxia that they will discount the achievement of an increase in the absolute number of intact survivors (see Chapter 4). The one point over which we think there must be general agreement is that an increase in the absolute number of survivors does not necessarily imply a corresponding increase in the number of handicapped children, as was once feared. In fact, the published results for babies of very low birth weight suggest that an increase in survival can be achieved while at the same time increasing the proportion of intact survivors.

Our own temperamental reactions, which have been incorporated into clinical policies, can be briefly summarised as follows. Overall, it seems justifiable to persist with attempts to achieve increased survival of the babies who are affected by these severe adverse factors (and others of a similar kind), but, in certain circumstances to which we have drawn attention, particularly in the sections on cardiac arrest and severe clinical asphyxia, the risk of brain damage is unacceptable, and it is right to desist from attempts to interfere with the course of nature. In all clinical categories, but particularly in the babies of very low birth weight, it is important to look at all aspects of the problems which threaten not only their survival but also their optimal development, to pay at least as much attention to their nutrition, for instance, as to their blood gases.

The overall effect of complications is also of obvious practical importance, even if we restrict this term to mean complication by one of the other severe adverse factors which we have selected for study, as we have done in this chapter. Among the 115 survivors of the three 'primary' factors of very low birth weight, cardiac arrest and severe clinical asphyxia, the 58 'uncomplicated' cases gave rise to 6 (10.3 per cent) moderately or severely handicapped children, while the 57 'complicated' cases gave rise to 13 (22.8 per cent). Early clinical intervention to prevent complications such as apnoeic attacks, convulsions and severe cerebral irritation should help to improve the quality of the survivors.

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11

THE COMPOSITE PICTURE

We now want to try to compose a single picture by bringing together the findings of our two Surveys. We have painted our Community Study's broad canvas with an epidemiological brush, using as our model a whole geographically defined three-year cohort of births followed through the first decade of the lives of those who survived the first month of life and remained accessible to us; we have tried to relate various antecedent factors to the quality of their performance over the whole range of categories, favourable as well as unfavourable, and we have, all along, viewed the scene through a clinician's eye. In the Hospital Study, we have used clinical definitions to focus that eye very narrowly onto the 1 per cent of the survivors who have been exposed to the most extreme adverse factors and are at such risk of extensive brain damage that the wisdom of attempting to interfere with the course of nature has to be seriously questioned. At the beginning of our Introduction we stated that we accept it as a fact that the adverse factors described by Little 130 years ago can cause permanent brain damage, and as a possibility that there is a 'continuum of reproductive casualty', as suggested by Lilienfeld and Pasamanick 20 years ago, so that potentially lethal factors can lead to clinically recognisable brain damage among the survivors and to lesser degrees of handicap on a much wider scale. Our purpose has been to establish some of the numerical facts concerning these relationships between antecedent factors and the quality of our community's children, since such facts are the necessary basis for rational clinical and social policies. Some of the facts which we have succeeded in establishing only pose new questions, but we hope they make it possible to frame these questions more precisely; and we believe that it should be possible to answer some of them in due course, by further analysis of the data which we already have available.

The main finding of our Community Study is that even

two of the most lethal perinatal adverse factors which can be directly modified by clinical actions (breech delivery and prolonged delay in establishing regular respiration) produce only trivial effects in terms of clinically recognisable signs of brain damage. Any effects which they may have upon the intelligence or the behaviour of the clinically normal survivors are explained by their association with two groups of much more potent factors.

Biological Factors

The first of these groups comprises what we have called 'biological' factors, of which sex and birth weight are the examples which we have studied. The well-known advantage of boys in terms of physical growth is established during the last four to six weeks of a normal period of growth in the uterus. However, girls score better in non-verbal intelligence tests at the ages of 5 and 10 years (in spite of the declared claim of the devisers of some of the tests to have eliminated this effect); they also show a lower incidence of abnormal behaviour as recorded by their teachers. These advantages are in line with the girls' markedly lower rates of neonatal mortality and clinical asphyxia, and the fact that only in boys does breech delivery produce a significantly adverse effect - which is no longer significant when the associated adverse factors are allowed for by analysis of variance.

Even greater effects are associated with variations in birth weight, which imply variations in the duration or the rate of intrauterine growth. Low birth weight implies that the duration has been too short, or the nett rate too slow, and we have not tried to distinguish between these two different factors' potentially different long-term effects in our present analyses; but we have shown that the adverse effects of low birth weight, after allowing for those of short gestation, are much greater than those of short gestation, after allowing for those of low birth weight. Moreover, low birth weight is the one adverse factor which really produces effects consistent with the theory of a continuum of reproductive casualty. It is associated with a high neonatal mortality; it is the adverse factor most commonly associated with the clinically recognisable forms of brain damage, if we exclude the association of Down's syndrome with severe mental subnormality; and it impairs the quality of the clinically normal survivors, when studied by epidemiological methods, in terms of growth and non-verbal intelligence.

There can be no doubt about the implications of high birth weight, however - it implies a rapid nett rate of intrauterine growth over an adequate period of time.

Its long-term effects upon the quality of the survivors in our population are entirely beneficial in terms of growth, non-verbal intelligence and behaviour (except in the case of boys delivered by the breech). Maternal diabetes mellitus plays no significant part in this section of the picture, since it was present in less than 1 per cent of the babies with a birth weight above 4.0 kg. The obstetrician can already influence the duration and the rate of intrauterine growth favourably in a number of practical ways, which in the past were designed purely to save life. It now looks as if he can also hope to improve the quality of the survivors by achieving an optimal rate of growth, or preventing undue prolongation of a poor rate, at this critical stage of development, when both body and brain should be growing most rapidly (the second half of the normal duration of pregnancy). When the baby is born too soon during this critical period, the responsibility passes to the paediatrician to maintain as near to an optimal rate of growth as possible. He, too, already has many practical means at his disposal for achieving this objective, but, like his obstetric colleague, he is still too often faced with adverse factors which he cannot adequately control, because he cannot fully understand them.

Social Factors

The second group of factors whose effects far outweigh those of the perinatal ones which we have studied comprise various 'social' attributes. The least specific, and yet the most powerful, of these is the child's social class of origin as determined by his father's occupation. The possible implications of growing up as a member of a family in a particular social class are very varied, ranging as they do from the genetic through the nutritional to the cultural and the general environmental. We hesitate, therefore, to draw any practical conclusions from our findings, but we have tried to learn something about the relative importance of these different implications. We have noted that the effect upon intelligence, measured by a non-verbal test, is greater than the effect upon growth when expressed in the same units, and that this disparity is much greater at the age of 10 than it is at 5 years, because the spread of the IQ scores is almost doubled at the later age, largely due to a threefold increase in the advantage of children in Social Classes I and II over those in IIIA. These findings suggest to us that the genetic and the nutritional implications can largely be discounted, as compared with the cultural and general environmental implications of social class, which are of paramount importance. We have also noted that children in the 'lower' social classes are at a further disadvantage, because

they more often display abnormalities of behaviour of a type which their teachers recognise.

Accepting the value judgments upon which these assessments are based, we have attempted to learn a little more about the nature of these environmental factors, which might help to explain this overwhelming power of social class. We have, so far, only examined the effects of two such factors, and found both to be very significant. One is the quality of the mother's care of her child, which the family's health visitor assessed for us when our Survey child was 3 years old, basing her subjective judgment upon defined criteria. The other is the number of previous pregnancies (or live children) which the mother had had before the birth of our Survey child. We have also noted an association between these two factors, in that as the number of previous children increases, the standard of care falls - an association which ensures multiplication of the proportion of our community's children who suffer from poor standards of mothering, and are likely to show the effects, at least up to the age of 10 years. Such are the factors which, no doubt combined with others which we have not yet examined, must explain the poor quality of our children's performance by comparison with other parts of Britain.

Clinical Factors

Although they nowadays play a negligible part in determining the overall quality of our community's children, as demonstrated by the epidemiological brush which painted the picture of our Community Study, the severe perinatal adverse factors whose immediate effects can be directly modified by clinical actions form a major part of the work of Special Care Nurseries. They are particularly important, in our view, because such actions can be modified relatively easily by deliberate changes of policy, if the facts call for it. Also, the survivors of the six severe factors which we have studied appear to be at risk of particularly severe, clinically recognisable, brain damage, if they do not escape unscathed (with the exception of babies of very low birth weight, in whom less severe degrees of brain damage appear to be relatively common). We have, therefore, largely confined ourselves to reporting the long-term effects in terms of moderate or severe handicaps, leaving to another occasion the complex results of the assessments of development or intelligence which formed part of our Hospital Study, and avoiding the need to allow for the powerfully modifying effects of social factors.

Although the incidence of moderate or severe handicap among the survivors of each of our six severe adverse factors is clearly higher than in the general population, we feel that the small miniature painting of our Hospital

Study can make a reasonably cheerful contribution to our composite picture. In the first place, we are happy to be able to make a contribution towards disproving the suggestion that an improvement in the survival rate, for instance, of very low birth weight babies, can only be achieved at the cost of a corresponding increase in the number of handicapped children. Secondly, and more importantly, there are clear opportunities for improving upon the results reported here by modifying clinical policies in the light of our findings. For example, we can try to improve further upon our methods for ensuring optimal nutrition of pre-term babies; we can be more selective about our treatment of cardiac arrest, recognising the very poor long-term prognosis of cases of fresh stillbirth due to causes other than shoulder dystocia; we can desist from further attempts to resuscitate cases of cardiac arrest who have not established active regular respiration within 30 minutes after the return of the heart-beat; and we can investigate the possibility that unacceptable risks of permanent brain damage may also be associated with some other indications for resuscitation by IPPV.

Composition

Our two pictures combine to form a view of the roads which we must build if we want to improve the quality of our community's children. One is a wide main road, which can bring much the greatest rewards, in general terms of improved performance, and in specific terms of reduction in the incidence of the commonest handicap which we recognise in our society, the milder degrees of mental subnormality. This road has to be built by the combined efforts of very large numbers of people taking concerted action in the biological, social and political fields, designed to improve both the intrauterine and the postnatal environments in which our children develop. The route of this road is very steep, however, and the foundations of knowledge on which it needs to be built are many of them either ill-defined or difficult to act upon. The second road is narrower, and can carry comparatively little traffic, but some of it is already built, and further progress in terms of both action and understanding can be achieved by the concerted efforts of only a few people. We hope that our findings may contribute towards the planning of both these roads.

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