



<https://theses.gla.ac.uk/>

Theses Digitisation:

<https://www.gla.ac.uk/myglasgow/research/enlighten/theses/digitisation/>

This is a digitised version of the original print thesis.

Copyright and moral rights for this work are retained by the author

A copy can be downloaded for personal non-commercial research or study,
without prior permission or charge

This work cannot be reproduced or quoted extensively from without first
obtaining permission in writing from the author

The content must not be changed in any way or sold commercially in any
format or medium without the formal permission of the author

When referring to this work, full bibliographic details including the author,
title, awarding institution and date of the thesis must be given

Enlighten: Theses

<https://theses.gla.ac.uk/>
research-enlighten@glasgow.ac.uk

SOCIAL AND OBSTETRIC FACTORS

RELATING TO PERINATAL MORTALITY IN GLASGOW

GILLIAN M. McILWAINE.

ProQuest Number: 10647899

All rights reserved

INFORMATION TO ALL USERS

The quality of this reproduction is dependent upon the quality of the copy submitted.

In the unlikely event that the author did not send a complete manuscript and there are missing pages, these will be noted. Also, if material had to be removed, a note will indicate the deletion.



ProQuest 10647899

Published by ProQuest LLC (2017). Copyright of the Dissertation is held by the Author.

All rights reserved.

This work is protected against unauthorized copying under Title 17, United States Code
Microform Edition © ProQuest LLC.

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

The perinatal mortality rate (defined as stillbirths and all first week deaths per 1000 total births) in the City of Glasgow is higher than that of other large cities in Scotland and England. It is generally agreed that this rate is influenced partly by the health and physique of the mothers and partly by the standard of obstetric care they receive.

Because of concern for Glasgow's high perinatal mortality rate this study was set up to examine all perinatal deaths occurring to women resident in Glasgow during 1970. The aims of the study were three-fold -

- (1) to classify all perinatal deaths by clinical cause using the Aberdeen Classification;
- (2) to identify the women who were losing their babies;
- (3) to determine what changes, if any, might be recommended in obstetric and paediatric care to improve the perinatal outcome.

A historical review of the development of Glasgow is given in the Introduction, showing how the poor environmental conditions of Glasgow today stem from the rapid growth of the city during the Industrial Revolution. The development of the Health Services and housing policies are also described.

Perinatal deaths to be studied were identified from data held in the computer-based system of linked child health records. Completeness of ascertainment was confirmed by examining hospital records. The clinical cause of each death was then determined and coded, using the classification designed by Baird, Walker and Thomson (1954).

During 1970 there were 16,748 births to Glasgow women. Two hundred and seventy-two babies were stillborn, and 190 died in the first week of life - a total of 462 perinatal deaths. The records of 437 cases (262 stillbirths and 175 neonatal deaths) including 33 deaths occurring in multiple pregnancies, were studied. Two-thirds of the deaths belonged to groups of deaths - unexplained

prematurity, fetal defects and antepartum haemorrhage, and are less amenable to obstetrical intervention, being related to the effects of unfavourable environmental influences on the mother's reproductive function.

The deaths in each of the eight groups were studied in detail and it was felt that improved care and a greater awareness of the importance of early antenatal care would have substantially reduced the number of perinatal deaths.

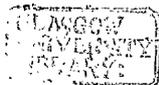
Particular attention was paid to maternal weight gain during pregnancy in those patients whose babies died. Unfortunately, however, the details of weight, weight gain and percentile baby weight were not known for all patients. The impression gained was that the association of maternal weight gain and fetal wellbeing was not thought to be important by the medical staff, as in the mature - cause unknown group particularly during the last trimester of pregnancy, the weight gain pattern was poor but no action was taken.

An attempt was made to assess the paediatric care during the neonatal period. Unfortunately the hospital records did not contain nearly enough information for retrospective analysis. It was found, however, that of the 175 neonatal deaths, 78 babies had an Apgar score of less than 5, 100 babies suffered from some form of respiratory distress, and 43 babies had an intracranial haemorrhage.

The perinatal deaths in multiple pregnancies were studied in detail, as were the domiciliary confinements. When the perinatal deaths were studied by place of delivery, it was seen that the percentage distribution of deaths varied from hospital to hospital due to a number of factors. A large number of patients booked late in pregnancy or received no antenatal care. In some hospitals there was a long delay between the time the request for booking was received and the patient being seen at the clinic. The housing conditions in which the patients lived were often very poor and there were marked differences in perinatal rates by ward of residence.

The clinical classification of death was compared with that of the registered cause of death.

Despite the poor environmental conditions of a large number of families in Glasgow it was felt that the perinatal mortality rate could be reduced immediately by stressing the importance of antenatal care, by closer supervision of the high risk patients, and by encouraging greater use of family planning services. In the long term a programme of health education and reorganisation of the obstetric services with close co-operation between all those involved with antenatal care should improve the perinatal outcome. Methods whereby this can be achieved are discussed.



SOCIAL AND OBSTETRIC FACTORS
RELATING TO PERINATAL MORTALITY IN GLASGOW

By

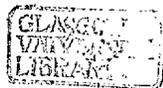
GILLIAN M. McILWAIN, M.D., D.R.C.O.G.

Thesis submitted for the Degree of
Doctor of Philosophy of the University
of Glasgow.

1974.

Social, Paediatric and Obstetric
Research Unit,
University of Glasgow.

Thesis
4181
Copy 2



CONTENTS.

	<u>Page.</u>
Acknowledgements.	
Preface.	i
Chapter I. Introduction.	1
Chapter II. The Survey.	13
Chapter III. The Classification of Perinatal Deaths.	20
Chapter IV. Maternal Characteristics.	25
Chapter V. Perinatal Deaths due to	35
(a) Mature - cause unknown	36
(b) Premature - cause unknown	45
(c) Trauma	56
(d) Toxaemia	62
(e) Antepartum Haemorrhage	69
(f) Maternal Disease	83
(g) Fetal Deformity	87
(h) Rhesus Incompatibility and Other.	97
Chapter VI. Weight Gain in Pregnancy ending in Perinatal Death.	106
Chapter VII. Neonatal Findings.	115
Chapter VIII. Multiple Pregnancies.	125.
Chapter IX. Domiciliary Deliveries	132
Chapter X. Perinatal Deaths by Place of Confinement.	143
Chapter XI. Housing Conditions.	149
Chapter XII. Geographical Distribution of Perinatal Deaths.	157
Chapter XIII. Comparison of Clinical and Pathological Classification.	164
Chapter XIV. Discussion.	173
Chapter XV. Summary.	189
Appendix.	198
References.	205
Addendum.	215

ACKNOWLEDGEMENTS.

The work described in this thesis has been carried out during the tenure of a research grant for the first year of study from the Wellcome Trust and for the following two years from the Scottish Home and Health Department. I am greatly indebted to Professor M.C. Macnaughton, who has been responsible for the overall supervision of this work, for his unfailing encouragement and advice.

I wish to acknowledge the assistance I received from the following people.

Professor I.D.G. Richards, Director of the Social Paediatric and Obstetric Research Unit during the first two years of research, for helpful advice in designing the proforma.

Mrs. Sandra Sweeney, Programmer, Social Paediatric and Obstetric Research Unit, for writing the computer programme and giving statistical help.

Sir Dugald Baird for helpful discussions.

Mr. Denys Raby, Department of Town Planning, Corporation of Glasgow, for information about housing in Glasgow.

Mr. William McNally for assistance with illustrations.

I am also extremely grateful to Miss Mary Grassick for converting an indecipherable scrawl into neat typewritten pages.

PREFACE.

The perinatal mortality rate (defined as stillbirths and all first week deaths per 1000 total births) in the City of Glasgow is higher than that of other large cities in Scotland and England. It is generally agreed that this rate is influenced partly by the health and physique of the mothers and partly by the standard of obstetric care they receive.

Because of concern for Glasgow's high perinatal mortality rate, this study was set up to examine all perinatal deaths occurring to women resident in Glasgow during 1970. The aims of the study were three-fold.

- (1) To classify all perinatal deaths by clinical cause using the Aberdeen classification.
- (2) To identify the women who were losing their babies.
- (3) To determine what changes, if any, might be recommended in obstetric and paediatric care to improve the perinatal outcome.

A historical review of the development of Glasgow is given in the Introduction, showing how the poor environmental conditions of Glasgow today stem from the rapid growth of the city during the Industrial Revolution. The development of the health services and housing policies are also described.

Perinatal deaths to be studied were identified from data held in the computer-based system of linked child health records. Completeness of ascertainment was confirmed by examining hospital records. The clinical cause of each death was then determined and coded, using the classification designed by Baird, Walker and Thomson (1954).

During 1970 there were 16,748 births to Glasgow women. Two hundred and seventy-two babies were stillborn and 190 died in the first week of life - a total of 462 perinatal deaths. The records of 437 cases (262 stillbirths and

175 neonatal deaths) including 33 deaths occurring in multiple pregnancies were studied. Two-thirds of the deaths belonged to groups of deaths - unexplained prematurity, fetal defects and antepartum haemorrhage - and are less amenable to obstetrical intervention, being related to the effects of unfavourable environmental influences on the mother's reproductive function.

The deaths in each of the eight groups were studied in detail, and it was felt that improved care and a greater awareness of the importance of early antenatal care would have substantially reduced the number of perinatal deaths.

Particular attention was paid to maternal weight gain during pregnancy in these patients whose babies died. Unfortunately, however, the details of weight, weight gain and percentile baby weight were not known for all patients. The impression gained was that the association of maternal weight gain and fetal wellbeing was not thought to be important by the medical staff, as in the mature - cause unknown group particularly during the last trimester of pregnancy, the weight gain pattern was poor but no action was taken.

An attempt was made to assess the paediatric care during the neonatal period. Unfortunately the hospital records did not contain nearly enough information for retrospective analysis. It was found, however, that of the 175 neonatal deaths, 78 babies had an Apgar score of less than 5, 100 babies suffered from some form of respiratory distress, and 43 babies had an intracranial haemorrhage.

The perinatal deaths in multiple pregnancies were studied in detail, as were the domiciliary confinements. When the perinatal deaths were studied by place of delivery, it was seen that the percentage distribution of deaths varied from hospital to hospital due to a number of factors. A large number of patients booked late in pregnancy or received no antenatal care. In some hospitals there was a long delay between the time the request for booking was received and the

patient being seen at the clinic. The housing conditions in which the patients lived were often very poor and there were marked differences in perinatal rates by ward of residence.

The clinical classification of death was compared with that of the registered cause of death.

Despite the poor environmental conditions of a large number of families in Glasgow, it was felt that the perinatal mortality rate could be reduced immediately by stressing the importance of antenatal care, by closer supervision of the high risk patients, and by encouraging greater use of family planning services. In the long term, a programme of health education and reorganisation of the obstetric services with close co-operation between all those involved with antenatal care should improve the perinatal outcome. Methods whereby this can be achieved are discussed.

CHAPTER I.

INTRODUCTION.

"No biological event has greater significance for society than reproduction and its outcome. Its importance is clearly recognised in concern over population problems, in the careful charting of national birth rates and the study of their response to boom, slump and war, and the impact of family planning on individual and national health and welfare.

"Despite such concern, one social aspect of reproduction has been curiously neglected; the influence of social conditions and behaviour on the actual course of pregnancy, labour and delivery. To many professionals and laymen, childbirth is a clinical phenomenon governed by physiological laws, guided by obstetric techniques and virtually unaffected by social processes and events. Resistance to sociological explanations of obstetric conditions is still widespread in medical and lay circles."

With these words Illsley (1967) introduced a chapter entitled "The Sociological Study of Reproduction and its Outcome," in the book "Childbearing, its Social and Psychological Aspects."

While this is so and concern for the health and welfare of children is of recent growth, the susceptibility of childhood to adverse social circumstances has long been recognised. In 1840 Cowan of Glasgow, writing about children,

said that "the contrast between the labouring classes and those in easy circumstances is in no way so strongly marked as in the relative number of births and deaths."

Much of the original work on social influences on reproductive performance has been performed by Sir Dugald Baird in Aberdeen. He has shown that the factors predisposing to reproductive efficiency, as measured by a very low perinatal mortality rate, are youth, good health and physique, a high standard of obstetric and paediatric care, and limitation of family size. Health and physique depend upon the quality of the environment from birth to maturity (Baird, 1973).

The City of Glasgow is a large industrial centre which grew rapidly during the industrial revolution, bringing prosperity to many but also producing appalling slums, some of which still exist today. (Table I).

TABLE I.

The Population of the City of Glasgow.

(Registrar General's Census Report, 1972).

1801	81,048
1851	344,986
1901	761,709
1951	1,089,767
1970	897,483

In order to understand the problems of social obstetrics in Glasgow it is necessary to trace the development of the city and its medical care from the time of the Industrial Revolution to the present, and to understand why newspapers can still write "It is an acknowledged fact that citizens of Glasgow are the worst housed, the most overcrowded, the most prone to tuberculosis and infant mortality, and the least healthy in Britain, and the situation appears to be getting worse." (The Guardian, 25th September, 1974).

To face Page 3.



Patients of Professor Murdoch Cameron, 1889 - 90.

There is some evidence according to Chalmers (1930) that in the first decade of the 19th century the population of Glasgow was not unhealthy even when judged by later standards. Industrial immigration had already begun, however, and in Glasgow, as in all industrial towns, aggregation was preparing new problems for administration. From 1816 until the early 1870s the closes and wynds of the city were devastated by recurring epidemics of infections. (In 1818 the first recorded epidemic of typhus fever occurred). The need to open up the lanes and widen the streets was recognised at this time, but as Chalmers (1930) states "The industrial concentration found a community increasingly appreciative of the economic and social changes which were taking place, but ill equipped administratively to deal with such as were obviously prejudicial to health."

The position of Glasgow was merely a particular illustration of conditions more widely prevalent, although it seems probable that its more rapid rate of growth and the greater degree of overcrowding which resulted tended to emphasise the grosser defects of the Scottish system of tenement housing. To a very considerable extent the history of sanitation reform in Glasgow has been the history of reform in its housing conditions and the collective circumstances which this implies.

It was this environment, therefore, the dark tenements that encouraged such diseases as rickets, rheumatic fever, tuberculosis and chronic bronchitis, which made childbirth even more hazardous than before. The picture opposite shows the first three ladies who had Caesarean Sections carried out by Professor Murdoch Cameron at the Glasgow Royal Maternity Hospital. The British Medical Journal of 1889 gives an account of a paper presented by Professor Cameron to the British Medical Association Meeting held in Glasgow in August, 1888. The paper was entitled "The Caesarean Section; with notes of a successful case." The mother and child

were presented at the meeting. The patient, a primigravida aged 27, was described as a "little woman, somewhat delicate, and with all the appearance of a patient deformed by rickets in a very marked degree. She stated that she was in labour, at the full term of pregnancy, and had lately enjoyed good health and that she felt foetal movements. The following measurements were taken:- height 49 inches, anterior superior spines $8\frac{3}{4}$ inches, iliac crests $8\frac{1}{2}$ inches, external conjugate $5\frac{3}{4}$ inches, internal conjugate $1\frac{1}{2}$ inches." The operation was successful, carried out under general anaesthesia and a male infant weighing 6 pounds 12 ounces was delivered. Writing the following year, Professor Cameron, after a second successful Caesarean Section, concluded his paper with the following remarks - "I may state it as my firm conviction that the time is speedily approaching when this operation will take the place of craniotomy when the child is alive, and that it only remains for each one who has occasion to perform it to faithfully follow up the work with the same spirit as Sir Spencer Wells and others, in order to sweep from our practice an operation which is antagonistic to our own feelings, and which demands the life of the child whilst it imperils that of the mother. As in this case, let us, when possible, decide beforehand if Caesarean Section is to be performed, avoid unnecessary manipulations or attempts at delivery by other means, and secure strictest antisepsis before, during and after the operation, which should be performed at an early stage."

The first Medical Officer of Health, Sir William Tennent Gardiner, was appointed in 1863 and he was followed some nine years later by Dr. J.B. Russell who remained in this post until 1898. This was the era of sanitary reform. Studies of acute fever and vital statistics were carried out and provision of hospital accommodation for cases of infectious disease and tuberculosis was made. Dr. Russell was very much a social reformer, and his paper entitled

"Children of the City" (1886) has much relevance to the Glasgow of today. In this paper he compares the differing birth and death rates of the town and country populations. "I reason thus to lead you to the conviction, without appealing to the comparative statistics of stature, rate of growth, weight, chest-girth, and all those facts of anthropometry or man-measurement, that it cannot but be that the physique of town born and bred men and women is inferior to that of men and women born and bred in the country. This being admitted, if the proportion of all the children of a nation who are town born is increasing from year to year, then the physique of the whole country must be deteriorating in quality. The rural districts furnish the only resistance to our progress down the inclined plane, and just as the towns absorb the inhabitants of the rural districts, this resistance will become less, and the national descent more rapid.

"Now, you know what I mean by "The Children of the City," and you can estimate the importance of my question - what can we do for them?"

He illustrates the problems of the city by telling the story of Baby Ginx who was probably one of a family of twelve of whom at least half have died during their early life.

"In Glasgow he would have occupied a one or two apartment house in a backland, up several flights of stairs, at the end of a dark lobby. There young Ginx would lie, his first breath, like every subsequent one, filling his lungs with the foul air of a crowded and not very clean house. By and bye he would get an airing, in the shape of a promenade in the arms of his little sister, through the back court, a sunless pit, full of the smoke of washing-houses and the smells of the ashpits. As soon as she got tired or felt anxious for a little independent diversion she would deposit him on the steps of some convenient stair, or on the shivery asphalt. At other times Mrs. Ginx would take him out shopping, having first carefully locked the others in, generally on the Saturday nights, in all weathers, and perhaps from certain difficulties in the way of getting Mr. Ginx

home, returning very late. The nearest place where she could find a sunlit space with trees and grass and some approach to fresh air is a public park a mile or two off, but Mrs. Ginx cannot leave the others, and she is unable to carry or pilot them through crowded streets so far, even if she has not lost all notion of the use of such a trouble, which is very likely. The outcome of the whole situation is that baby is scarcely ever out of that stuffy room. When he begins to creep about, he is constantly in the way, and is either put up on the bed to keep him out of the risk of being scalded or burned or trodden upon, or is sent out with his little nursemaid sister to look at the shop windows, or he planted down in his familiar back court, where he may be seen, like that other child seen by Aurora Leigh on the "uneven pavement" of St. Margaret's Court -

"Whose wasted right hand gambled 'gainst his left,
With an old brass button in a blot of sun."

"Those restless movements of the tiny hands and feet which are so troublesome in the small house and must be restrained, are the first manifestations of that instinctive craving for exercise which characterises all young animals. They are essential for the proper development of the body, and ought to be encouraged. As intelligence grows and as the child obtains more and more command over its body, this instinct expands into the desire to play. Then begin, for all the tribe of Ginx, troubles which thicken the further they advance into boyhood and girlhood."

Russell believed that the only hope for the future was making the cities more like places where children form part of the population and are intended to be reared rather than "places which seem to have been laid out by some Board of Bachelors, or Malthusians, or Herods."

The Maternity and Child Welfare Movement belongs to this century. It began in Glasgow as an effort to reduce the mortality among infants who were fed wholly on artificial milk, but soon developed a much wider purpose when the significance

of a large proportion of the infant deaths was recognised.

Chalmers (1930) states that "These deaths for the most part are not attributable to disease in the ordinary sense - or indeed to disease at all. They arise from defect in the development of the child while as yet unborn - defect, that is, in some primary function of life, the exercise of which is necessary to enable him to meet the new demands of physiologically independent existence, and become equipped to meet the perils of infant life. Their occurrence was recognised as carrying the pattern of infant mortality far beyond the accident of the physical surroundings into which the child is born, save insofar as these might have affected prejudicially the health of the mother, and so impaired the vitality of the child she was nourishing. The prominence thus given to the mother's health opened up for enquiry the whole field of ante-natal influences - not only as represented by her immediate environment and social circumstances, insofar as these fitted her to nourish adequately the child she was to bear, but as being herself a complex of heredity and habit, which in varying directions and in different degrees might impress themselves on her offspring."

Social factors in obstetrics were thus being recognised. In 1873 Farr had shown that, while the infant death rate among legitimate births was 154 per 1000 births, it was 293 per 1000 for illegitimate births. Dr. Chalmers, the Medical Officer of Health for Glasgow (1898 to 1925), introduced a discussion on infant mortality in the first four weeks of life at the XVII International Congress of Medicine held in London in 1913. He showed that there was a relationship between employment of mothers and stillbirths, i.e. in the 4632 employed women 5.4 per cent of their pregnancies ended in stillbirths, whereas in the 29,547 housewives 3.7 per cent of their pregnancies ended in stillbirth. He also showed that there was a relationship between social conditions and stillbirth and infant mortality (Table II).

TABLE II.

Average Rates of Stillbirth and Infant
Mortality (1909-1912)

	<u>Stillbirths</u>	<u>Infant Mortality/1000</u>	
	<u>per cent.</u>	<u>Males.</u>	<u>Females.</u>
Poor Districts	5.7	201	146
Artisan Districts	3.8	121	89
Residential Districts	2.4	70	39
City		143	113

Chalmers (1930) stated that "It is necessary to remember that in these as in all kindred enquiries the poor are more easy of access, and their interest in the object of the enquiry more readily awakened than in other social groupings. For this reason the facts regarding them may be quite accurately stated, and yet error result in comparing them with other groups where corresponding information is less complete."

These studies were made possible by knowing the number of births and deaths in the City. The Registration Act of 1873 as applied to births permitted an interval of 21 days to elapse between the birth and its registration and by this time many infants were dead. In a number of cases it was not known that a birth had occurred until the death was registered. This problem was removed by the Notification of Births Act, 1907, which required information within 36 hours. This Act enabled a direct approach to be made to the mothers of newly born infants not medically attended, while the Extension Act of 1915 enabled Local Authorities to make certain provision for children up to five years of age and enabled both the expectant and nursing mother to be included within any scheme of welfare.

In the City of Glasgow by 1914, there were 14 consultation centres where the mothers could bring their babies for help and advice, and in the Maternity and Child Welfare Departments there were four Medical Officers and 10 trained nurse Health Visitors. This latter number was increased to 20 during the first World War.

The Glasgow Infant Health Visitors Association was formed in 1908. It consisted of a group of 350 ladies, most of whom were well known for their social activities in the City. They co-operated with the Health Visitors by visiting certain families and giving advice on infant care. They watched over and reported the progress of the infants so that appropriate action could be taken, if required. The Midwives (Scotland) Act was passed in 1915, bringing domiciliary midwifery practice under control. There followed the opening of ante natal wards in the Royal Maternity Hospital, the development of Maternal and Child Health Clinics throughout the City. Sir Alexander McGregor, Medical Officer of Health for Glasgow (1925 - 1946) writing in his book "Public Health in Glasgow, 1905 - 1946," says that "The new maternity and child welfare service enabled us to get closer to the people and spread among them a knowledge of mothercraft through the medium of the Medical Officers of the centres and Health Visitors radiating from them, with the assistance of the corps of voluntary visitors who kept in touch with and reported on the progress of individual children."

During the industrial depression of the 1920s the Corporation introduced a free milk scheme and free school meals. During the initial high wave of unemployment in 1921 and the local strike of that year, mothers and young children from over 70,000 families received milk almost entirely free. In 1922, the worst year of the depression, nearly 10 million meals were provided free. Thereafter the number of dinners provided fell to just under a million per annum. The nutrition of school children was greatly improved by the "Milk in Schools" Scheme of 1934. During 1934 to 1936 over 20 per cent of insured persons in the towns of the West of Scotland were unemployed (McGregor, 1967).

Following the first World War an attempt was made to improve housing conditions. A series of Housing Acts were passed, (1919, 1923, 1924) whereby a number of new housing schemes were created. Mansley (1973) in his report "Housing and Social Deprivation" states, however, that "a measure of selectivity was practised by the Corporation in accepting only respectable, skilled, and semi-skilled artisan and lower paid white collar workers for those 1919, 1923, and 1924 Act houses."

The Housing (Scotland) Act, 1930, was introduced to speed up the removal of the slums, and the Housing (Scotland) Act, 1935, was specifically directed at making further and better provision for the prevention of overcrowding and to ensure that the clearance of the slums, redevelopment and planning would go forward as a co-ordinated plan. There then followed the second World War and dissipation of financial resources.

Although public health measures were improving, conditions remained dreadful for a large number of Glasgow families.

In the book "Socially Deprived Families in Britain" (Holman, Lefitte, Spencer and Wilson, 1970), comparisons are made of certain indices of deprivation in various cities in Britain (Table III).

TABLE III.
Indices of Social Deprivation in Britain.

	Over-crowding.	No use of hot tap	No fixed bath	Shared inside W.C.	Outside W.C. only
Great Britain	1.6	12.5	15.4	4.4	16.7
Greater London	2.4	15.0	14.8	13.3	12.6
Birmingham	3.0	17.5	18.6	6.0	27.8
Liverpool	2.6	20.2	25.6	6.4	31.6
Manchester	2.1	13.0	19.5	6.3	31.9
Glasgow	11.8	23.8	32.7	16.1	1.8

Glasgow compares very unfavourable with the other large cities in all respects except that there are now very few outside lavatories. There are, however, a large number of shared W.C.s in the older tenements.

Stillbirths were first required to be registered in England and Wales in 1928 and in Scotland in 1939. By 1950 the term perinatal mortality (stillbirths plus deaths in the first week of life per 1000 total births) had come into general use and is now regarded as the most convenient measure of the standard of obstetrics (Baird, 1970).

Following the rapid reduction of maternal mortality after the introduction of sulphonamides in 1935, obstetricians turned their attention to perinatal loss. As the causes of perinatal death are numerous, attempts to reduce the number of deaths have been slow.

During the 1950s, Baird, Walker and Thomson (1954) in Aberdeen introduced a classification for perinatal deaths based on clinical cause. This proved to be a very useful classification for studying perinatal deaths and it was therefore used in a national survey of perinatal deaths carried out in England, Scotland and Wales for one week during 1958.

This British Perinatal Mortality Survey was carried out in March, 1958. For every baby born between the first minute of March 3rd and the last minute of March 9th of that year a detailed questionnaire was completed. In addition to this, a questionnaire was completed for all stillborn babies and each infant dying before the age of 28 days for the months of March, April and May, 1958. The information obtained has been the result of two publications - "Perinatal Mortality, 1st Report of the 1958 British Perinatal Mortality Survey, under the auspices of the National Birthday Trust Fund (Butler and Bonham, 1963)" and "Perinatal Problems," the second report edited by Butler and Alberman (1969).

Sir John Peel says in his foreword to the second report "The impact that the first report had upon all those working in the field of Obstetrics and Neonatal

Paediatrics has been considerable. Not only have its numerous statistical tables been quoted on many platforms but the emphasis placed upon the avoidable factors has been a great stimulus to further research, to re-thinking many obstetric procedures, and to reorganising a good deal of obstetric practice. Focus of attention upon the high risk group has appreciably influenced the progressive decline in perinatal mortality which fell from 35.0 per thousand in 1958 to 26.3 in 1966."

The perinatal rate of Glasgow has been consistently higher than that for Scotland (Table IV).

TABLE IV.

Birth Rates and Perinatal Mortality Rates for Scotland and the City of Glasgow.

(Registrar General's Report 1972 + M.O.H. Reports for City of Glasgow 1939-1970)

	<u>Birth Rate.</u>		<u>Perinatal Mortality Rate</u>	
	<u>Glasgow.</u>	<u>Scotland.</u>	<u>Glasgow.</u>	<u>Scotland.</u>
1939	19.8	17.4	*	67.9
1945	18.6	16.9	*	52.8
1950	18.4	18.1	49.1	45.1
1955	19.5	18.1	45.6	41.1
1960	21.8	19.6	41.8	37.2
1965	20.8	19.3	35.7	31.5
1970	17.9	16.8	27.6	24.8

Studies of perinatal deaths in the Cities of Aberdeen and Dundee have been very useful in determining the problems in these areas. The maternity services have then been organised to deal with the problems.

At this time of reorganisation of the Health Services in general and the medical services in particular, it is important to assess the problems in a bad area, and this study was undertaken to analyse the causes of the perinatal deaths in the City of Glasgow and attempt to determine the reasons for this high rate. Such a study had not been undertaken previously in this area.

CHAPTER II.

THE SURVEY.

The perinatal mortality rate in the City of Glasgow is higher than the overall Scottish rate and considerably higher than the rate in the other cities in Scotland. (Table V).

TABLE V.

Perinatal Mortality Rates and Birth Rates for the Cities of Scotland, 1970.

<u>Area.</u>	<u>Perinatal Mortality Rates (per 1000 total births).</u>	<u>Birth Rates (live births per 1000 total population)</u>
Scotland	24.8	16.8
Glasgow	27.6	17.9
Edinburgh	22.2	14.0
Aberdeen	20.0	14.4
Dundee	19.0	16.1

(from the Report of the Medical Officer of Health, City of Glasgow, 1970).

Because of concern for this high perinatal mortality rate, a number of studies were carried out in Glasgow during the 1960s. In the first, Smith and Macdonald (1965) showed that in 1963 there was not a very efficient selection of cases for hospital delivery. More than 10 per cent of primiparae and 41 per cent of mothers having their fourth or subsequent child were not delivered in hospital. They also found that a history of previous stillbirth did not appreciably raise the chances of hospital delivery, and a history of previous abortion was actually associated with a hospital confinement rate below that for all births. A similar analysis was made of births in 1967 (Richards, Hamilton

and Nicholson, 1969). They found that in spite of a rapidly rising hospital confinement rate, an appreciable number of high risk births continued to occur at home. Among births to primigravidae, 93.9 per cent were in hospital, but only 75.5 per cent of births to women having a fourth or subsequent baby. Previous stillbirths did not raise the chances of hospital delivery (84.3 per cent), nor did maternal age of 35 years or over (82.5 per cent). Reasons for this included, on the part of the mothers a dislike of hospitals and a desire to be with their families, and on the part of the general practitioners a failure to ensure hospital confinement for many high risk cases. Richards, Donald and Hamilton (1970), in their paper on the Use of Maternity Care in Glasgow, state that "it is clear from this investigation that the G.P. is the key figure in selecting the place of confinement and that a high proportion of mothers will accept his decision. It is disturbing, therefore, that less than 40 per cent of the doctors stated that, in their practices, first and high parity births were among the most common indications for hospital delivery." Richards et al. (1970) also found that although a large proportion of Glasgow's population lives in conditions of squalor which are among the worst in Europe, nevertheless the Domiciliary Midwifery Service is prepared to deliver women in any home if it is the general practitioner's wish. Many of these houses are the notorious "single ends," one room which is used for living, sleeping, cooking and eating - conditions which make home delivery particularly difficult.

A statistical examination for other possible factors responsible for Glasgow's high rate has shown that a high birth rate, a large population of high parity births, adverse socio-economic conditions (both past and present) and poor maternal physique, all contribute to the current situation (Richards, Hamilton and Nicholson, 1969).

In 1970 there were 818 obstetric beds in the City of Glasgow. The increase

in the hospital confinement rate from 71 per cent in 1963 to 85 per cent in 1967 was achieved by the earlier discharge of patients from hospital. The opening of a new maternity hospital of 114 beds in 1964 was offset by the closure of two obstetric units with 157 beds.

Almost all patients first attend their general practitioner for confirmation of pregnancy and to make arrangements for antenatal care and confinement. A small number go direct to a Local Authority Clinic. If hospital confinement is considered desirable, the general practitioner writes to the maternity hospital of his choice (usually that nearest to the patient's home) and, if the hospital agrees to the booking, the patient is sent an appointment to attend the antenatal clinic. If the patient is not accepted for booking, the hospital refers her to the Western Regional Hospital Board's Hospital Admission Department where hospital accommodation is found, but sometimes 8 to 10 miles from the patient's home.

This present study was designed to carry the examination of the problem a stage further. The aims were three-fold - firstly to classify all perinatal deaths in 1970 by clinical cause, secondly to identify the women who were losing their babies and to determine whether they came for and received adequate antenatal care, and finally to determine what changes, if any, might be recommended in obstetric and paediatric care to improve the perinatal outcome.

METHOD

In order to classify all the perinatal deaths in 1970, it was necessary to study the individual case records of Glasgow women having perinatal deaths in that year in the City of Glasgow. The information required was set out on a five-page coding sheet under the various headings detailed in Table VI.

TABLE VI.

Information for Coding.

1. Basic Data - including age
height
Social Class
marital status
ethnic classification
2. Social Data - housing conditions.
3. Previous Obstetric History.
4. Present Pregnancy - antenatal care
antenatal complications
antenatal procedures
weight gain
5. Details of Labour.
6. Details of Infant.
7. Neonatal Findings.
8. Transfers.
9. Autopsy Report.
10. Registered Cause of Death.
11. Clinical Cause of Death.

A copy of the Coding Sheet is contained in the Appendix.

FINDINGS

During 1970 there were 16,748 births to women resident in the City of Glasgow. There were 462 perinatal deaths, giving a perinatal mortality of 27.6 per thousand live and stillbirths.

The majority of births (15,835) in Glasgow occurred in one of the nine maternity hospitals, while 913 births occurred at home. From this domiciliary group there were 34 deaths, giving a perinatal mortality rate of 37.3 per thousand live and stillbirths.

The total number of deliveries to women resident in Glasgow by the various hospitals is shown in Table VII.

TABLE VII.

Number of Births by Place of Confinement.

	Births to Glasgow Women.	Perinatal Deaths.	Perinatal Mortality Rates.
Glasgow Royal Maternity Hospital	3423	123	35.9
Southern General Infirmary (+ G.P. Unit)	2039	54	26.5
Robroyston Hospital	2008	51	25.4
Queen Mother Hospital	1800	49	27.2
Stobhill Hospital	1658	58	35.0
Belvidere Hospital (+ G.P. Unit)	1361	9	6.6
Redlands Hospital for Women	1211	21	17.3
Duke Street Hospital	859	37	43.1
Ross Hospital	851	1	1.2
Home	913	34	37.3
Other - Nursing Home	337		
N/S	288	25	
Total	16,748	462	27.6
Glasgow Royal Maternity Hospital + Ross Hospital	4274	124	29.0
Duke Street Hospital + Belvidere Hospital	2220	46	20.7

As the Ross Hospital is staffed by obstetricians from the Royal Maternity Hospital and Belvidere Hospital is staffed by obstetricians from Duke Street Hospital and the deliveries in these hospitals are essentially normal, it is necessary when looking at perinatal mortality rates to combine the figures for these hospitals.

Before details of the perinatal deaths were obtained, permission was received from the obstetricians in each hospital to study the relevant case records. Permission was also obtained from the Corporation of Glasgow Medical

Officer in charge of Obstetric Services to study the domiciliary notes.

A record of perinatal deaths was not kept by all hospitals but the Records Officers were usually able to make a list of the names of the patients who had had perinatal deaths during 1970. The tracing of records was greatly helped by using information recorded and stored by the Glasgow Corporation Health Department, using the Glasgow Child Health Record linkage system. A print-out of all perinatal deaths occurring in 1970, by hospital of delivery, hospital number, date of birth and mother's name, age, and address, was obtained. This information often proved to be more accurate than that obtained from the various hospitals, and combining the information from both sources the perinatal list was drawn up.

Each hospital was visited. The case records of all perinatal deaths were scrutinized and the information recorded on the coding sheets. The records of the domiciliary confinements where perinatal death occurred were also studied. The clinical cause of death was then determined and coded according to the Aberdeen classification of Baird, Walker and Thomson (1954) which will be described in detail in the following chapter.

The social data of each mother and the registered cause of the perinatal death were obtained from the Glasgow Corporation Health Department, which has information routinely recorded for each birth in Glasgow and stored in the computer-based system of linked Child Health Records (Smith, Richards, Nicholson and Granick, 1970; Richards and Nicholson, 1970).

The records of 437 perinatal deaths (262 stillbirths and 175 neonatal deaths) were studied in detail. Records were not available for the remaining 25 cases; 11 were delivered in hospitals a considerable distance from Glasgow. These babies were born to women whose normal residence was Glasgow, but who had gone into labour while on holiday or away from home for the day. The hospital records

of a further 12 cases could not be traced, and no details at all were available for the remaining 2 cases.

All the information recorded for the 437 cases studied in detail was analysed with the help of the Glasgow Corporation I.B.M. Computer (I.B.M. 360 Model 30), and the following chapters deal with the results of this analysis.

CHAPTER III.

CLINICAL CLASSIFICATION

The classification that was used in studying the perinatal deaths (occurring in Glasgow during 1970) was devised in Aberdeen by Baird, Walker and Thomson (1954). This classification was drawn up because the Aberdeen group felt that a classification of deaths according to clinical findings was more meaningful in terms of causation than a classification based primarily on pathological findings. The aim is to classify each death according to the factor which is most likely to initiate the train of events resulting in perinatal death.

The following eight main groups were differentiated. (Table VIII)

TABLE VIII.

Clinical Classification

<u>Groups</u>	<u>Classification</u>
(1) Mature, Cause unknown; birth weight over 2500 g.	M.U.
(2) Premature, Cause unknown; birth weight 2500 g. or less	P.V.
(3) Trauma	Tr.
(4) Toxaemia	Tox.
(5) Antepartum Haemorrhage	APH
(6) Maternal Disease	M.D.
(7) Fetal Deformity	F.D.
(8) Other (Rh factor, infection, etc.)	Other

Beird et al. (1954) suggested that the causes of perinatal death fell into two broad categories. The first group are deaths due to obstetrical causes (toxaemia, mechanical causes, unexplained deaths of mature babies and Rhesus incompatibility), and the number is capable of being reduced by high standards of obstetric care. The second group (unexplained prematurity, fetal defects, and antepartum haemorrhage) is less amenable to obstetrical intervention, being related to the effects of unfavourable environmental influences on the mother's reproductive function.

This classification was also used in the British Perinatal Survey which took place in 1958, and the following detailed description of the classification is largely taken from the second report of that survey entitled Perinatal Problems, edited by Butler and Alberman (1969).

(1) Mature, Cause Unknown.

The baby weighs more than 2500 g. at birth. Many die in utero not infrequently before the membranes rupture or labour begins. Others after a normal labour of average length are born asphyxiated and fail to establish or maintain respiration. There is often evidence of fetal distress during or occasionally before labour, but no obvious reason for it. This group is frequently associated with postmaturity.

(2) Premature - Cause Unknown.

The baby weighs 2500 g. or less at birth. Pregnancy and labour are clinically normal, apart from early onset of labour or low birth weight at or near term. There is no obvious clinical cause for the immaturity or impaired intra-uterine growth. If the baby is born alive and in good condition it may die of a variety of causes, but where the birth weight is under 1815 g. the category prematurity takes precedence over terminal states such as infection. The finding at postmortem of atelectasis with or without hyaline membrane or of

massive pulmonary haemorrhage is consistent in this classification with an allocation to premature - cause unknown. If there is a clinical "cause" for the prematurity such as toxæmia, this takes precedence. This group is further divided into light-for-dates babies and pre-term babies.

(3) Trauma.

This group includes all cases where the apparent or actual cause of death (in the absence of malformation or Rhesus incompatibility) is attributable to mechanical obstruction or to damage during labour. The death can be taken as mechanical in origin, even in the absence of postmortem findings such as a tentorial tear, if the history of the labour is highly suggestive, and it seems reasonable to suppose that the child would have survived if delivery had been uncomplicated. Conversely, a postmortem finding of "cerebral birth trauma" is considered but may be disregarded if the labour appeared to have been perfectly normal and unlikely to be associated with sufficient stress to kill the baby. The assumption that is made in such cases is that there is some intrinsic fault in the baby and that improved management of labour would not have been likely to prevent the death.

All deaths associated with breech or shoulder presentation are considered as being due to mechanical causes unless a serious malformation is present or the baby is extremely small, weighing less than 1815 g. In such cases of low birth weight, the condition causing the premature delivery is given priority as the cause of death. Prolapse or compression of the cord except in the presence of malformation or low birth weight is included in this group.

(4) Toxaemia.

The definition of toxæmia is based on that of Nelson (1955). It is a condition in which blood pressure rises after the 24th week of pregnancy to 90 mm. Hg. diastolic or more, recorded on at least two separate days; the

differentiation into severe and mild forms depends on the presence or absence of proteinuria. In cases when there is both toxæmia and antepartum hæmorrhage, toxæmia takes precedence over antepartum hæmorrhage as a cause of death.

Essential hypertension and hypertension secondary to chronic nephritis are considered to be maternal disease, but only if there is definite evidence that the condition was present before pregnancy. Where there was high blood pressure in early pregnancy, but without any further evidence of pre-existing hypertensive complications, cases are allocated to toxæmia, rather than to maternal disease.

(5) Antepartum Hæmorrhage.

This category includes all cases where death is obviously or probably the result of antepartum hæmorrhage, except hæmorrhage associated with toxæmia. Concealed accidental hæmorrhage is included.

(a) Accidental Hæmorrhage: Definite or highly suggestive evidence of abruptio placentæ, e.g. acute abdominal pain or retroplacental clot.

(b) Placenta Prævia: Objective diagnosis is required.

(c) Uncertain Origin: Includes all cases which could not be allocated to the above.

(6) Maternal Disease.

Cases in which the mother suffered during pregnancy from an incidental medical or surgical condition such as diabetes, pneumonia, syphilis or appendicitis, apparently leading to intra-uterine death or to the birth of a premature or feeble baby.

(7) Fetal Deformity.

In this classification serious malformations take precedence over all potential causes of death in a given case, and with few exceptions the decision of the pathologist in cases of congenital malformation is decisive.

(8) Other.

This group is mainly composed of Rhesus incompatibility which takes precedence over all others, except where there is a malformation. In the absence of a diagnosis based on postmortem examination findings, reliance is placed upon evidence from blood groups and antibody titres or a highly suggestive clinical history. Also included in this group are infections of fetus and infant and a miscellaneous group of specific causes of death not already covered.

CHAPTER IV.

MATERNAL CHARACTERISTICS.

The successful outcome of a pregnancy depends to a large extent on the health and physique of the mother, and her physiological response to pregnancy often reflects the type of baby which will be produced. If there is a good response in terms of absence of disease and a high weight gain, a baby of average or above average weight will probably be produced. When there is a poor response in terms of maternal weight gain the baby may be "small for dates."

Duncan, Baird and Thomson (1952) more than twenty years ago, wrote that "the lowest obstetric mortality is to be found in communities or groups in which most women are healthy and have always lived in a good social environment, have their first baby before the age of 30, limit their family to three or four, and in which skilled medical attention is available." The influence of age, parity, social class and place of confinement was clearly demonstrated and in some centres awareness of the mothers at risk has resulted in altered obstetric management and reduction in perinatal loss.

All the perinatal deaths (including multiple pregnancies) occurring in the City of Glasgow during 1970 were classified by the clinical classification described in the previous chapter, divided into environmental and obstetric groups, and then analysed by maternal characteristics.

The causes of perinatal death are shown in Table IX.

TABLE IX.

Perinatal Death Rate by Clinical Cause (Glasgow City Births, 1970)

Number of Births = 16,748

	No.	Rate /1000 live and stillbirths.
M.U.	50	3.0
P.U.	101	6.0
Tr.	23	1.4
Tox.	29	1.7
APH	94	5.6
M.D.	12	0.7
F.D.	105	6.3
Other	23	1.4
Total	437	26.1
Unknown	25	1.5
Final Total	462	27.6

Two-thirds of the 437 perinatal deaths studied in detail fell into the "environmental" group, and one third were from "obstetrical" causes (Table X). The leading causes of mortality were, in rank order - fetal deformity (6.3 per thousand), premature - cause unknown (6.0), antepartum haemorrhage (5.6), and mature - cause unknown (3.0).

Table X shows how perinatal mortality is related to a number of maternal variables.

TABLE X.

Perinatal Mortality Rates (per 1000 births) for Individual Causes according to several Maternal Characteristics

(Figures in brackets are those where twin deaths have been regarded as single deaths).

Maternal Characteristics	Births	Environmental				Obstetrical				Grand Total		
		P.U.	APR	M.D.	Total	M.U.	Tr.	Tox.	M.D.		Other	
All births	16,748	6.0	5.6	6.3	17.9	3.0	1.4	1.7	0.7	1.3	8.1	27.6
Age: Less than 20	2,060	10.2	7.8	7.8	25.7	1.5	0.5	2.4	1.0	0.0	5.3	31.1
20-29	9,815	5.2	5.1 (4.7)	6.1 (5.0)	16.4	2.6	1.6	1.4	0.5 (0.4)	1.7	7.9	24.4
30-39	3,601	7.5	5.8	7.2	20.5	5.0	1.7	2.8	1.4	1.7	12.5	33.0
40 and over	367	5.4	19.1	5.4	30.0	8.2	0.0	0.0	0.0	0.0	8.2	38.1
Parity: 0	5,622	7.8	4.6 (7.1)	6.4 (4.5)	18.9	2.3	1.6	3.4	0.0	0.7	8.0	26.9
1	4,272	5.6	4.4 (4.9)	6.6	16.6	1.2	2.1	0.5	0.7	0.7	5.1	21.8
2	2,589	3.5	5.8	6.2	15.4	3.1	0.0	0.7	0.7	0.7	5.4	20.9
3 and over	3,588	6.7	9.5	7.0	23.1	6.7	1.4	1.7	2.0 (1.7)	3.9	15.6	38.7
Height: - 5'1"	5,184	9.3	6.0 (8.7)	7.9 (5.8)	23.1	4.2	2.3	2.5	1.4 (1.2)	1.5	12.0	35.1
5'2" - 5'4"	8,966	4.5	5.9 (4.0)	6.1	16.5	2.7	0.9	1.6	0.2	1.4	6.8	23.3
5'5" +	1,641	4.3	4.9	4.3	13.4	1.2	1.2	0.6	1.2	0.6	4.9	18.3
Social Class: I	548	1.8	1.8	5.5	9.1	1.8	1.8	1.8	0.0	0.0	5.2	14.6
II	1,028	3.9	2.9	6.8	13.6	1.0	1.0	1.9	0.0	1.0	4.9	18.5
III	8,428	5.2	5.2 (5.0)	5.7 (5.1)	16.1	3.4	1.5	2.1	0.5	0.9	8.5	24.7
IV	3,203	8.1	4.7 (7.2)	8.4	21.2	1.9	1.6	1.6	0.9	1.9	7.8	29.0
V	2,245	9.4	10.7 (8.5)	6.7	26.7	3.4	0.9	1.3	1.8 (1.3)	3.1	10.2	37.0
Legitimacy: Para 0 legitimate	4,772	6.7	4.0 (6.1)	6.7	17.4	2.3	1.5	3.1	0.0	0.6	7.3	24.9
illegitimate	807	14.9	8.7 (13.6)	5.0	28.6	2.5	2.5	5.0	0.0	1.2	11.2	39.7
Para 1+ legitimate	9,560	5.6	6.7 (5.3)	6.8 (6.6)	19.1	3.3	1.3	0.9	1.2 (1.1)	1.9	8.6	27.7
illegitimate	874	3.4	4.6	4.6	12.6	2.3	2.3	0.0	0.0	1.1	5.7	18.3
Place of confinement: Hospital	15,835	5.7	5.9	5.9	17.5	2.7	1.3	1.8	0.8	1.3	7.9	25.4
Home	913	11.0	1.1	13.1	25.2	7.7	3.3	0.0	0.0	1.1	12.1	37.8
Previous history: Abortion	2,185	9.6	12.8	12.8	35.2	5.0	0.9	1.8	1.4	1.4	10.5	41.2
Stillbirth	486	10.3	14.4	24.7	49.4	8.2	4.1	6.2	2.1	10.3	30.9	80.2
Neonatal Death	394	7.6	22.8	5.1	35.5	10.2	2.5	0.0	0.0	5.1	17.8	53.3

Note: The grand total includes the 25 deaths for which no records were available.

Significantly high rates are shown throughout the test as follows:

+p < 0.05 **p < 0.01 *** < 0.001.

- (1) Age: Mortality was lowest (24.4 per thousand) in mothers aged 20-29 years. The high rate at age 40 and over (38.1) was due to high mortality in the environmental group of deaths.
- (2) Height: Mortality was lowest (18.3) in tall women and highest in small women (35.1 ***).
- (3) Parity: Mortality was lowest in second and third births (21.8 and 20.9 respectively). The high rate in fourth or subsequent births (38.7***) was due to high mortality in both groups of causes.
- (4) Social Class: There was a steep gradient with rates increasing from 14.6 in Class I to 37.0* in Class V. This trend reflects largely the gradient in the environmental group.
- (5) Legitimacy: Mortality was high among primigravidas with illegitimate births (39.7) due to a high rate in the environmental group.
- (6) Place of Confinement: Overall mortality was much higher in domiciliary births (37.3) than in hospital births (25.4), with a similar pattern in the two groups of causes.
- (7) Previous History: Particularly high mortality occurred if there had been a previous abortion (41.2***), stillbirth (80.2***) or neonatal death (53.3***). For each of these complications, rates were high in both groups of causes.

Individual Causes:

The following high rates are noteworthy.

- (1) Premature - Cause Unknown (overall rate 6.0): age under 20 (10.2*), social class V (9.4), primigravid illegitimate (14.9**), home confinement (11.0), previous abortion (9.6) or stillbirth (10.3), and small women (9.3**).
- (2) Antepartum Haemorrhage (overall 5.6): age 40 and over (19.1**), parity 3 and over (9.5*), social class V (10.7*), primigravid illegitimate (8.7), previous abortion (12.8***), stillbirth (14.4*) or neonatal death (22.8***).

- (3) Fetal Defect (overall rate 6.3): home confinement (13.1*), previous abortion (12.8***) or stillbirth (24.7***).
- (4) Mature - cause unknown (overall rate 3.0): age 40 and over (8.2), parity 3 and over (6.7***), home confinement (7.7*), previous stillbirth (8.2) or neonatal death (10.2*).
- (5) Trauma (overall rate 1.4): illegitimate - whether primigravid (2.5) or multigravid (2.3), home confinement (3.3), previous stillbirth (4.1) or neonatal death (2.5).
- (6) Toxaemia (overall rate 1.7): age under 20 (2.4*) or 30-39 years (2.8), primigravidae (3.4) whether illegitimate (5.0) or legitimate (3.1), previous stillbirth (6.2).
- (7) Maternal Disease (overall rate 0.7): parity 3 and over (2.0).
- (8) Other Obstetrical Causes (overall rate 1.3): parity 3 and over (3.9**), social class V (3.1), previous stillbirth (10.3***) or neonatal death (5.1).

Inter-related Variables:

Several of the maternal variables examined are inter-related. The separate effects of age and parity are shown in Table XI and of social class and parity in Table XII. Perinatal mortality was highest in

- (i) births to primiperae below the age of 20 and in the age groups over 30,
- (ii) fourth or subsequent births, especially in classes III, IV and V,
- (iii) other births in classes IV and V (except the second).

TABLE XI.

Perinatal Mortality Rates according to
Maternal Age and Parity.

(related births are shown in parenthesis)

Maternal Age (Years)	Parity.				Total *
	0	1	2	3 and over.	
Less than 20	35.3 (1503)	25.6 (390)	27.0 (37)	0.0 (2)	31.1 (2060)
20 - 29	24.0 (3379)	22.7 (3000)	20.3 (1672)	40.5 (1384)	24.4 (9815)
30 - 39	44.9 (356)	22.3 (629)	26.0 (693)	38.6 (1840)	33.0 (3601)
40 and over	45.5 (22)	0.0 (38)	21.3 (47)	47.4 (253)	38.1 (367)
Total *	26.9 (5622)	21.8 (4272)	20.9 (2589)	38.7 (3588)	26.1 (16748)

* Totals include 677 births in which parity was not known
and 905 births in which age was not known.

TABLE XII.

Perinatal Mortality Rates according to Social Class

and Parity.

(related births are shown in parenthesis).

Social Class.	Parity.				Total *
	0	1	2	3 and over	
I and II	20.0 (701)	11.5 (434)	15.4 (195)	29.1 (172)	17.9
III	25.0 (2917)	21.6 (2272)	17.0 (1295)	38.1 (1679)	25.5
IV and V	36.5 (1697)	25.8 (1318)	31.1 (900)	38.3 (1354)	33.4
Total *	26.9 (5622)	21.8 (4272)	29.9 (2589)	38.7 (3588)	26.1 (16748)

* Totals include 677 births in which parity was not known

and 1137 births in which social class was not recorded
(mainly illegitimate births).

DISCUSSION

Analysis of the maternal characteristics involved in perinatal loss has confirmed the well known increase in risk of a perinatal death when the mother is aged less than 20 or more than 30, when she is primiparous or of high parity, or if she is in Social Class IV or V. The risk is also high in illegitimate primiparous births and when there is a previous history of abortion, stillbirth or neonatal death. In this study, a high mortality rate also occurred among domiciliary births.

The various causes of deaths will be discussed in greater detail in subsequent chapters but the general findings were as follows. In the obstetric group of deaths, the leading diagnosis was nature - cause unknown. In these deaths, the rate was greatly increased among high parity births and an awareness of the possibility of fetal loss in these women and close surveillance during pregnancy might reduce the rate significantly. Improved care can also be expected to reduce the number of deaths from trauma, toxæmia, maternal disease and Rhesus incompatibility.

Two-thirds of the deaths were in the environmental category and to bring about a significant reduction in the overall perinatal mortality rate some improvement must be looked for in this group. Although in the long term, the rate will fall as maternal health improves, physique increases and living conditions become better, some improvement could be produced immediately. Patients should be made aware of the benefits of early antenatal care and adequate nutrition.

Macnaughton (1974), writing in *The Practitioner* about antenatal care, states that "It is desirable that all confinements should take place in a properly equipped maternity hospital where mother and child can be closely supervised and provision made for any emergency. Antenatal care is really preventive medicine and during this period arrangements should be made, so

far as is possible, to ensure that the patient is delivered in a hospital where all the facilities that she is likely to require are available. This poses some problems since not all maternity hospitals are equipped to deal adequately with all emergencies. This is particularly so in the case of intensive neonatal care facilities, which are not available in all maternity units. Ideally therefore whenever it is known that a woman may deliver a child likely to require intensive neonatal care, arrangements should be made to confine the patient in a unit with a comprehensive neonatal service. If this is not possible, proper transfer facilities, with possibly a specially equipped ambulance, are a second best. It is better to transfer the baby in utero than ex utero."

Moreover, the high mortality from environmental causes in high parity women suggests that a reduction in such pregnancies by contraceptive advice if this is what the patient wishes, will bring about a marked lowering of the mortality rate. Hellman (personal communication) from Downstate Medical Center in the heart of a socially deprived area of Brooklyn has shown what can be achieved by introducing very active family planning clinics into the area. There was much opposition to his setting up such a clinic for the patients of the area, a number of whom were second and third generation unemployed of negro or Puerto Rican origin. His critics said that the patients did not wish help and would not attend the clinic, but the opposite proved to be true.

It is surely a criticism of the medical and social services of Glasgow that the perinatal mortality has remained higher than most other cities in the country. The existence of the poor socio-economic conditions and religious beliefs of a number of the inhabitants has been used as an excuse over the years instead of a challenge. Other centres have managed to improve

perinatal loss by active obstetric care and much can be done in Glasgow by identifying the women at risk early in pregnancy, monitoring them carefully during the antenatal period and delivery, and making family planning advice easily and freely available.

Close liaison between obstetrician, general practitioner, health visitor and social worker, provided they were all aware of the high risk patients, would ensure that the patients were seen early in their pregnancy. Those patients who refused to attend hospital antenatal clinics could be visited at home, and more clinics could be carried out in housing schemes for those patients living far from obstetric hospitals.

CHAPTER V.

PERINATAL DEATHS DUE TO -

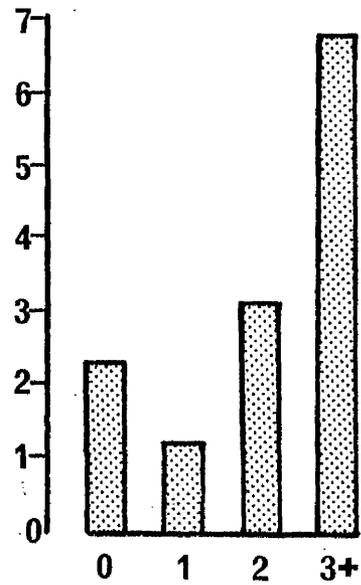
- (a) Mature, Cause unknown.
- (b) Premature, Cause unknown.
- (c) Trauma.
- (d) Toxaemia.
- (e) Antepartum Haemorrhage.
- (f) Maternal Disease.
- (g) Fetal Deformity.
- (h) Rhesus Incompatibility and Other.

FIG. 1.

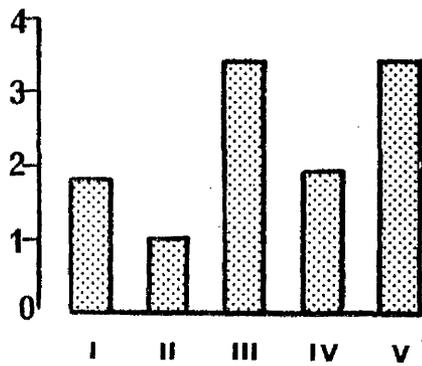
Mature (cause unknown)



Parity



Social Class



(a) MATURE - CAUSE UNKNOWN

All the babies in this group weighed more than 2500 g., appeared normal and the cause of death was not obvious. Baird, Thomson and Duncan (1953) first drew attention to this cause of perinatal deaths when they noted a temporary increase in the stillbirths from this group during the late 1940s. They suggested that the postponement of childbearing during the war years had been one of the factors causing this temporary increase as these deaths were found to be uncommon at early ages and increased steadily with age. Work by Walker (1954) suggested that the postmature fetus was striving to make the best use of a diminishing oxygen supply. Walker showed that as pregnancy is prolonged beyond 40 weeks the fetus suffers from an increasing shortage of oxygen, which under certain conditions can prove fatal. He states that "It is not at all clear why this deterioration should occur. There is still a great deal to be learned about the physiology of normal pregnancy and the effects and mechanisms of deviations from the normal. It would appear that, in most women, prolonged pregnancy is pathological, but it is impossible to say why labour should fail to occur, or when, in the given case, a delayed onset of labour becomes particularly dangerous to the foetus." It therefore appears that the important factor in this group is the relationship between the fetus and the placenta. Relative placental insufficiency occurs either when an enhanced growth potential of the fetus outstrips the resources of a normal placenta, or when the placenta becomes unable to support even the normal growth structurally or functionally. As Alberman (1974) states "the latter situation may arise when a fetus outstays its normal time in the uterus and continues to grow or attempts to grow at a time when the placenta is beginning to involute. The resulting state of relative malnutrition is merely an exaggeration of the normal slowing down of growth near term, but may become

lethal if it continues too long or is followed by a prolonged or difficult labour." A high standard of obstetric care and awareness of the patients at risk ought to reduce the number of perinatal deaths in this category.

FINDINGS

During 1970 there were 50 perinatal deaths classified as mature, cause unknown in the City of Glasgow. The perinatal mortality rates by age, parity and social class are illustrated in Fig. 1. There was a sharp increase in perinatal loss with increasing age and in those women having their fourth or subsequent child. The perinatal mortality rate in small women (4.2 per 1000) was almost four times that found in tall women (1.2 per 1000). Seven of these deaths occurred at home and are discussed in detail in the chapter on Domiciliary Confinement. The maternal characteristics and gestation lengths of the pregnancies are shown in Table XIII.

TABLE XIII.

Maternal Characteristics and Gestation Lengths of Mature, Cause Unknown Domiciliary Deliveries.

Age.	No.	Parity.	No.	Social Class.	No.	Height.	No.	Gestation Length.	No.
< 20 yrs.	1	0	1	I	1	0 - 5'1"	3	38 wks	2
20 - 29	2	1	0	II	0	5'2" -		38 wks.	0
30 - 39	4	2	1	III	6	5'4"	3	39 wks.	1
40 & over	0	3+	5	IV V	0 0	5'4"+	1	40 wks. 40+ wks.	4 0

The interesting findings in this group were that none of the patients was suitable for delivery at home on medical and obstetric grounds (one patient had a psychiatric history) although all had good homes, (one primigravida and 5 highly parous patients) and none of the babies was postmature. Two patients received no antenatal care, one was an unmarried teenage primigravida, and the other had requested termination which had been refused.

Forty three patients were delivered in hospital. There were 12 primiparae and 31 multiparous patients in this group. Table XIV shows the maternal characteristics of the primiparous patients.

TABLE XIV.

Maternal Characteristics.

Age	No.	Social Class	No.	Height.	No.
< 20 years	2	I	0	0 - 5'1"	4
20 - 24 "	6	II	1	5'2" - 5'4"	5
25 - 29 "	1	III	8	5'4"+	2
30 and over	3	IV	1	N/S	1
		V	2		

The details of the babies and gestation length when these primiparous patients first attended the antenatal clinic are shown in Table XV.

TABLE XV.

Details of Babies and First Antenatal Visit

No.	1st Visit to Clinic	Gest. Length	Birth Weight	Percentile Baby Wts.	Labour.	Outcome.	Comment.
1	9	37	2540	-	Spont.	NND	
2	No care	38	2835	-	Spont.	IUD	Aged 33, married 7 yrs. Did not know she was pregnant.
3	?	38	3650	< 90th	Spont.	IUD	
4	14	40	2800	-	Spont.	IUD	
5	23	40	3170	< 10th	Induced	NND	Aged 34. Buccal pitocin T+3. ARM - no liquor. Irreg. F.H. in labour. Meconium aspiration.
6	21	40	3982	< 95th	Induced	NND	Aged 31. Aspiration Syndrome.
7	9	42	2630	< 5th	Induced	NND	Aspiration Syndrome.
8	31	42	2665	< 10th	Spont.	NND	No wt. gain for 6 weeks before delivery.
9	10	42	4153	< 90th	Induced	SB	
10	13	43	3340	-	Spont.	SB	
11	16	43	2608	< 5th	Induced	IUD	
12	?	?	3260	-	Spont.	SB	Fetal heart stopped after 17 hrs. labour, then ARM carried out, thick meconium.

Only 6 of these primiparous patients attended the antenatal clinic before 20 weeks gestation. Five patients were more than 14 days past term but none of these patients was more than 30 years old. A number of these babies (Cases 5, 7, 8, 9, 10, 11) might have been saved if a more active induction policy had been undertaken.

Tables XVI and XVII illustrate the maternal characteristics of the parous patients.

TABLE XVI.

Maternal Characteristics of Para 1 and Para 2 Patients (12)

Age.	No.	Social Class.	No.	Height.	No.
< 20 years	0	I	0	0 - 5'1"	7
20 - 29 years	11	II	0	5'2" - 5'4"	5
30 - 39 years	1	III	6	5'4"+	0
		IV	4		
		V	2		

There were 7 small women in this group, and 6 patients came from Social Classes IV and V.

TABLE XVII

Maternal Characteristics in Para 3+ patients (19)

Age.	No.	Social Class	No.	Height.	No.
< 20 years	0	I	0	0 - 5'1"	8
20-29 years	6	II	0	5'2" - 5'4"	4
30-39 years	10	III	9	5'4"+	6
40 and over	3	IV	1	N/S	1
		V	3		
		N/S	6	<u>Marital Status</u>	
				Single	1
				Married	1
				Sep/div.	2
				Unknown	2

The only unusual finding in this group is the number of patients where the Social Class is not known. This was presumably due to these patients changing marital status.

Tables XVIII and XIX show the details of the babies and when the mother first attended the antenatal clinic in these two groups of parous patients.

TABLE XVIII.

Details of Babies and first antenatal visit in Para 1 and Para 2 Patients.

No.	1st A/N Visit	Gest. at delivery.	Birth Weight.	Percentile Baby Wt.	Labour.	Outcome.	Comment.
1	19	36	2551	< 10th	Spont.	IUD	
2	22	36	3280	< 75th	Spont.	IUD	Wt. loss throughout pregnancy.
3	27	38	3500	-	Spont.	IUD	Wt. gain not recorded
4	No care	39	2580	-	Spont.	IUD	
5	34	39	2800	-	Spont.	IUD	
6	14	39	3629	< 50th	Spont.	IUD	Wt. gain only 3½ lb. in last 10 weeks.
7	19	40	3118	< 75th	Spont.	IUD	No wt. gain for last 2 clinic visits.
8	19	40	3544	< 90th	Spont.	SB	
9	No care	41	2721	-	Spont.	IUD	
10	16	41	3111	< 25th	Spont.	IUD	Weight gain satisfactory.
11	26	41	3680	-	Spont.	IUD	
12	27	44	2495	< 5th	Spont.	IUD	Weight loss during pregnancy.

TABLE XIX.

Details of Babies and time of first antenatal visit in Para 3+ patients.

No.	1st a/n Visit	Gest. at Delivery	Birth Weight	Percentile Baby Weight	Labour.	Outcome.	Comment.
1	27	36	2835	< 10th	Spont.	IUD	
2	?	36	3359	-	Spont.	IUD	Very poor social condns.
3	34	36	3657	< 90th	Spont.	SB	Fetal heart stopped 3 hrs. before delivery.
4	26	38	3220	-	Spont.	SB	No record of weight gain
5	28	39	2920	< 50th	Induced	NND	
6	33	39	2920	< 25th	Spont.	SB	
7	21	39	3180	< 50th	Spont.	IUD	No weight gain for last 8 weeks.
8	21	39	3260	< 25th	Spont.	IUD	4 lb. weight loss noted at last clinic visit
9	No care	40	2920	-	Spont.	IUD	No action taken. In prison until 2 wks. before del.
10	29	40	2948	< 50th	Spont.	IUD	Wt. loss in last 6 wks.
11	29	40	3912	< 75th	Spont.	NND	Aspiration syndrome.
12	26	41	2750	< 10th	Spont.	IUD	Wt. loss noted at term.
13	20	41	2764	< 5th	Spont.	IUD	
14	26	41	4000	< 90th	Spont.	NND	Aspiration syndrome.
15	25	42	3230	< 50th	Spont.	SB	No wt. gain last 8 wks.
16	19	42	3230	-	Spont.	SB	5lb. wt loss in 2 wks.
17	19	42	4139	< 90th	Induced	IUD	No wt. gain in last 14 wks
18	?	?	3540	-	Spont.	SB	
19	?	?	3574	-	Induced	IUD	

DISCUSSION

The association of postmaturity in the elderly primigravida with perinatal loss is now well recognised by obstetricians and in this survey none of the three primigravid patients were more than three days past term (Table XV). A number of perinatal deaths, however, occurred in parous patients. In the group of women who had one or two children, 8 of the 12 deaths occurred at or before term and only 4 were postmature. These patients did not attend very early for antenatal care, 5 came before 20 weeks gestation, 4 between 20 and 30 weeks, 1 patient attended first at 34 weeks, and 2 received no care at all. This was even more marked in those with 3 or more children (Table XIX). Two patients first attended at 19 weeks, 11 patients between 20 and 30 weeks, 2 came later than 30 weeks, one received no care at all, and in 3 patients the gestation length when they first attended the clinic was not known.

Maternal weight gain in pregnancy is known to affect the ultimate weight of the baby. Unfortunately the weight gain of the patients was not recorded on a number of occasions but in 7 of the 19 patients having their 4th or subsequent child, the weight gain was noted as being unsatisfactory but no action was taken. As the weight gain was not recorded it was often not possible to determine the percentile baby weights using the tables devised by Thomson, Billewicz and Hytten (1968), standardising for maternal height, parity and sex of infant, as these factors are all known to have an effect on the birth weight of the child. In the 7 primigravid patients where percentile baby weight could be calculated, 4 were noted to be less than the 10th percentile, whereas 3 were large - greater than the 75th percentile (Table XV), illustrating the point that in the small baby the placenta has been unable to support normal growth, whereas with the large babies the normal resources of the placentae have been outstripped. There was not a clear division with the two groups in the parous patients.

In the primigravid patients 4 babies died before labour began, 3 during

labour and 5 during the postnatal period (3 from aspiration syndrome) (Table XV). In the patients with 1 or 2 children, all the babies were born dead, 11 before labour began and 1 during labour (Table XVIII). In the women of high parity, perinatal death occurred before labour in 10 cases, during labour in 6 cases, and in the neonatal period in 3 cases (Table XIX).

To improve the perinatal outcome, therefore, a change in obstetric management must take place before labour begins. Obstetricians must be aware of poor weight gain during pregnancy, that in women from poor social backgrounds and with high parity the placenta is very liable to fail before term. These points must also be made known to the general practitioners and Health Visitors, so that patients may be encouraged to attend the antenatal clinic early in their pregnancy. If the patient attends late in pregnancy and there was developed some degree of in-utero growth retardation, the patient's date of her last menstrual period is queried and it is never clear whether growth retardation exists or not. Vaginal examination to assess the size of the uterus, carried out before 12 weeks gestation, would eliminate this problem.

One is left with the impression that much closer antenatal supervision during the last 10 weeks of pregnancy is required for the highly parous patients; too often combined care appeared to be inadequate from both the hospital and the general practitioner's side.

Baird and Thomson (1969), writing in "Perinatal Problems," maintain that a very high standard of supervision, especially during labour, and the immediate availability of facilities for operative delivery are as important in the prevention of deaths in this group as they are in deaths from mechanical causes. The situation in Glasgow seems more severe in that it is before labour starts that the high standard of supervision is required and an early recourse to induction of labour after careful monitoring of fetal wellbeing throughout pregnancy might greatly reduce perinatal deaths from the category - mature, cause unknown.

Once the risk of perinatal loss from the cause - mature, cause unknown, is appreciated, much can be done to monitor the condition of the fetus during the antenatal period and during labour. During the antenatal period the measurement in maternal blood and urine of specific products of the feto-placental unit provides a valuable and widely applied means of assessing the fetal wellbeing. It has been well documented that the oestriol output in cases of intra-uterine growth retardation is well below normal levels and the use of serial assays is of great value to the obstetrician when he is deciding the best time for delivery (Klöpffer, 1969, in "Fetus and Placenta"). Lechworth and Chard (1972) have shown in a prospective survey of the use of placental lactogen that serial levels can be used to predict fetal distress and/or neonatal asphyxia in cases which show no other clinical abnormality and in which birth weight is normal. Elective early delivery can remove the fetus from an unfavourable intra-uterine environment before death occurs. With modern methods of induction, combining artificial rupture of membranes with intravenous Syntocinon (not buccal pitocin which is still used and which can cause irregular very strong contractions leading to uterine rupture, close attention to the state of the fetus during labour, by means of a continuous cardiotocography and scalp-blood sampling, permits the diagnosis of acute fetal distress. Labour when induced in this way should not last longer than 6 hours in the majority of patients.

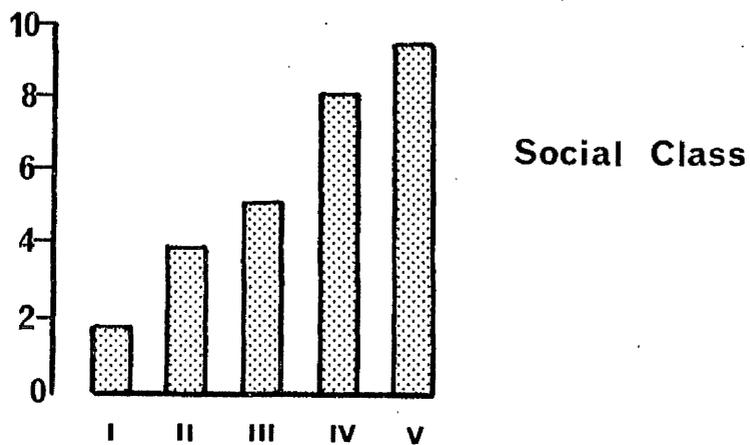
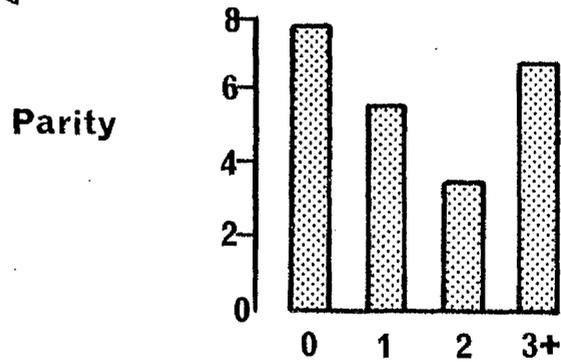
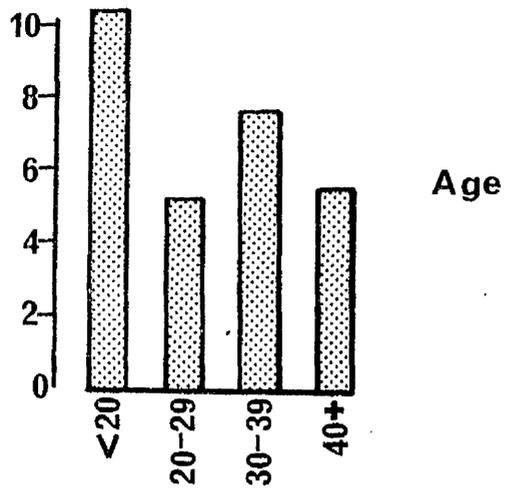
As a result of this study and concern about the number of mature deaths, an elective induction trial is being carried out at Glasgow Royal Maternity Hospital to determine if the morbidity of induction is greater than the morbidity of non-induction. Obstetrically normal patients who would normally be left until 41 weeks gestation are randomly selected for induction at 39+ weeks (i.e. artificial rupture of the membranes + intravenous Syntocinon or

spontaneous onset of labour if it occurs before 41+ weeks). If labour has not started by then and no indication for induction between 39 and 41 weeks occurs, induction is then carried out at 41+ weeks. The preliminary results of the first 141 patients studied are that the morbidity of induction is, at least, as low as that of non-induction and elective induction may reduce the incidence of meconium staining in labour, the Caesarean Section rate, the blood loss at delivery, but increase the amount of sedation required during labour.

A policy of early induction between 38 weeks and term for patients of high parity might therefore do much to reduce the perinatal loss in this group of mature babies.

FIG. 2.

Premature (cause unknown)



(b) PREMATURE, CAUSE UNKNOWN

Low birth weight infants are divided into two main groups, (1) pre-term infants, and (2) term infants who are light-for-dates. This division was decided upon at the Second European Congress of Perinatal Medicine held in London in 1970. It was felt that the previous definition by birth weight alone was not sufficient and that birth weight should be related to gestational age and that infants born before 37 completed weeks gestation should be called pre-term infants while those born after 37 weeks should be called term infants. Light-for-dates infants can also be defined as those weighing less than the 10th percentile (Thomson, Billewicz and Hytten, 1968).

Crosse, in her book "The Pre-Term Baby and other Babies with Low Birth Weight" (1971) states that "Low birth weight may be due to a curtailed pregnancy (pre-term infant), retarded growth in utero (light-for-dates infant) or a combination of both factors.

"Conditions which are associated with both curtailed pregnancy and retarded intra-uterine growth include -

- (1) Maternal complications during pregnancy.
- (2) Multiple pregnancy.
- (3) Congenital malformation.
- (4) Sex of the infant.
- (5) Biological factors.
- (6) Socio-economic conditions.
- (7) Smoking by mother.

Curtailed pregnancy may result from blood incompatibility, incompetent cervix, premature separation of the placenta, premature rupture of membranes, and

occasionally physical or emotional trauma. Spontaneous premature rupture of membranes plays a relatively important part in causing a curtailed pregnancy. Early rupture may be due to such causes as abnormal presentation, placenta praevia, multiple pregnancy or hydramnios; but in many cases the cause is unknown and there may be some abnormality of the membranes."

In this chapter the babies born weighing 2500 g. or less, where there is no obvious clinical cause for the premature delivery or impaired intra-uterine growth, are being studied in detail. For this present analysis twin pregnancies and those babies born at home have been excluded.

As more than 20 per cent of the perinatal deaths in Glasgow in 1970 occurred in this group, premature, cause unknown, any improvement in the perinatal mortality rate from this cause is of considerable importance. One important aspect which was necessary to determine when studying this group of perinatal deaths, was to discover whether the mothers of these babies came for and received adequate antenatal care, or whether they did not seek medical help until labour had started.

FINDINGS

During 1970, in the City of Glasgow, there were 101 perinatal deaths in the premature cause unknown group. The premature mortality rates by age, parity and social class are illustrated in Fig. 2. High rates were found in primigravidae and women having their 4th or subsequent child. There was a steady rise in perinatal mortality rate from social class I to social class V. Seventy-six perinatal deaths classified as premature, cause unknown, occurred in single pregnancies. As 8 of these cases occurred at home and have been discussed in the chapter on Domiciliary Deliveries (Chapter IX), the remaining 68 cases will be discussed in detail here.

Table XX shows that there were many more pre-term babies in this group of deaths than light-for-dates term babies.

TABLE XX.

Details of Small Babies

Pre-term	53
Light-for-Dates	11
Not Stated	4

In four patients the gestation length was not known. They received no antenatal care and did not reach hospital until well in labour. Details of these 4 patients are given in Table XXI.

TABLE XXI.

Details of four patients where gestation length was not known.

Age.	Parity.	Social Class.	Antenatal Care.	Baby Weight.	Outcome.
21	0 ⁺⁰	II	Nil	1090	MND
34	3 ⁺³	IV	Nil	964	IUD
36	3 ⁺⁰	IV	Nil	559	MND
36	3 ⁺¹	V	Nil	1920	IUD

Pre-Term: Table XXII illustrates that 18 of these patients received no antenatal care, and of these that did, half presented themselves to the clinic after 20 weeks gestation.

TABLE XXII.

Pre-term Infants (53). Gestation Length at first antenatal visit.

< 10 weeks	3
10 - 19 weeks	15
20 - 29 weeks	15
30 weeks and over	2
Nil	18

The age, parity and social class distribution of the mothers who received no care is shown in Table XXIII, and the gestation length at the time of delivery is shown in Table XXIV.

TABLE XXIII

Characteristics of Mothers with no antenatal care.

Age	No.	Parity.	No.	Social Class	No.	Marital Status
< 20 years	5	0	4	I	1	Married 16
20-29 years	10	1	7	II	0	Single 2
30-39 years	3	2	4	III	7	
40 and over	0	3+	3	IV	1	
				V	7	
				N/S	2	

TABLE XXIV

Gestation Length at Delivery

< 28 weeks	8
28 - 32 weeks	8
32 - 38 weeks	2

Sixteen of the 16 babies were born before 32 weeks gestation, so unless these mothers had some distinguishing feature, it is unlikely that antenatal care would have greatly affected the perinatal outcome. In this group there were 4 stillbirths (only in one very small baby was the fetal heart heard on admission to hospital) (Table XXV) and 14 neonatal deaths.

TABLE XXV.

Details of Unbooked Patients (18)-Babies - pre-term.

Maternal Age.	Parity.	Social Class.	Baby Weight.	Outcome.
<u>Gestation 28 weeks.</u>				
1. 28	1+4	V	319	NND
2. 17*	0+0	V	496	NND
3. 24	2+0	V	595	NND
4. 18*	0+0	V	650	NND
5. 31	5+1	?	790	NND
6. 37	1+5	V	907	NND
7. 25	2+1	I	798	NND
8. 27	2+1	III	1010	NND
<u>Gestation 28 - 32 weeks</u>				
1. 26	4+0	III	810	SB SRM. Labour 5 days later.
2. 17*	0+0	V	964	NND
3. 26	1+0	?	1080	NND
4. 27	1+0	IV	1171	NND
5. 17*	0+1	III	1240	SB Obstructed Breech.
6. 22	1+0	V	1304	IUD
7. 19*	1+0	III	1361	NND
8. 24	2+0	III	1420	NND
<u>Gestation 32 weeks</u>				
1. 26	3+0	III	1077	NND
<u>Gestation 37 weeks</u>				
1. 31	1+0	III	1503	IUD

* = Single or Prenuptial Conception.

Table XXVI shows the characteristics of the mothers who received antenatal care.

TABLE XXVI

Characteristics of Mothers who received antenatal care (35)

Age	No.	Parity	No.	Social Class.	No.	Marital Status
< 20 years.	9	0	23)	I	0	Married
20 - 29 "	16	1	4) had	II	2	Single
30 - 39 "	10	2	1) prev-	III	21	
40 & over	0	3+	7) sious	IV	8	
			history	V	3	
			of small	N/S	1	
			baby			

An interesting finding here is that 23 of the 35 mothers were primigravidae, whereas only 4 of the 18 mothers who received no care were. In this group also, of the 12 women who had had previous pregnancies, 7 gave a history of having delivered a baby of 2500 g. or less, whereas only two of the 14 parous patients in the group who received no care had had a previous small baby. There were 16 stillbirths and 19 neonatal deaths in the women who received antenatal care. The gestation lengths of the stillborn babies were very different (being longer) than the babies who were born during the neonatal period (Table XXVII). The maternal characteristics of these two groups were compared (Table XXVIII). They were found to be surprisingly similar.

TABLE XXVII.

Gestation Length at Delivery

	Stillbirths.	Neonatal Deaths
< 28 wks.	0	12
28-29 weeks	1*	1
30-31 weeks	1	3
32-33 weeks	3	2
34-35 weeks	7	1
36-37 weeks	4	0

* F.H.H. on admission.

TABLE XXVIII.

Maternal Characteristics of Mothers whose babies had (A) stillbirth
(B) neonatal death.

Age	A.	B.	parity.	A.	B.	Social Class	A.	B.
< 20 years	4	5	0	11	12	I	0	0
20 - 29	8	8	1	1	3	II	2	0
30 - 39	4	6	2	1	0	III	9	12
40 & over		0	3+	3	4	IV	3	5
						V	1	2

Light-for-dates:

None of the mothers of the light-for-dates infants were under 20 years of age, and there was a higher percentage of highly parous women than in the mothers of pre-term infants (Table XXIX).

TABLE XXIX.

Light-for-Dates Infants (11).
Maternal Characteristics.

Age.	No.	Parity.	No.	Social Class.	No.	Marital Status
< 20 years	0	0	2	I	0	Married 11
20 - 29 years	6	1	3	II	1	
30 - 39 years	4	2	1	III	4	
40 and over	1	3+	5	IV	3	
				V	3	

There was no definite pattern in the mothers' attendance for antenatal care. Some mothers came early, some came late and some did not come at all. (Table XXX).

TABLE XXX

Gestation Length at first
Antenatal Visit

< 10 weeks	0
- 20 weeks	3
- 30 weeks	3
30 weeks and over	3
Nil	2

Table XXXI shows the details of the babies. The two striking findings are (a) intra-uterine death occurred in 7 cases, and (b) in the two primiparous patients, weight gain had been unsatisfactory during the last trimester, one patient gained no weight during the last 7 weeks and the other gaining only 4 pounds in the last 10 weeks. Both patients were, in fact, admitted for induction of labour at term.

TABLE XXXI.

Details of Infants.

No.	Gestation Length at Delivery.	Outcome.	Weight.	Comment.
1.	38 weeks	IUD	992	Previous small baby
2.	38 weeks	IUD	1389	
3.	39 weeks	IUD	1560	
4.	39 weeks	IUD	?	
5.	40 weeks	SB	2410	Prim. 4 lb. weight gain in last 10 weeks.
6.	40 weeks	SB	2098	Previous small baby.
7.	40 weeks	IUD	2325	
8.	40 weeks	NND	2098	Prim. No weight gain for last 7 weeks.
9.	41 weeks	IUD	2490	Previous small baby.
10.	45 weeks	IUD	2096	
11.	46 weeks	NND	1921	Previous small baby.

There was no striking finding during the antenatal period to differentiate these patients apart from the fact that of the 54 pre-term labour, spontaneous premature rupture of membranes occurred in 12 patients.

DISCUSSION

As the physiology of labour is not fully understood it is not surprising that the trigger mechanism which gets a woman into premature labour, for no obvious reason, remains unknown also. Recent work in Oxford by Turnbull et al. (1974) suggests that the onset of labour in human pregnancy occurs after the withdrawal of an inhibitory effect of progesterone on the myometrium and at the time of active oestrogen dominance. A Leader in the Lancet (1974) in the same issue as Turnbull et al.'s paper is published, states that "a note of caution should perhaps be sounded. Experience shows that the apparently conclusive results of one group of workers are often overturned by that of another group. The investigation should be repeated and confirmed. In view of the fundamental clinical importance of the subject, this would not be a waste of scarce resources."

In a subsequent paper by Raja et al. (1974) describing endocrine changes in premature labour, they suggest that the onset of premature labour is preceded by a marked increase in peripheral plasma oestradiol levels which may be of value not only in the prediction of premature labour, but also in its prevention by the suppression of the premature oestradiol surge. They make the valuable comment that if high perinatal mortality and morbidity now occurring in infants of low birth weight is to be reduced, one of the most vital needs is to find a means of predicting and preventing premature labour of spontaneous unexplained onset. If Raja et al.'s (1974) findings are correct, some means must be obtained whereby the women at risk can be identified early in their pregnancy and steps taken to prevent the premature onset of labour. At our present stage of knowledge, this simply means admission to hospital for rest and observation, and immediate action to control uterine activity should premature labour begin. The two main approaches used to control premature labour meantime are the B-adrenergic stimulant Ritodrine being most promising (Wesselius de Casparis et al. 1971) and intravenous alcohol (Tuchs et al. 1967). In the study with Ritodrine, which was carried out as a double blind multicentre trial, comparing Ritodrine and a placebo in 66 patients with intact membranes in premature labour, labour was postponed for 7 days or more in 78 per cent of those receiving Ritodrine compared with 55 per cent of those on placebo. In the ethanol trial, labour was postponed in significantly more patients with ethanol than with placebo.

It appears that the problem is to identify the patient at risk but unfortunately this study makes very little contribution to the solution.

Thirty-four per cent of the mothers with pre-term infants who died, did not receive any antenatal care, and of these that did half did not attend until the second half of their pregnancy. It is debatable whether the perinatal outcome would have been different if they had attended the antenatal clinic. Butler and Alberman (1969) state that "prematurity of unexplained origin is very difficult to prevent by measures available to the obstetrician. Most experienced clinicians would probably agree that the effect of rising standards of antenatal care is

disappointingly small. The best hope probably lies in improving the environment and thereby the standard of physique and general health among mothers."

This belief is not entirely held, however, by the World Health Organisation. The W.H.O. Expert Committee (1961) made the excellent suggestion that health education and prenatal care might sometimes have to be brought to the patient (rather than the other way round) because the patients most in need of treatment and advice are least likely to seek it. This latter fact is borne out in the findings of the domiciliary, premature - cause unknown deaths, where the major factors appeared to be social problems leading to lack of any care (Chapter 9).

The picture is very different in the light-for-dates infants. No cases occurred in mothers under 20 years of age; only 2 of the 11 occurred in primigravidae whereas 5 occurred in the highly parous women. Particularly important in the primiparous patients was failure to gain weight. Information of the parous patients' weight gain was not available, possibly because their antenatal care was shared between hospital and general practitioner. Weight in pregnancy is discussed in greater detail in Chapter 6, but it is obvious that more information is required about maternal weight gain towards the end of pregnancy. Attention to weight gain in association with other methods of assessing fetal wellbeing, e.g. oestriol estimation, biparietal diameter measurements using ultrasound, etc. ought to improve the perinatal outcome of these growth retarded babies.

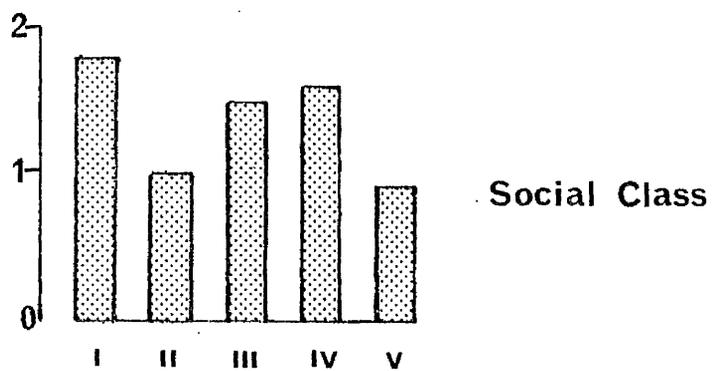
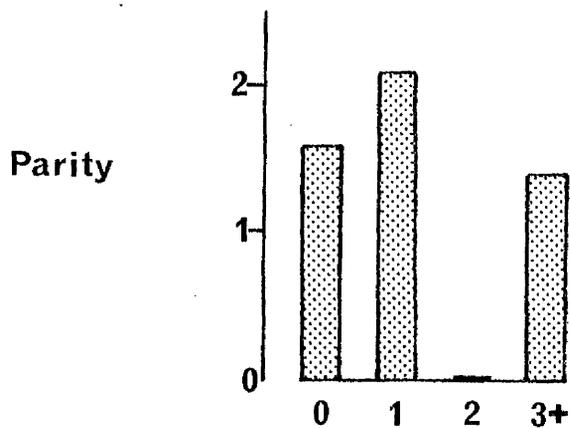
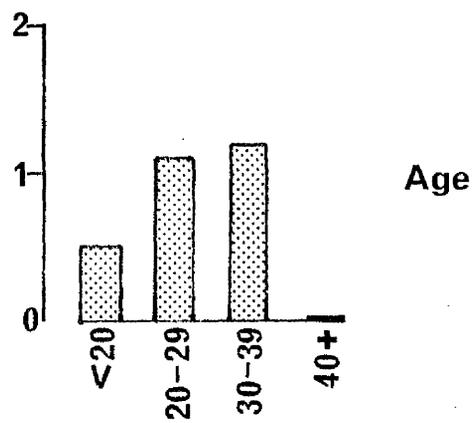
One finding that was common to both groups of small babies was the history of a previous small baby (13 of the 35 parous patients, i.e. 37 per cent). The accepted incidence of a low birth weight baby is 8 per cent. It would therefore be of interest to determine the perinatal outcome of intensive antenatal care in a group of women with a previous low birth weight baby.

The results are disappointing in that they fail to pinpoint the women at risk. What is now required is a more detailed study of all women with pre-term

babies in the Glasgow region to determine whether other characteristics come to light, and to follow up Raja et al.'s (1974) findings so that once the patients at risk have been identified, serial blood determinations can be carried out to see if the premature oestradiol surge is detected and attempts can then be made to suppress it.

FIG. 3.

Trauma



(c) TRAUMA

Perinatal deaths due to mechanical causes (birth trauma) can be divided into three main groups -

- (1) breech delivery,
- (2) cord complications,
- (3) other traumatic deliveries.

Good obstetric care during the antenatal period and during labour contributes greatly to low perinatal mortality rates in this group. Baird (1969), writing about perinatal deaths, points out that the Registrar General's stillbirth rate for Scotland for 1958 and 1966 shows a marked drop from 6.2 per thousand to 1.1 per thousand in deaths associated with "difficult labour and birth injuries." This reduction occurred in each of the three types of difficult labour mentioned above. It would therefore be expected that in 1970 there would not be many deaths due to traumatic delivery in Glasgow.

FINDINGS

The perinatal mortality rates in the trauma group by age, parity and social class are shown in Fig. 3.

Twenty-three deaths were classified as being due to traumatic delivery. Three of these deaths occurred where the mother received no antenatal care, and one where the patient only attended hospital once. Three patients were delivered at home and the details are given in the chapter on Domiciliary Confinements. The fourth patient was an unmarried primigravida aged 37 years. She was admitted in labour and the fetal heart was noted to be irregular, and the liquor was stained with meconium. One sample of fetal scalp blood was analysed and found to be satisfactory. As she was single, the decision against Caesarean Section was taken. After a labour lasting more than 24 hours, a limp baby weighing 2835 g. Apgar 1 at 1 minute, was delivered, which died during the first day. Postmortem

examination showed intracranial haemorrhage, a right-sided tentorial tear, atelectasis and bilateral pneumothorax. This death was surely preventable.

The other 19 deaths fell into three categories -

- (1) Breech deliveries 5
- (2) Cord complications 8
- (3) Other traumatic deliveries 6

(1) Breech Deliveries:

Table XXXII illustrates the maternal characteristics, and Table XXXIII gives the details of each delivery.

TABLE XXXII
Breech Deliveries - Maternal Characteristics

Age	No.	Parity	No.	Social Class	No.	Height	No.
< 20 years	0	0	2	I	0	0 - 5'1"	3
20-29 years	5	1	3	II	1	5'2" - 5'4"	0
30-39 years	0	2	0	III	3	5'4"+	2
		3+	0	IV	0		
				V	0		
				N/S	1		

TABLE XXXIII
Details of Breech Deliveries

No.	Gestn. Length.	Parity.	Height.	Birth Weight.	Labour.	Outcome.	Comment.
1.	35	1+0	4'11"	2155 g.	Spont. Labour 12 hrs.	S.B.	APH at 27 weeks. Admitted. Labour 35 weeks. Breech stuck; 2nd stage 2 hrs 15 mins. Prev. LUSCS. Perforation of head.
2.	38	1+0	5'7"	3560 g.	Induced Labour 12 hrs.	S.B.	Precipitate labour. Uncomplicated delivery. P.M. Subarachnoid haem. Bilateral tentorial tears.
3.	39	6+0	5'0"	2608 g.	Spont. Labour 12 hrs.	NND	2nd stage 1 hr 14 min. Delivery relatively easy. P.M. Subarachnoid haemorrhage.
4.	39	0+0	5'0"	2948 g.	Labour 12 hrs.	NND	Not difficult delivery. Cord X3 round neck. P.M. Tentorial tear.
5.	39	1+0	5'7"	4020 g.	Induced Labour 12 hrs.	NND	Great difficulty with delivery of aftercoming head. Three doctors attempted it.

Earlier recourse to Caesarean Section might have greatly altered the perinatal outcome. The five patients did not have long labours, but Cases 1 and 3 had prolonged second stages, and even at that point Caesarean Section should have been carried out.

Three of the women were small so that complications with breech delivery should have been anticipated, and the two tall women had good sized babies, so again complications with delivery should have been expected and Caesarean Section carried out.

To improve perinatal mortality rates in breech deliveries, close supervision of the patient must be carried out during labour and the obstetrician must be prepared to carry out a Caesarean Section as soon as any sign of fetal distress or obstruction occurs.

(2) Cord Complications:

Table XXXIV shows the maternal characteristics.

TABLE XXXIV.

Maternal Characteristics

Age.	No.	Parity.	No.	Social Class.	No.	Height.	No.
< 20 years	0	0	3	I	0	0 - 5'1"	4
20-29 years	6	1	2	II	0	5'2"-5'4"	4
30-39 years	2	2	0	III	6	5'4"+	0
40 and over	0	3+	3	IV	1		
				V	1		
				N/S	0		

The details of the deliveries with cord complications are shown in Table XXXV.

TABLE XXXV.
Details of Deliveries with Cord Complications

No.	Gestn. Length.	Birth Weight.	Labour.	Method of Delivery	Outcome.	Comment.
1.	38	4840	Spont.	Assisted Vertex	S.B.	Difficulty with shoulders. Cord very tightly round neck.
2.	39	2203	Induced	Forceps	S.B.	Prev. growth retarded. S.B. Very carefully monitored throughout pregnancy. Fetal heart stopped in 2nd stage. Cord X 3 very tightly round neck.
3.	39	2750	Induced	Forceps	S.B.	Cord X 2 tightly round neck.
4.	39	2977	Induced	External Version & Breech Extr.	S.B.	Induced because of mild P.E.T. Head displaced 8 hrs. later. No fetal heart heard. Cord prolapse. Transverse lie.
5.	40	2930	Spont.	Classical Section	S.B.	Fetal distress. Cord very tightly round neck.
6.	40	3515	Spont.	Forceps	NND	Prolapsed cord. P.M. Necrosis of nerve cells in mid brain and pons.
7.	40	3714	Spont.	Spont. Vertex	S.B.	Cord presentation. Fetal heart not heard on admission, therefore LUSCS not carried out.
8.	?	2580	Induced	Spont. Vertex	S.B.	Fetal movements not felt for a few days before delivery. Cord X 2 very tightly round neck and under left arm.

In Case 4 the fact that the patient had had a surgical induction of labour contributed to the perinatal death. However, in this case only the membranes were ruptured and Syntocinon was not given intravenously at the same time. The management of this case was, therefore, not ideal.

In five of the remaining cases the cord was very tightly round the neck, leading to death of the baby.

(3) Other Traumatic Deliveries:

Table XXXVI shows the maternal characteristics in this group.

TABLE XXXVI.
Maternal Characteristics.

Age.	No.	Parity.	No.	Social Class.	No.	Height.	No.
< 20 yrs.	1	0	2	I	1	0 - 5'1"	3
20-29 years	3	1	3	II	0	5'2"-5'4"	2
30-39 years	2	2	0	III	2	5'4"+	0
		3+	1	IV	2	W/S	1
				V	1		

The details of these deliveries are shown in Table XXXVII.

TABLE XXXVII.
Details of other Traumatic Deliveries

No.	Gestn. Length.	Birth Weight.	Labour.	Method of Delivery.	Outcome.	Comment.
1.	34	2126	Spont.	Breech Extraction	S.B.	Previous LUSCS. Admitted in labour with uterine infection. Shoulder presentation. Internal version. Ruptured uterus.
2.	36	3230	Induced	Forceps	MND	2nd stage 2½ hours. Failed Kiellands - Milne Murrays - del. with "more than average traction." Caput ++.
3.	39	1960	Spont.	LUSCS	S.B.	Obstructed labour. Transverse lie.
4.	40	3260	Induced	Forceps	S.B.	2nd stage = 2 hrs. Attempted Kiellands - failed. Easy forceps under G.A.
5.	41	4026	Induced	LUSCS	S.B.	Previous LUSCS ?T-shaped incision. ARM - "bloody tap." 6 hours labour - pain - ruptured uterus.
6.	7	3062	Induced	Classical Section	S.B.	Booked for home confinement. ARM by G.P. Admitted following day - not in labour. Transverse lie with prolapsed arm. Pus draining.

It would appear that these traumatic deaths could have been prevented with improved obstetric care. In Case 1, despite the presence of infection, Caesarean Section ought to have been carried out. The neonatal death in

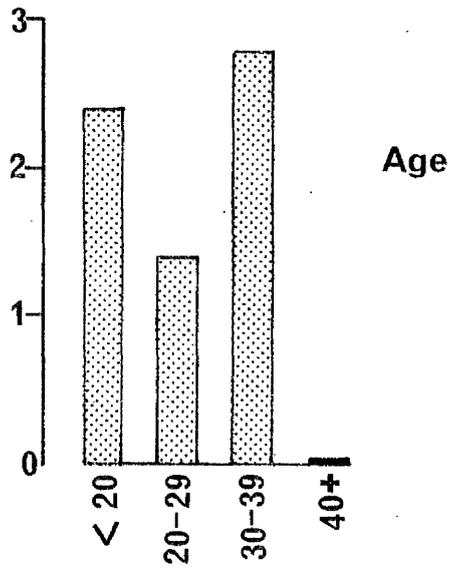
Case 2 would have been prevented by Caesarean Section carried out in the second stage. The baby in Case 3 was growth retarded, weighing less than the 5th percentile. This fact ought to have been detected during the antenatal period. In Case 4, the second stage should have been interrupted and Caesarean Section carried out, thus preventing the perinatal loss, as the fetal heart stopped during the last hour. In Case 5, it was thought at section that the previous section had been carried out by a T-shaped incision. If this had been known prior to labour, elective Caesarean Section would have been carried out.

As there is no place for artificial rupture of the membranes to start off labour in domiciliary midwifery, patient 6 should have been admitted to hospital for this procedure to be carried out, and to be carefully monitored during labour.

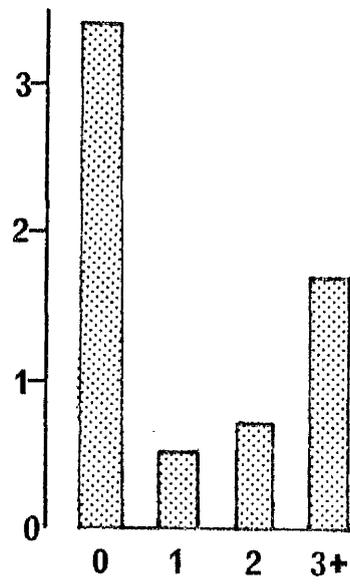
In all these cases, therefore, death should not have occurred, and the kind of obstetrics practised in these patients is no longer acceptable by modern standards.

FIG. 4.

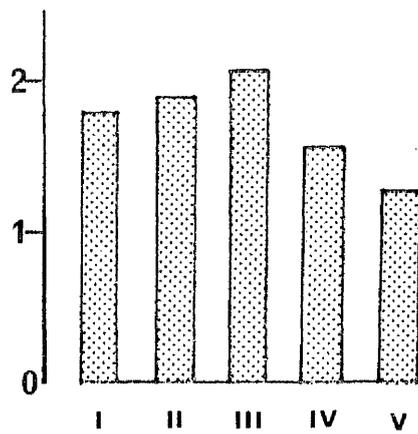
Toxaemia



Parity



Social Class



(d) TOXAEMIA

Pre-eclampsia or pregnancy toxæmia is a condition in which blood pressure rises after the 24th week of pregnancy to 90 mm. Hg. diastolic or more, recorded on at least two separate days; the differentiation into severe and mild forms depends on the presence or absence of albuminuria.

Although the aetiology of pre-eclampsia remains obscure, it is well recognised that it is a condition of first pregnancies (MacGillivray, 1961); that there is a greater incidence of the disease in women with a high weight gain between 20 and 30 weeks gestation (MacGillivray, 1967), and that if the condition is severe the babies are light-for-dates (Baird et al. 1957).

There is no doubt that the incidence of pre-eclampsia is falling throughout the world. Whether this is due to better maternal health, improved standards of antenatal care, or some unknown factor, is not understood. There are still a number of perinatal deaths, however, due to pre-eclampsia, and there is a greater degree of maternal and infant morbidity consequently.

FINDINGS

During 1970 in the City of Glasgow there were 29 perinatal deaths classified as being due to toxæmia. The perinatal mortality rates by age, parity and social class are illustrated in Fig. 4, showing high rates in the women under 20 years and those in their thirties. The rate is high in primigravid patients and begins to increase again in those having their fourth or subsequent child. There is little social class variation, the highest rate occurring in Social Class III.

There were 19 primiparous patients, one of whom had eclampsia at 29 weeks gestation, and 10 multiparous patients (5 of whom gave a previous

history of toxæmia). Fourteen of the primiparous and 5 of the multiparous patients had albuminuria as well as hypertension.

Tables XXXVIII to XLI deal with the findings in the primiparous patients.

TABLE XXXVIII
Characteristics of Primiparae (19)

Age	No.	Social Class	No.	Marital Status
< 20 years	3	I	1	Married 15
20 - 29 years	13	II	0	Single 4
30 - 39 years	3	III	12	
40 and over	0	IV	4	
		V	2	

TABLE XXXIX.
Gestation Length at first Antenatal Visit

< 10 weeks	4
10 - 19 weeks	9
20 - 29 weeks	3
Unknown	3

TABLE XL
Gestation Length at Delivery

< 28 weeks	1)	
28-29 weeks	4)	8
30-31 weeks	3)	
32-33 weeks	0)	
34-35 weeks	2)	
36-37 weeks	5)	10
38-39 weeks	1)	
40-41 weeks	2)	
Not stated	1	

Table XXXVIII shows some of the maternal characteristics. The majority of the women were in their twenties although three were still in

their teens and three were in their thirties (elderly primigravidae).

Twelve of the patients were from Social Class III. Details about antenatal care were known for 16 patients, 13 of whom were seen for the first time before 20 weeks gestation (Table XXXIX).

Eight babies were delivered before 34 weeks gestation, and 10 babies were delivered at 34 weeks gestation or later (Table XL).

It was thought reasonable that babies from 34 weeks ought to have had a chance of survival. Details of the babies born at 34 weeks gestation or later are shown in Table XLI.

TABLE XLI.

Primiparous Patients: Details of Babies born at 34 weeks gestation or later (10).

No.	Gestn. Length.	Birth Weight.	Outcome.	Comment.
1.	34	1021	S.B.*	Shared antenatal care. Seen at hospital at 22 weeks and asked to return at 34.
2.	35	2120	S.B.*	Induced because of P.E.T. F.H. 100/min. for 2 hours before it stopped.
3.	36	2325	S.B.	Induced. No sign of fetal distress until F.H. stopped during 2nd stage.
4.	37	1900	IUD	To be induced because of falling oestriols but IUD occurred.
5.	37	2325	IUD*	Mild hypertension. Weight gain poor. No monitoring.
6.	38	3280	NND*	Long labour -- LUSCS. Baby good condition at birth. Aspiration pneumonia.
7.	37	1670	NND	Induced because of P.E.T. Meconium, therefore LUSCS. Baby developed fulminating infection post-natally.
8.	37	1640	IUD	Oestriol excretion satisfactory.
9.	40	3527	NND	Mild hypertension throughout pregnancy. Baby limp at birth.
10.	40	3487	NND	LUSCS - Cause of death atelectasis ? intra-uterine pneumonia.

* = Death preventable.

It is easy to be wise after the event, but it would appear that in Cases 1, 2, 5 and 8 the perinatal deaths ought to have been avoidable, either

by better care during the antenatal period, or more vigorous action during labour, and a case could be made for all ten deaths being prevented.

Tables XLII to XLV deal with findings in the parous patients.

There was very little difference between the two groups of parous patients, i.e. those who had a previous history of toxæmia and those who had not, with regard to their age, parity, social class, marital status and gestation length at the first antenatal visit. (Tables XLII and XLIII). The gestation lengths of the two groups are dissimilar. It is of interest to note that all the women who had no history of pre-eclampsia had intra-uterine deaths.

TABLE XLII.

Parous Patients - Characteristics. A - No previous history of toxæmia 5
 B - Previous history of toxæmia 5

<u>Age</u>	<u>No.</u>		<u>Parity.</u>	<u>No.</u>		<u>Social Class</u>	<u>No.</u>		<u>Marital Status</u>		
	A.	B.		A.	B.		A.	B.	A.	B.	
< 20 wks.	0	0	1	1	2	II	0	2	Married	5	5
20-29 weeks	1	2	2	1	0	III	4	2	Single	0	0
30-39 weeks	4	3	3+	3	3	IV	0	1			
						V	1	0			

TABLE XLIII

Gestation Length at first Antenatal Visit

	<u>A.</u>	<u>B.</u>
< 10 weeks	0	0
10 - 19 weeks	2	3
20 - 29 weeks	2	2
Not Stated	1	0

TABLE XLIV

Gestation Length at Delivery

	<u>A.</u>	<u>B.</u>
< 28 weeks	1	0
28 - 29 weeks	0	0
30 - 31 weeks	0	1
32 - 33 weeks	0	1
34 - 35 weeks	2	0
36 - 37 weeks	0	1 - NND
38 - 39 weeks	2	0
40 and over	0	2 - NNDS.

Group A - All IUDs
Group B - 3 NNDS

Table XLV gives some of the deaths of the babies born to multiparous patients at 34 weeks gestation and later. It could be argued that all of these deaths could have been prevented if the patients had come for and received closer antenatal supervision.

TABLE XLV.

Multiparous Patients - Details of babies born at 34 weeks gestation or later (7)

<u>No.</u>	<u>Gestn. Length.</u>	<u>Birth Weight.</u>	<u>Outcome.</u>	<u>Comment.</u>
1.	34	1389	SB	Multipara. Threatened abortion. One antenatal visit. Admitted with APH.
2.	35	2260	IUD	Severe P.E.T. Not admitted. Admitted with Abruptio.
3.	38	1559	IUD	Multipara. First seen 22 wks. No further appointment. Admitted with severe P.E.T.
4.	38	1673	SB	Mild H.B.P. Proteinuria. Elective Caesarean Section. Previous C.S. Dysmature baby. No fetal monitoring.
5.	37	2140	NND	Severe H.B.P. Refused admission. Admitted in labour. Doubtful F.H. Apgar 2. Cause of Death: Atelectasis.
6.	40	3230	NND	H.B.P. Induced. Uneventful labour. Infant collapsed. ?R.D.S.
7.	42	4092	NND	Uneventful labour. Apgar 8. pH 7.17 2 hours later. Cause of death - pulmonary oedema, Atelectasis.

Baby 5 collapsed after delivery for no obvious cause, but the mother had been treated with large doses of the anticonvulsant drug, Chloromethiazole, by intravenous infusion during labour. This drug has been shown to be of value in the management of pre-eclampsia and eclampsia but it is known that too large a volume given too rapidly just before delivery does affect the baby, as it crosses the placental barrier (Duffus et al., 1968).

It is interesting to note that the weight/height ratio was known for 24 of the 29 patients with toxæmia. Only one patient was light for her height, 7 had a normal weight/height ratio, and 16 patients were obese.

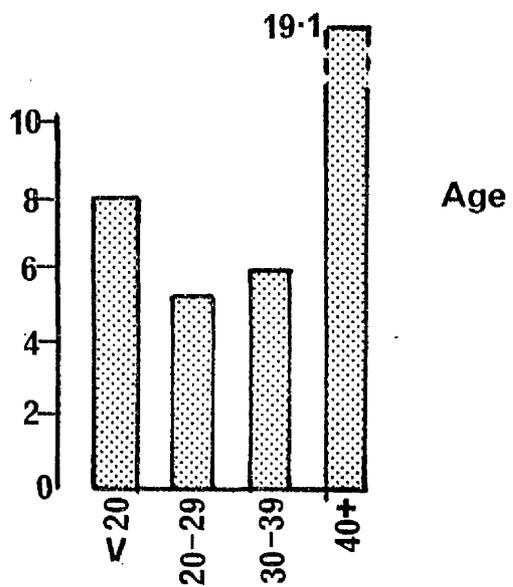
DISCUSSION

As the aetiology of pre-eclampsia remains unknown, the disease cannot be prevented. Good antenatal care is therefore essential so that the first signs of the condition can be recognised and the patient admitted to hospital for rest and assessment, because both the mother and the fetus are at risk. The mothers are at risk from eclampsia and severe hypertension leading to a cerebral vascular accident, and the baby from impaired placental function, hence the large number of intra-uterine deaths. In this group of perinatal deaths all the patients whose details were known (26 out of 29 deaths) received antenatal care, although 7 patients were first seen after 20 weeks. When the condition occurs in a fulminating form before 32 weeks gestation, the prognosis for the baby is not good. Terminations of the pregnancy at this stage will produce a baby that is usually too small to survive, yet continuation of the pregnancy will very likely lead to intra-uterine death.

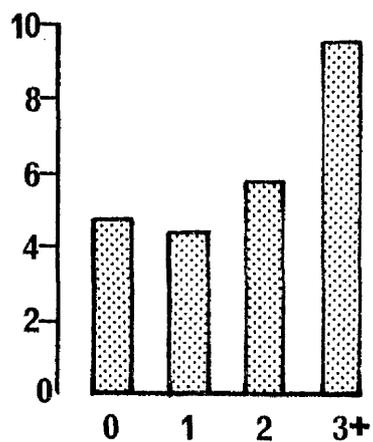
The use of anti-hypertensive drugs is of little value, if any, in such cases, since intra-uterine death can occur even when the blood pressure is well controlled. Similarly, the use of diuretics to control oedema does not halt

the toxæmic process and can be dangerous to the mother (Gray et al. 1968). In this group of deaths, 17 of these babies were born after 33 weeks gestation, and more intensive antenatal care might have improved the outcome. This opinion is shared by MacGillivray and Davey (1963) who state in their paper on "Hypertension in Late Pregnancy," that "even in the absence of a known cause for pre-eclampsia, the mortality among babies and mothers can be kept at a minimum, but only if a constant and careful watch is maintained on all pregnant women, and hospitals with sufficient beds and skilled staffs are available to treat all women in whom early warning signs appear."

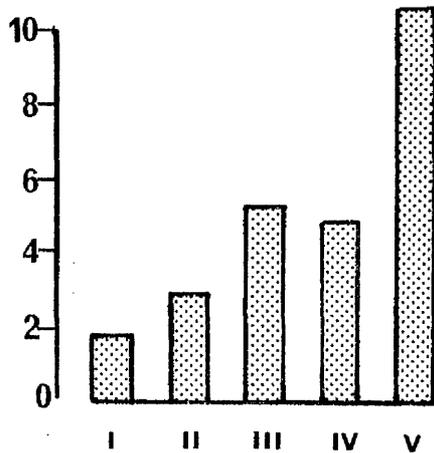
APH



Parity



Social Class



(c) ANTEPARTUM HAEMORRHAGE:

Antepartum haemorrhage is an important cause of perinatal loss and is also associated with considerable risk to the mother. Previous studies have shown that the incidence of antepartum haemorrhage varies from 1:23 (Murdoch and Foulkes, 1952) and 5.6 per cent (Fish et al. 1951). In a study of all births in a community over a 10-year period 1949 to 1958, Paintin (1962) found that the incidence of antepartum haemorrhage was 3.0 per cent, and the total mortality attributable to antepartum haemorrhage was 5.5 per thousand live and stillbirths.

METHOD

For this study antepartum haemorrhage was defined as blood loss from the vaginal orifice occurring after the end of the 24th week of gestation, but before the delivery of the infant. The haemorrhage was classified as belonging to one of three groups -

(1) Placenta praevia, which was diagnosed when the placenta had been seen to be implanted in the lower uterine segment at Caesarean Section, or felt there on vaginal examination.

(2) Placental abruption (accidental haemorrhage), which was said to have occurred where a retroplacental clot had been demonstrated, or when the patient had had a tense tender uterus associated with antepartum haemorrhage.

(3) Other - all cases of antepartum haemorrhage which did not fall into the above categories were classified as "Other."

FINDINGS

During 1970 in the City of Glasgow there were 94 perinatal deaths because of antepartum haemorrhage, giving a perinatal mortality rate of 5.6 per thousand live and stillbirths. The maternal characteristics are shown in Fig. 5, illustrating the high perinatal loss in those over 40 years of age,

of high parity and of Social Class V. Three deaths occurred in twin pregnancies and one in domiciliary practice. The remaining 90 perinatal deaths are discussed in detail below.

The distribution of the deaths is shown in Table XLVI.

TABLE XLVI

Distribution of Deaths - Antepartum Haemorrhage.

Type.	Number.	Percentage
Placenta Praevia	8	8.9
Abruptio Placentae	62	68.9
Other	20	22.2

Table XLVII gives details of the maternal characteristics by age, parity, social class and previous history. All the patients were married. The details of the babies and method of delivery are shown in Table XLVIII.

TABLE XLVII.

Placenta Praevia - Maternal Characteristics (8 patients)

Age.	No.	Parity.	No.	Social Class.	No.	Previous History.
< 20 yrs.	1	0	1	I	0	APH 1
20-29 yrs.	3	1	4	II	1	Low birth weight 2
30-39 yrs.	3	2	0	III	3	
40 & over	1	3+	3	IV	0	
				V	3	
				N/S	1	

TABLE XLVIII.
Details of Delivery (8 patients)

No.	Gestn. Length.	Birth Weight.	Method of Delivery	Outcome.	Comment.
1.	27	1410	Breech Extraction	S.B.	Fetal heart stopped in 2nd stage. Really an abortion (if gestation length accurate), but registered as a stillbirth.
2.	29	580	Breech	S.B.	Four previous abortions. In hospital for 21 weeks.
3.	32	1670	LUSCS	NND	Type II diagnosed by ultrasound. Allowed home - admitted following day with APH.
4.	33	1840	LUSCS	NND.	
5.	33	3100	Classical Section	S.B.	
6.	36	1890	LUSCS	S.B.	
7.	38	1176	LUSCS	NND	
8.	?	2835	Forceps	S.B.	No antenatal care. Admitted shocked. Resuscitated - fully dilated - forceps.

Case 3 should not have been allowed to go home once the suggestion of placenta praevia had been made. The perinatal outcome might have been quite different if the haemorrhage had occurred in hospital and Caesarean Section could have been undertaken quite quickly. The lack of antenatal care in Case 8 prevented the possibility of placenta praevia being diagnosed before the haemorrhage occurred.

Details of the "Other" group are given in Tables XLIX, L, and LI.

TABLE XLIX.
Maternal Characteristics (20 patients)

Age	No.	Parity	No.	Social Class	No.	Marital Status.	Previous Obstetric History
< 20 yrs.	4	0	8	I	1		APH 1
20-29 years	11	1	4	II	0	Married 16	Low Birth Weight 3
30-39 years	4	2	3	III	7	Single 3	
40 & over	1	3+	5	IV	5	Other 1	
				V	5		
				N/S	2		

In 5 of the 20 babies the fetal heart was not heard on admission to hospital. All had received antenatal care. The details of these babies are given in Table I.

TABLE I.

Details of Delivery (Fetal heart not heard on admission).
(5 patients)

No.	Gestation Length	Birth Weight.	Method of Delivery.	Comment.
1.	37	1700	S.V.D.	Baby small for dates.
2.	38	3090	S.V.D.	Booked for Home Confinement. Para 3+3.
3.	38	3170	S.V.D.	6 years involuntary infertility - combined care.
4.	39	2410	S.V.D.	
5.	40	3147	S.V.D.	

Although these patients were receiving antenatal care, in Cases 2 and 3 this was not adequate. Case 2 was booked for home confinement despite her parity, and Case 3 was not suitable for combined care because of her history of infertility.

Table II give s the details of the babies in whom the fetal heart was heard on admission to hospital.

TABLE LI.

Details of Delivery (Fetal heart heard on admission) (15 patients)

No.	Gestn. Length.	Birth Weight.	Method of Delivery.	Outcome.	Day of Death.	Comment.
1.	22	570	SVD	NND	1	No antenatal care.
2.	23	624	Breech	NND	1	
3.	25	879	SVD	NND	1	
4.	26	397	SVD	NND	1	
5.	26	1219	SVD	NND	1	
6.	27	964	SVD	NND	1	
7.	28	1042	SVD	NND	1	
8.	28	1150	LUSCS	NND	1	Prolapsed cord.
9.	28	1361	SVD	BBB	1	No antenatal care.
10.	30	1840	Breech	SB		Intra-uterine pneumonia.
11.	31	900	Breech	SB		APH - SB in previous pregnancy.
12.	38	1630	Breech	SB		Attempted abortion in first trimester.
13.	?	480	Breech	NND	1	SRM for 48 hours; pyrexial then APH
14.	?	964	Breech	NND	1	No antenatal care.
15.	?	836	Breech	NND	1	

All these babies were very small and all but one very immature.

Cases 1 and 2 should not really have been included as they were born before 24 weeks gestation, but they were born alive following haemorrhage of unknown origin. It therefore seemed sensible to include them in this group. Three patients did not receive any antenatal care, but it is unlikely that any of these deaths were in fact preventable, although in Case 10 the baby is of a size that might have survived if Caesarean Section had been carried out. This baby was obviously growth retarded.

Tables LII, LIII and LIV demonstrate the maternal characteristics of the 62 patients with abruptio placentae.

TABLE LII

Abruptio Placentae - Maternal Characteristics (62 patients)

Age.	No.	Rate.	Parity.	No.	Rate.	Social Class.	No.	Rate.	Marital Status.	Previous History.
< 20	11	5.3	0	17	3.0	I	0	0	Married 56	APH 11
20-29	34	3.5	1	9	2.1	II	2	1.9	Single 5	Low
30-39	13	3.6	2	11	4.2	III	31	3.7	N/K. 1	Birth
40 & over	4	10.9	3+	25	7.0	IV	10	3.1		Weight 13
						V	15	6.7		
						N/S	4			

There is a high rate of perinatal deaths in those women over 40 years (10.9 per thousand), having their 4th or subsequent child (7.0 per thousand), or those in Social Class V (6.7 per thousand).

Table LIII looks at the combined effect of age and parity on perinatal mortality from abruptio placentae, and Table LIV looks at the combined effect of parity and social class.

TABLE LIII.

The Effect of Maternal Age and Parity on Perinatal Mortality Rates.

Age.	Parity			
	0	1	2	3+
< 20 years	6.0	5.1	0	0
20 - 29 years	2.4	2.3	4.2	8.7
30 - 39 years	0	0	5.8	4.9
40 & over	0	0	0	15.8

TABLE LIV.

The Effect of Maternal Parity and Social Class on
Perinatal Mortality Rates.

Social Class.	<u>Parity</u>			
	0	1	2	3+
I	0.0	0.0	0.0	0.0
II	2.4	3.5	0.0	0.0
III	3.4	0.9	6.9	6.0
IV	3.5	0.0	2.1	7.5
V	3.6	7.4	2.4	11.7

High perinatal mortality rates are found in those women over 40 years having their fourth or subsequent child (15.8 per 1000) and those women of Social Class V having their fourth or subsequent child (11.7 per thousand).

Twelve patients received no antenatal care, and abruption of the placenta tended to occur earlier in these patients. Table IV compares the length of pregnancy in those patients who received antenatal care and those who did not receive any antenatal care.

TABLE LV.

Gestation Length at Delivery in Patients who did and
who did not receive antenatal care.

Total.	28	28-29	29-31	32-33	34-45	36-37	38-39	40 & over	N/S
A (50)	1	4	9	7	6	10	2	6	5
B (12)	2	1	1	2	3	0	0	0	3

A = Received antenatal care.
B = No antenatal care.

Eight stillbirths and four neonatal deaths occurred to the women who received no antenatal care. The fetal heart was heard on admission to hospital in 6 patients. Details of these babies are given in Table LVI.

TABLE LVI.

No antenatal care - Fetal heart heard on admission (6 patients)

No.	Gestn. Length	Birth Weight	Outcome.	Day of Death.	Comment.
1.	25	737	NND	1	No baby record.
2.	25	1049	NND	1	R.D.S. Mother unmarried.
3.	29	1420	NND	5	R.D.S.
4.	34	2155	SB		Slow fetal heart for 2 hours before SB.
5.	?	1276	SB		Threatened abortion.
6.	?	1980	NND	1	R.D.S.

In the three cases of neonatal death where there were records of the babies, all developed respiratory distress syndrome. It would appear from the case record that Case 4 might have been saved if more prompt medical action had been taken. The patient was admitted at 34 weeks in her second pregnancy by the Flying Squad because of vaginal haemorrhage. The decision was taken to observe her. The fetal heart was noted to be slow for 2½ hours. Examination under anaesthesia was then carried out. The cervix was found to be fully dilated but the fetal heart had stopped by this time.

The following tables give details of the babies whose mothers received antenatal care and who had a placental abruption.

TABLE LVII.

Antenatal care received (50 patients)

Comparison of Gestation Length of Pregnancies where Fetal Heart was heard on admission with those where fetal heart was not heard.

Total.	28 wks.	28-29	30-31	32-33	34-35	36-37	38-39	40 & over	N/S
X (27)	1	3	8	2	1	4	1	3	4
Y (23)	0	1	1	5	5	6	1	3	1
		51.9%							
		30.4%							

X = Fetal heart heard on admission.
Y = Fetal heart not heard on admission.

In 27 cases the fetal heart was heard on admission to hospital and in this group just over half (51.9 per cent) were less than 34 weeks gestation at delivery. In 23 cases the fetal heart was not heard on admission to hospital. A third of this group (30.4 per cent) were delivered before 34 weeks gestation.

Details of the 14 babies whose fetal hearts were heard on admission and who were delivered before 34 weeks gestation are given in Table LVIII.

TABLE LVIII.

Method of Delivery of Babies less than 34 weeks gestation (14 patients)
(Fetal heart heard on admission)

No.	Gestn. Length.	Birth Weight.	Method of Delivery	Outcome.	Day of Death.	Comment.
1.	25	765	Breech	NND	1	Respiratory Distress Syndrome.
2.	28	1210	SVD	NND	1	Respiratory Distress Syndrome.
3.	28	1361	Breech	NND	1	Respiratory Distress Syndrome.
4.	29	1219	LUSCS	NND	1	Respiratory Distress Syndrome.
5.	30	1814	SVD	SB		In hospital 3 weeks before delivery. Fetal heart stopped in labour. SRM. 43 hours before.
6.	30	1729	SVD	NND	4	No neonatal records.
7.	30	980	SVD	SB		Previous small baby.
8.	30	1280	Breech	NND	1	Traumatic delivery.
9.	30	1320	SVD	NND	5	No baby notes.
10.	30	3062	SVD	SB		Obviously wrong with dates. 1st visit at 28 weeks. Size = dates.
11.	31	1600	SVD	NND	4	Intussusception - baby too small for operation.
12.	31	2640	Breech	NND	1	Respiratory Distress Syndrome.
13.	33	1474	SVD	SB		Fetal heart stopped less than 1 hour before delivery.
14.	33	2722	SVD	SB		Fetal heart stopped less than 1 hour before delivery.

Of the babies who died in the neonatal period, 5 suffered from respiratory distress because of immaturity. There were no records for two babies. Case 8

suffered a traumatic breech delivery, and Case 11 had intussusception but was thought to be too small for operation to be undertaken. Four of the stillbirths might have been prevented if Caesarean Section had been undertaken. In Case 5 the patient had been in hospital 3 weeks because of vaginal bleeding which was slight but almost continuous. The membranes ruptured more than 48 hours before delivery. Spontaneous labour occurred, the fetal heart stopped and the baby was a good size, 1814 g. Case 10 was a big baby, 3062 g., so that gestation length ought to have been questioned at the antenatal clinic. The two babies, Nos. 13 and 14, both might have survived in Caesarean Section had been carried out.

The details of the babies whose gestation length was greater than 33 weeks and whose fetal heart was heard on admission to hospital are given in Table LIX.

TABLE LIX.

Method of Delivery in Babies of 34 weeks gestation or more.
(Fetal Heart heard on admission) (9 patients)

No.	Gestn. Length.	Birth Weight.	Method of Delivery	Outcome.	Day of Death.	Comment.
1.	35	2100	SVD	SB		Fetal heart stopped soon after admission.
2.	36	2000	FD	SB		
3.	36	2400	SVD	NND	3	Pneumonia. Intracranial haemorrhage.
4.	36	3997	SVD	SB		Hydramnios. Irregular fetal heart.
5.	37	2600	SVD	SB		
6.	38	2722	SVD	SB		Fetal heart stopped early in labour.
7.	40	3400	SVD	SB		
8.	41	3650	SVD	SB		Para 6+1, aged 29. At T+11. Admitted in labour - blood-stained liquor. Fetal heart stopped after 10 minutes.
9.	41	4111	SVD	SB		Slight bleed at home for few days. Admitted in 7early labour following day. Abruption. Weight gain poor.

All the babies in this group were certainly heavy enough to survive. All but one were stillborn and not one was delivered by Caesarean Section. Case 4 was noted to have hydramnios at the antenatal clinic but the patient refused admission. She was admitted in established labour. The fetal heart could not be heard initially but was heard with the Doptone at 100 to 116 beats per minute for two hours. It thereafter remained regular until the second stage, when it was not monitored. After 15 minutes a fresh stillborn baby was delivered. There was a retroplacental clot covering half the placenta. In Case 5 the fetal heart was heard to be irregular soon after admission, and artificial rupture of the membranes was carried out, and as the fetal heart was then regular the decision was taken against Caesarean Section; the fetal heart then stopped. If induction at term had been carried out in Cases 8 and 9, antepartum haemorrhage might not have occurred. This policy is indicated in Case 8 as the patient was at risk, being of high parity and of Social Class V, and Case 9 had not gained any weight for 6 weeks before delivery. More active management by Caesarean Section might have prevented some of these perinatal deaths.

The details of the babies of unknown gestation and in whom the fetal heart was heard on admission are given in Table LX.

TABLE LX.

Babies of unknown gestation - Fetal heart heard on admission.
(4 patients).

No.	Birth Weight.	Method of Delivery.	Outcome.	Comment.
1.	709	Breech	SB	
2.	1191	Breech	SB	Last pregnancy 31 weeks previously.
3.	1701	SVD	SB	
4.	3374	LUSCS	SB	Fetal heart heard prior to Section

There is not enough information to determine whether a change in obstetric management would have altered the perinatal outcome.

In 23 patients the fetal heart was not heard on admission to hospital following abruption of the placenta. The first antenatal visit was quite late on in pregnancy. Only six patients were seen before 20 weeks, 7 patients between 20 and 29 weeks gestation, 6 patients did not attend the clinic until the 30th week or later, and it was not known when 4 of the patients first attended.

The patients whose fetal heart was heard on admission to hospital had a slightly better record at the antenatal clinic; 8 attended before 20 weeks, 9 attended between 20 and 30 weeks, and no one attended later than 29 weeks gestation. Information was not available for 10 patients as they either attended Corporation clinics or their own general practitioner for antenatal care. These details are given in Table LXI.

TABLE LXI.

Gestation Length at first antenatal Visit.

<u>Totals.</u>	<u>10 weeks.</u>	<u>10-19</u>	<u>20-29</u>	<u>30+</u>	<u>N/S</u>
X (27)	0	8	9	0	10
Y (23)	0	6	7	6	4
No care (12)					

X = Fetal heart heard on admission to hospital.

Y = Fetal heart not heard on admission to hospital.

Antenatal care in the patients losing their babies because of abruption of the placenta was certainly not adequate in 22 cases where the patients did not attend until after 20 weeks gestation and in the 12 patients who received no care at all.

Maternal hypertension was recorded in 7 of the 50 patients during the antenatal period.

The Flying Squad was called out on only ten occasions. Seven patients had abruption of the placenta, two had placenta praevia, and one had a haemorrhage of unknown origin.

DISCUSSION

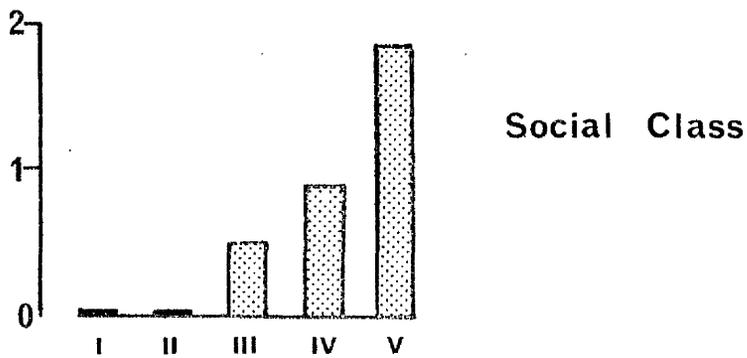
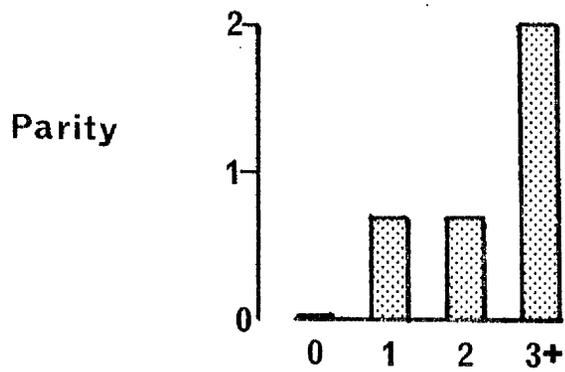
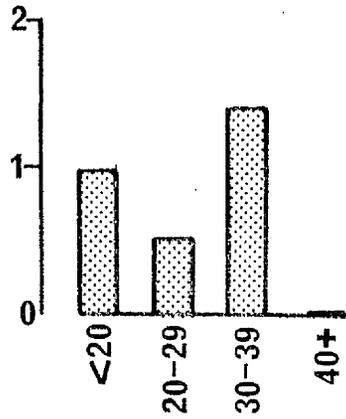
A number of studies have provided evidence that there is a place for delivery by Caesarean Section in the management of abruptio placentae (Hibbard and Jeffcoate, 1966; Donald, 1969; Hellman and Pritchard, 1971). Lunan (1973) in a recent survey of the management of abruptio placentae at the Glasgow Royal Maternity Hospital during the five years 1966-1970 concludes that "the best one can therefore do is to advocate intensive fetal monitoring and a readiness to do a Caesarean Section for the sake of the fetus alone in all patients with abruptio placentae whose babies are judged to weigh more than 2000 g." This policy is also advocated by Blair (1973) who reported a series of 189 cases of abruption of the placenta admitted in the five years from 1965 to 1969 to Bellshill Maternity Hospital, Lanarkshire. In his series the Caesarean Section rate was 16.4 per cent, compared to 8.6 per cent reported by Hibbard and Jeffcoate (1966). In Blair's study there were five neonatal deaths following Caesarean Section, but three of these babies weighed less than 1.5 Kg. and were less than 35 weeks gestation.

It would appear that a number of perinatal deaths from antepartum haemorrhage might be prevented by more active obstetric management. This means that if the baby is thought to be 2000 g. or so and the fetal heart is present, Caesarean Section should always be considered. If the baby is not thought to be big enough for delivery, antenatal monitoring with the help of oestriol estimations and biparietal diameter measurements would give a guide as to how the baby was developing, and if no further haemorrhage occurred the pregnancy could be continued until the baby was mature enough for delivery.

In general, it would appear that the care of these patients, who lost their babies because of antepartum haemorrhage, was unsatisfactory. The patients most at risk are those of high parity and low socio-economic status. Perhaps if contraceptive advice was made more freely and easily available for them, if they wished this advice, these problems might not occur.

FIG. 6.

Maternal Disease



(F) MATERNAL DISEASE:

Certain medical conditions of the mother are known to affect adversely the fetus, leading to intra-uterine death or premature delivery. Awareness of the effect of certain maternal conditions of the baby, for example, diabetes and heart disease, has led to the development of combined medical/antenatal clinics, and a great improvement in the perinatal outcome.

It was pleasing to find that during 1970 in Glasgow only 12 perinatal deaths (including one twin pregnancy) were attributed to maternal disease. This is a rate of 0.7 per 1000 births, which is a very small number of deaths, amounting to 2.7 per cent of all perinatal deaths. This is half the number that Baird and his colleagues found in Aberdeen twenty years ago (Baird et al. 1954). This difference is perhaps due to better maternal health now or improved antenatal care, or a combination of both. Fig. 6 illustrates the age, parity and social class distribution, showing a steady increase with all these factors.

FINDINGS:

As the number of patients in this group is so small, each case will be discussed separately. Three patients were found to have impaired glucose tolerance before delivery. They were all noted to be obese, but had not been known diabetics prior to pregnancy. Details of these patients are given in Table LXII.

TABLE LXII.

Patients with impaired Glucose Tolerance

No.	Age.	Parity.	Gestn. Length.	Birth Weight.	Outcome.	Time of first visit for antenatal care.
1.	30	1+0	41	5215	SB	No care.
2.	36	7+0	42	5414	IUD	34 weeks.
3.	39	8+1	40	3200	IUD	?

The first patient, whose first child also weighed over 5000g., received no antenatal care, was admitted ^{early} in labour at 41 weeks gestation. A glucose tolerance test was carried out and a mild diabetic curve was obtained. Her diabetic condition was treated by diet alone, but she failed to keep the clinic appointments. The second patient attended one of the Corporation antenatal clinics. She gave a history of large babies and had persistent glycosuria at the antenatal clinic. She was allowed to go 16 days past term and on admission it was noted that intra-uterine death had occurred. A glucose tolerance test at this time was abnormal, but was normal six weeks after delivery. The third patient was noted to have an abnormal glucose tolerance test at 37 weeks, but labour was not induced until term. These patients were all in the pre-diabetic state, and with more careful antenatal care, the perinatal outcome might have been different.

The maternal conditions contributing to the other nine perinatal deaths (including one set of twins) were very varied. The details of the pregnancies are shown in Table LXIII.

TABLE LXIII

Other Maternal Conditions

Condition.	Age.	Parity.	Gestn. Length.	Birth Weight.	Outcome.	Time of 1st visit for antenatal care.
1. Grade IV Cardiac	19	1+0	27	1040	NND	8
2. Syphilis + Drug Addict	27	2+0	36	2810	NND	17
3. Liver Cell Necrosis	30	2+0	36	3520	IUD	16
4. Acute Pancreatitis	28	3+0	40	2183	NND	22
5. Appendicectomy	25	4+0	40	1588	NND	27
6. Gastro-intestinal Upset	35	3+0	23	870	NND	17
7. Severe Pyrexia	19	1+0	31	1638	NND	23
8. Haematemesis)	28	3+2	36	1758	NND	?
) Twins						
9. Haematemesis)				1800	SB	?

The number of perinatal deaths due to maternal disease should be very few now and only rather odd bizarre conditions or circumstances should result in this. The most important single condition appears to be diabetes and the pre-diabetic condition, and perhaps not enough attention is paid by the obstetricians to the hazards of this condition, e.g. the second patient in the pre-diabetic group was allowed to go 16 days postmature, and the other two patients were allowed to go past term also. One patient received no antenatal care, so it would not have been possible in her case to alter the perinatal outcome.

As the number of diabetic patients is relatively few in each maternity hospital, there is much to be said for care to be concentrated in one centre where obstetricians, paediatricians and diabetic specialists can become expert in the care of the diabetic pregnant women. This is the practice in the Aberdeen Maternity Hospital where all diabetic patients in the North-East of

(g) FETAL DEFORMITY:

The vast majority of fetal abnormalities are due to faulty organogenesis, although a few are caused by pathological processes in the fetus during the last few weeks of pregnancy. As the editorial team and W.H. Schutt point out in their chapter on Congenital Malformations in "Perinatal Problems," (Butler and Alberman, 1969) "the effect of any given teratogenic agent will depend on the metabolic activity of the target organ and it is clear that an insult during embryogenesis will result in a disturbance of the processes of formation while the same insult, occurring later in pregnancy, is unlikely to result in a severe defect, although it might retard the growth of the fetus." It has been shown that the predisposing factors are parental, particularly maternal ageing, multiparity, low social class, irradiation before conception, viral infections and some drugs (Eriksson, Catz and Jaffe, 1973). The main hope of reducing fetal congenital malformations lies, therefore, in fundamental research on genetic and teratogenic influences. Butler (1967) has stated that chromosomal abnormalities probably account for less than 10 per cent of fetal congenital anomalies and teratogenic influences such as intra-uterine rubella for less than 5 per cent.

Regional variations have been reported in the incidence of central nervous system abnormalities. Rogers (1969) has shown that in England and Wales, the frequency of neural tube malformations is highest in the north and north-west and lowest in the east, south-east and south. Edwards (1958) has reported differences in the regional frequency of anencephalus, spina bifida and hydrocephalus in Scotland.

Richards, Roberts and Lloyd (1972) in their study of area differences in the prevalence of neural tube malformations in South Wales, conclude that

their findings "considered alongside the absence of an association between grouped area prevalence and ethnic origin, social class, parity or maternal age, suggests that the factors principally responsible for the area differences in neural tube malformation prevalence in South Wales, though still unknown, may well be external environmental characteristics associated with the nature of the area itself, such as the softness of local water supplies (Love et al. 1971) rather than personal environmental factors associated with the population of that area, such as diet, occupation, recreational activities and religion." An association between anencephaly and potato blight has also been reported (Renwick, 1972). This finding, however, has not been confirmed (Field and Kerr, 1973).

In a genetic study of anencephaly and spina bifida in Glasgow, Richards, McIntosh and Sweeney (1972) found that the incidence of anencephaly and spina bifida among all Glasgow births in 1964 to 1968 were each 2.8 per 1000. Among the viable siblings of 146 cases of anencephaly the incidence of congenital defects was 8.4 per cent (neural tube defects 5.7 per cent) and among the siblings of 172 cases of spina bifida it was 10.4 per cent (neural tube defects 5.6 per cent). Anencephaly in primigravidae was associated with a high incidence of congenital defects in later pregnancies (all defects 12.9 per cent, neural tube defects 8.6 per cent).

They also noted several aberrations in the structure of the families investigated, which suggest the action of sex-based genetic factors in these families, although precise reasons for these findings were not elucidated.

In another study from Glasgow, Federick and Wilson (1971) found considerable geographical variation in all stillbirths, and early deaths recorded as having malformations of the central nervous system delivered between 1st January, 1964 and 31st December, 1968. They demonstrated a clustering in space and time among the cases of hydrocephalus which was not evident for

cases of spina bifida and anencephalus. They state, however, that it is possible that the picture would have been altered by the inclusion of those infants with spina bifida and hydrocephalus who survived the neonatal period.

Baird (1974) in a report of a study of central nervous system abnormalities, has shown suggestive evidence that in Scotland women born during the worst of the industrial depression during the years 1928 to 1932 had subsequently higher stillbirth rates from central nervous abnormalities. His suggestion that the fact that the mother's reproductive mechanism was affected during her own intra-uterine life implies that her oocytes must have been damaged either before or soon after she was born. He states that "the high death rate from C.N.S. malformations has been the main reason for the higher perinatal mortality rate in Britain compared with other North European countries, for example, in 1961-1963 the stillbirth rates in the Netherlands, England and Wales and Scotland were 13.1, 15.3 and 16.2 respectively but when deaths from C.N.S. malformations are removed the rates for all other causes were 12.8, 13.4 and 13.2 respectively.

FINDINGS

During 1970 the greatest number of deaths occurred in the abnormality group. There were 105 perinatal deaths, giving a rate of 6.3 per 1000 live and stillbirths. Fig. 7 shows the perinatal mortality rates by age, parity and social class. There was very little variation. The majority of the deaths were due to central nervous system abnormalities. Table LXIV shows the distribution of the deaths. The incidence of anencephaly (2.8 per 1000) was identical with that found by Richards et al. (1972) for the years 1964 - 1968.

TABLE LXIV.

Distribution of Deformity Perinatal Deaths.

Abnormality	
Central Nervous System	63
Anencephaly	46
Other	17
Heart	14
Alimentary	8
Other	20

The characteristics of the mothers with anencephalic fetuses were looked at in detail, but no definite pattern emerged with regard to their age, parity or social class (Table LXV). The distribution of blood groups and country of origin (Table LXVI) showed no pattern either.

TABLE LXV.

Maternal Characteristics (with anencephalic fetuses)

Age	No.	Parity.	No.	Social Class.	No.
< 20 years	5	0	17	I	1
20 - 29 years	30	1	15*	II	2
30 - 39 years	10	2	7**	III	20
40 and over	1	3+	7***	IV	13
				V	8
				N/S	2

* = Previous Anencephalic.

TABLE LXVI

Blood Group and Ethnic Origin

Blood Group	Ethnic Origin	
	+ve	-ve
O	14	1
A	10	2
B	3	2
AB	0	2
N/S	12	
		Scotland 33
		Ireland 1
		Europe 1
		India/Pakistan 3
		N/S 8

Previous studies have shown that frequently anencephalic pregnancies go postmature (Milic et al., 1969), and that there is a greater number of female babies delivered. When an anencephalic fetus is now diagnosed, labour is frequently induced, usually by the use of prostaglandins. (Previous induction was not carried out because of the inefficiency of the various methods and the risk of intra-uterine infection). The gestation lengths of these pregnancies which were not induced and those which were induced have been looked at separately by male/female ratio (Table LXVII).

TABLE LXVII.

Length of Pregnancy and Male/Female Ratio

	<u>Non-Induced Pregnancies.</u>			<u>Induced Pregnancies.</u>		
	<u>Male.</u>	<u>Female.</u>	<u>N/S.</u>	<u>Male.</u>	<u>Female.</u>	<u>N/S.</u>
< 35 wks.	7	8		2	7	1
35-36 wks.	0	1		0	2	
37-39 wks.	2	4	1	3	2	
40 & over	1	0		0	1	
N/S	0	2	1	0	1	
	10	15	2	5	13	1
	Male = 15			Female/Female Ratio = 0.54		
	Female = 28					

In this small number of anencephalic fetuses, there was no evidence of postmaturity, there was a considerable increase in the number of female fetuses born. In 18 of the 41 anencephalic pregnancies delivered in hospital, the abnormality was known before labour began. In a number of cases the fetuses were not weighed but generally anencephalic fetuses are light-for-dates.

The characteristics of the mothers whose babies had other C.N.S. abnormalities are shown in Table LXVIII, and the percentage distribution by age, parity and social class of mothers with C.N.S. abnormalities are compared with those with other abnormalities (Table LXIX).

TABLE LXVIII.

Maternal Characteristics - Other C.N.S. Abnormalities
(17 patients)

Age	No.	Parity.	No.	Social Class.	No.	Ethnic Group
< 20 years	1	0	2	I	0	Scotland 15
20-29 years	10	1	3	II	2	India/Pakistan 1
30-39 years	6	2	4	III	6	N/S 1
40 and over	0	3+	8	IV	5	
				V	3	Previous History
				N/S	1	of Deformity 3

There was a predominance of female fetuses 11:7, and nearly all the babies were stillborn (14). Three babies were born alive but all died during the first day of life, leaving no time for corrective operation. The presence of abnormality was diagnosed before delivery in 4 cases.

TABLE LXIX.

Distribution of Deaths (in percentages)

	<u>Age.</u>		<u>Parity.</u>		<u>Social Class.</u>	
	A.	B.	A.	B.	A.	B.
< 20	9.5	23.8	0	30.2	40.5	I 1.6 4.8
20-29	63.5	47.6	1	28.6	23.8	II 6.4 7.1
30-39	25.5	26.2	2	17.5	11.9	III 41.3 52.4
40 +	1.6	2.4	3+	23.8	23.8	IV 28.6 21.5
						V 17.5 9.5
						N/S 4.8 4.8

A = C.N.S. Abnormalities.
B = Other Abnormalities.

In the mothers of babies with central nervous system abnormalities there were fewer women under 20 years and fewer primigravidae than in the other abnormality group, but there was a higher percentage of patients in Social

Classes IV and V (46.1 per cent as opposed to 30.9 per cent).

The maternal characteristics of the mothers whose babies died from cardiac, alimentary or other abnormalities are shown in Table LXX.

TABLE LXX.

Maternal Characteristics

	Age.	No.	Parity.	No.	Social Class.	No.
<u>Cardiac:</u>	< 20 yrs.	3	0	5	I	0
	20 - 29 years	8	1	3	II	1
	30 - 39 years	3	2	2	III	8
	40 & over	0	3+	4	IV	3
					V	1
					N/S	1
<u>Alimentary:</u>	< 20 yrs.	3	0	3	I	0
	20 - 29 years	1	1	2	II	0
	30 - 39 years	4	2	1	III	5
	40 & over	0	3+	2	IV	2
					V	0
					N/S	1
<u>Other:</u>	< 20 yrs.	4	0	9	I	2
	20 - 29 years	11	1	5	II	2
	30 - 39 years	4	2	2	III	9
	40 & over	1	3+	4	IV	4
					V	3

No definite trend is shown in Table LXX to indicate what type of woman is at risk from non central nervous system abnormality.

Details of the babies who died because of congenital heart disease are given in Table LXXI.

TABLE LXXI.

Details of Babies with Congenital Heart Disease (14 patients)

No.	Gestn. Length.	Birth Weight.	Sex.	Percentile Weight.	Day of Death.
1.	32	2041	M	-	IUD - macerated. Congenital heart lesion ? extent.
2.	32	1800	F	< 25th	2. AMN - Other.
3.	35	1928	F	< 5th	1.
4.	35	2244	M	< 25th	4. Diabetic - threatened abortion.
5.	38	2310	M	< 5th	2.
6.	39	2800	F	< 10th	3.
7.	39	3740	M	< 75th	4.
8.	40	2840	F	-	5.
9.	41	2700	M	< 5th	6. Threatened abortion. Slight bleeding at term.
10.	41	3160	F	-	4.
11.	41	3370	M	< 25th	5.
12.	41	3629	M	-	2.
13.	41	4054	M	< 90th	4.
14.	?	1650	M	-	1.

Male/Female Ratio = 8:6

Of the 9 babies where the percentile weight could be calculated, 4 of them weighed less than the 10th percentile.

Details of the babies with alimentary complications are given in Table LXXII.

TABLE LXXII.

Details of Babies with Alimentary Abnormalities.
(8 patients)

No.	Gestn. Length.	Birth Weight.	Sex.	Percentile Weight.	Day of Death.
1.	31	1400	F	-	IUD. Macerated. Exomphalus.
2.	32	-	F.	-	1. Exomphalus and other abnormalities.
3.	33	3070	F.	-	1. P.M. Congenital meconium peritonitis, volvulus of ileum and congenital bands. Intestinal obstruction.
4.	37	2835	M	< 50th	4. Meconium ileus.
5.*	37	2843	M.	< 10th	3. Oesophageal atresia and others.
6.	40	3005	M	< 10th	1. Tracheo-oesophageal fistula.
7.*	?	1219	F	±	1. Meconium peritonitis.
8.*	?	2381	M	±	1. Oesophageal atresia.

Male/Female Ratio = 4:4

* = Maternal Hydramnios recorded.

One patient was a rubella contact and one patient had hydramnios, otherwise the pregnancies in the women with multiple congenital abnormalities appeared to be progressing normally and abnormalities were not suspected.

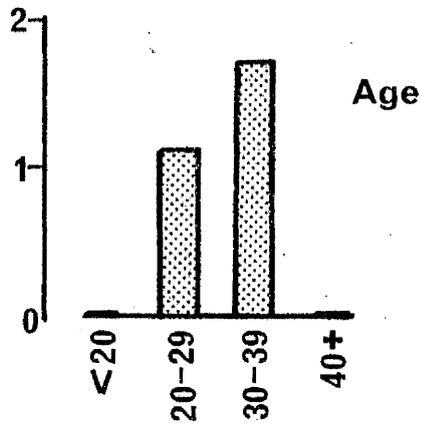
DISCUSSION

The numbers in each group of abnormality in this survey are too small to draw definite conclusions about the aetiology of the various conditions. There was, however, a larger percentage of patients in Social Class IV and V (46 per cent) compared with all deliveries in Glasgow (32 per cent). There was also an area difference of central nervous system abnormalities. This has been discussed in Chapter XII. Until the aetiology of the various abnormalities is known it is unlikely that perinatal mortality rates will be greatly reduced.

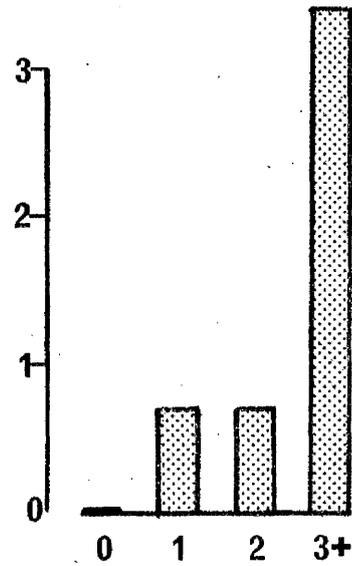
Considerable maternal suffering, however, could be reduced by earlier diagnosis of the condition and termination of the pregnancy offered to the patient. Much research has been directed towards the detection of fetal abnormality early in pregnancy. Brock and Sutcliffe (1972) reported raised alpha-fetoprotein levels in late pregnancy in the amniotic fluid of patients with anencephalic fetuses. Subsequent reports (Lorber et al. 1973; Nevin et al. 1973) have recorded raised alpha-fetoprotein levels in cases of spina bifida before 20 weeks gestation.

Studies of amniotic fluid can be made to detect chromosomal abnormalities, fetal sexing and certain metabolic disorders. Anencephaly and spina bifida can also be detected by the use of ultrasound. Turnbull (1973) stated that "antenatal diagnosis of fetal abnormality before the twentieth week of pregnancy is a fast expanding field. Down's syndrome and other chromosomal abnormalities, fetal sexing for sex-linked disorders, some of the dysraphic neural tube malformations and one or two of the inherited metabolic disorders are now amenable to this approach, and other conditions will almost certainly be added in the near future. This work is specialised and should be carried out by teams of experts."

Rhesus Incompatibility



Parity



Social Class



(h) RHESUS INCOMPATIBILITY AND OTHER CAUSES OF PERINATAL DEATH:

Rhesus incompatibility (anti-D) causes most of the severe cases of haemolytic disease of the newborn. A number of relatively mild cases and a few severe ones are caused by anti-A or anti-B immunisation. The disease is also caused occasionally by other Rh antigens or by incompatibilities within other blood groups such as Kelly or Duffy.

Immunisation usually occurs in an Rh negative woman as a result of pregnancy with an Rh positive baby. It can also occur, however, after a transfusion with Rh positive blood. If sufficient Rh positive fetal red cells enter the mother's circulation, she will develop antibodies, but a small transplacental haemorrhage may only lead to sensitization and a further Rh positive pregnancy is required to produce detectable antibodies. Sensitization can also occur if the patient has an antepartum haemorrhage and an amniocentesis.

According to Grosse (1971), among Caucasians approximately 15 per cent are Rh negative, 38 per cent are Rh positive, homozygous, and 47 per cent are Rh positive, heterozygous. She states that "It has been estimated that 11 to 13 per cent of marriages are between Rhesus negative women and Rhesus positive men, but only 1 in 20 of these marriages will at some time produce an affected infant. The first Rh positive child is rarely affected unless the mother has had a previous Rh positive miscarriage, blood transfusion or intramuscular blood injection, or there has been a considerable "foetal bleed" during pregnancy. The second or subsequent children may be affected, but some Rh negative mothers may never produce antibodies even after many pregnancies with Rh positive children. However, once a mother has produced antibodies, all her subsequent Rh positive children will be affected although some may be mildly affected."

During the last decade there have been great advances in the antenatal care of Rhesus sensitised women. The principal problem of the fetus prior to birth is anaemia and the fetus may succumb in utero of high output heart failure.

The obstetrical objectives of antenatal care have therefore been designed (Talbert, 1972) as follows -

- (1) Prevention of intra-uterine death of the fetus secondary to erythroblastosis by early delivery or by intra-uterine transfusion.
- (2) Prevention of neonatal death resulting from prematurity due to unnecessary early delivery.
- (3) Delivery of an infant with the greatest possible maturity and in the least possible distress from haemolytic disease.

In order to achieve these objectives it is essential to estimate the severity of the haemolytic disease process during the antenatal period. This is carried out by (a) obtaining an accurate obstetric history from the patient to determine the severity of the disease in the past, (b) the detection of Rh antibodies in the maternal circulation. The most commonly used test is the indirect Coombs test. In this test a suspension of Rh positive red cells are reacted with maternal serum and treated with an anti-human globulin suspension prepared in the rabbit. A positive test is agglutination of Rh positive red cells by rabbit antibody to human globulin. Although the height of the Coombs titre correlates with the degree of anaemia in the fetus, the correlation is not sufficiently accurate to allow therapy to be based entirely on titres, therefore (c) an analysis of the amniotic fluid is carried out. The fluid is obtained by transabdominal amniocentesis. The liquor of an affected fetus has a different optical density from that of a normal fetus because of the presence of bilirubin, a break-down product of haemoglobin. A series of zones have been devised by Liley (1965) which by plotting the optical density of the amniotic fluid against gestation illustrates how severely affected the fetus is. The height of the peak correlates well with the degree of anaemia. If a high peak is obtained and the fetus is severely

Scotland are delivered. These women attend a special antenatal clinic and are seen at each visit by a gynecologist, whose particular interest is diabetes in pregnancy, and a diabetic medical specialist. Because of this united approach the care of the diabetic pregnant patient has been greatly improved, and the stimulus to investigate the problems of the pregnant diabetic woman has been enormous.

affected the decision must be taken whether delivery is indicated or an intra-uterine transfusion. This decision will be based upon the activity of the fetus. This procedure is done by infusion of Rh negative red cells cross-matched with the mother's blood into the fetal peritoneal cavity by way of a needle inserted transabdominally under fluoroscopic control. The procedure may need to be repeated two or three times in order to permit the pregnancy to advance sufficiently to give a reasonable chance of extra-uterine survival. This procedure has a fetal mortality rate from the procedure of around 15 per cent and should not be done unless there is a severely affected infant or an intra-uterine death, along with high titres and at least two amniotic fluid analyses suggesting fetal death in utero before 32 weeks. After 32 weeks delivery is preferable to intra-uterine transfusion (Talbert, 1972).

FINDINGS

During 1970 in the City of Glasgow there were 17 perinatal deaths classified as being due to Rhesus incompatibility. The perinatal mortality rates by age, parity and social class are illustrated in Fig. 8. The maternal characteristics are given in Table LXXIII. The mothers were all, of course, Rhesus negative.

TABLE LXXIII.

Maternal Characteristics

<u>Age.</u>	<u>No.</u>	<u>Parity.</u>	<u>No.</u>	<u>Social Class.</u>	<u>No.</u>
< 20 yrs.	0	0	0	I	0
20-29 years	11	1	3	II	0
30-39 years	6	2	2	III	5
40 & over	0	3+	12	IV	4
				V	7
				N/S	1

Twelve of the 17 patients were having their fourth or subsequent child, and 11 patients came from Social Class IV or V.

Details of the patients' first attendance are given in Table LXXIV.

TABLE LXXIV

Gestation Length at first antenatal Visit

<u>< 10 weeks.</u>	<u>10-20 weeks.</u>	<u>20-30 weeks.</u>	<u>30+ weeks.</u>	<u>No care</u>
<u>1</u>	<u>4**</u>	<u>8**</u>	<u>2</u>	<u>2**</u>

* = Previous perinatal death due to Rh incompatibility.

The patients did not attend for their first clinic visit until the second half pregnancy, and two received no care despite the fact they had both lost babies because of Rhesus incompatibility. These two women were both separated from their husbands, and presumably this was a contributory factor in their failing to come for care during their pregnancies.

Of the 15 patients who received antenatal care, all but one had amniocentesis attempted (this was not successful in two patients) to assess the degree of severity of the Rhesus isoimmunisation. One patient went into labour and delivered at 30 weeks gestation before amniocentesis was carried out.

In the 9 patients where amniocentesis only was attempted, it proved to be successful in 7 cases. Details of these nine babies are given in Table LXXV.

TABLE LXXV.

Details of Babies - Amniocentesis Only

No.	Gestn. Length.	Birth Weight.	Sex.	Labour.	Outcome.	Comment.
1.	32	1899	M	Spont.	NND	Intra-uterine transfusion refused. Kernicterus.
2.	34	2511	M	Spont.	NND	Cyanotic attacks.
3.	34	?	M	Spont.	NND	Undiagnosed twins. Other baby alive and well.
4.	35	2010	F	Induced	NND	R.D.S. Septicaemia.
5.	35	2900	M	Spont.	IUD	
6.	35	4309	M	Spont.	SB	Hydrops.
7.	?	2060	M	Induced	NND	Severe R.D.S. Failed Amniocentesis.
8.	35	2722	F	Induced	SB	Hydrops.
9.	38	3510	M	Induced	SB	Hydrops.

In the successful amniocentesis group, neonatal death occurred in two babies where labour was induced because of the severity of the Rhesus incompatibility, but both babies died because of respiratory distress syndrome. One patient refused intra-uterine transfusion, and then went into premature labour at 32 weeks. The baby died, and at postmortem, bilateral intra-ventricular haemorrhages and kernicterus were demonstrated.

Following amniocentesis in 5 patients, the degree of Rhesus isoimmunisation was found to be so severe that intra-uterine transfusion had to be carried out. Details of the babies whose mothers received an intra-uterine transfusion are given in Table LXXVI.

FIG. 7.

Fetal Deformity

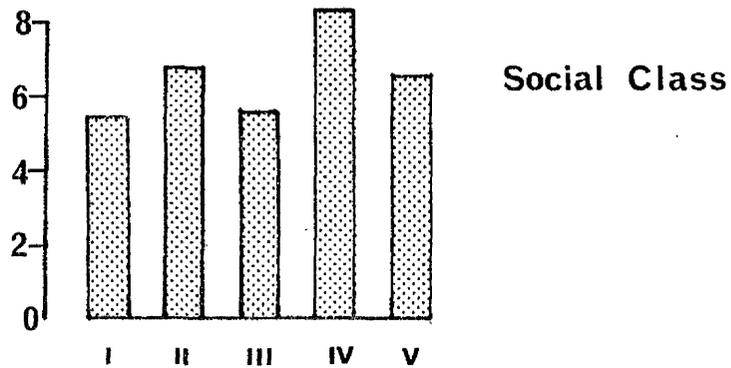
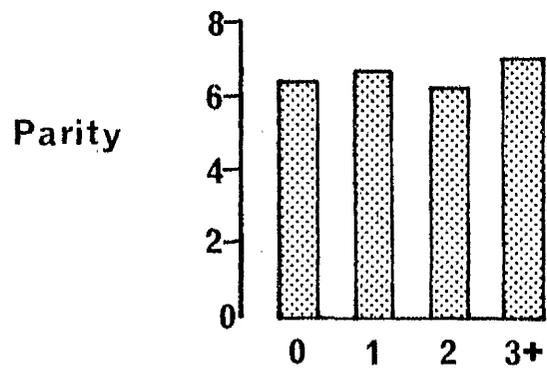
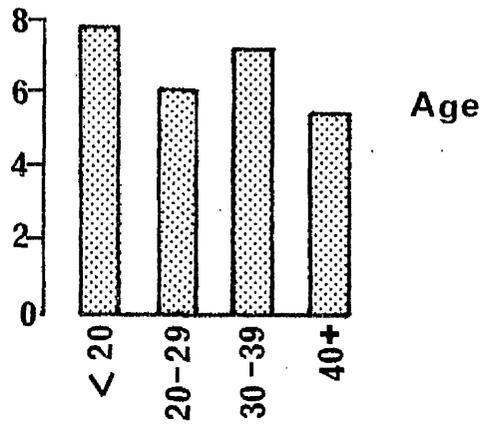


TABLE LXXVI

Details of Babies delivered after Intra-uterine Transfusion.

No.	Gestn. Length.	Birth Weight.	Sex.	Labour.	Outcome.	Comment.
1.	29	1490	M	Spont.	NND	Hydrops.
2.	34	1857	F	Spont.	IUD	
3.	33	1148	M	Spont.	NND	Respiratory Distress.
4.	34	1361	F	Induced	IUD	Macerated hydropic fetus.
5.	35	2608	M	Induced	SB	Severe abruptio placentae following induction.

All the babies delivered after intra-uterine transfusion were severely affected. Two deaths occurred before labour began, a severe degree of abruption of the placenta occurred in another case following artificial rupture of membranes despite immediate transfer to theatre for Caesarean Section, fetal death occurred before the anaesthetic was given. Two babies were alive at birth but one was hydropic and the second baby died on the third day because of respiratory distress. Looking at the notes in retrospect, it is difficult to see how these five deaths could have been prevented.

DISCUSSION

Great advances have been made in the prevention of Rhesus isoimmunisation. Research at the Nuffield Unit of Medical Genetics (Liverpool) suggested that it might be possible to prevent immunisation by neutralising the transplacental haemorrhages occurring at delivery by the administration of anti-Rh (anti-D) antibodies after delivery (Finn, 1960). It has now been shown that the administration within 72 hours after birth of 1 ml. anti-D gamma-globulin to an Rh negative mother after her first Rh positive ABO compatible infant can suppress immunisation (Clarke, 1968)

Immediately following delivery of the Rh negative patient, a Coombs test is performed on the mother and the infant's blood group is typed. If the mother's indirect Coombs test is negative and the fetus is Rh positive then anti-D is given within the following 72 hours following delivery. As there are still women already sensitised having babies, the problem of Rh haemolytic disease remains with us meantime. Careful management of pregnancy is still therefore necessary, so that the severely affected child is detected early and steps can be taken to prevent stillbirth and hydrops. Once the baby is born, extreme care must be taken to prevent the baby developing kernicterus. There is a very fine line between delivering the baby in time to prevent it becoming hydropic in utero and delivering it so early that it dies from severe respiratory distress in the neonatal period. Measuring the Sphingomyelin-Lecithin ratio in the amniotic fluid to indicate maturity of the fetal lungs (Whitfield, 1972) would help to decide whether immediate delivery or intra-uterine transfusion was indicated. Walker (1968) has shown that previous obstetric history is extremely important. There is a 60 per cent risk of intra-uterine death before 36 weeks gestation if there has been a previous stillbirth or severely affected infant. The risk is 33 per cent with a history of a less severely affected infant requiring treatment and this risk falls to 20 per cent if a previous child was mildly affected and did not require treatment.

Only 5 of the patients who received antenatal care attended the clinic before twenty weeks of pregnancy. To improve the perinatal outcome the patients must be seen early in their pregnancy so that amniocentesis can be carried out and the condition of the fetus assessed. Every patient ought to know what her blood group is and if she is found to be Rhesus negative the importance of early care in subsequent pregnancies should be explained to her. To ensure that the patient receives expert care, she ought to be seen at a special clinic for the care of Rhesus sensitised patients, and there is something to be said for this

to be concentrated in a few centres in a City the size of Glasgow, so that intra-uterine transfusion can be carried out and specialised neonatal facilities are available. Close co-operation is obviously required between obstetrician, haematologist, radiologist and paediatrician.

Until Rhesus isoimmunisation no longer exists, it is only by careful antenatal and neonatal care that the perinatal mortality from this cause can be reduced.

OTHER DEATHS

Six perinatal deaths occurred during 1970 in the City of Glasgow which were not thought to be suitable for inclusion with any of the previous categories. They have now been grouped together and details of each pregnancy are given in Table LXXVII below.

TABLE LXXVII.

Details of Pregnancies of "Other" Group.

No.	Age.	Parity.	Marital Status.	Social Class.	Gestn. Length.	Birth Weight.	Outcome.	Comment.
1.	24	0+0	S	IV	40	?	IUD	Probably M.U. or P.U. but no birth weight.
2.	21	0+0	M	IV	40	3940	NND	** ++
3.	20	0+0	M	III	38	3350	NND	Intra-uterine pneumonia.
4.	29	3+0	M	III	36	2820	SB	Intra-uterine pneumonia.
5.	28	5+0	M	III	34	3289	SB	Hydrops - mother Rh +ve, no antibodies.
6.	27	0+1	M	II	26	500	NND	Cervical incompetence.

**
++ Classified as unexplained but probably should be either M.U. or Trauma as second stage over 1 hour.
P.M. "Fetal distress, unexplained death."

CHAPTER VI.

WEIGHT GAIN IN PREGNANCY ENDING IN PERINATAL DEATH.

Many factors are known to affect the birth weight of the baby. These include length of gestation, age, parity, height and social class of the mother, sex of the baby, also maternal disease and smoking habits.

Maternal weight gain is another factor closely connected with baby weight. Davis (1923), Slemmons and Fagan (1927), Bingham (1932) and Hanley (1934) suggested that the weight of the fetus at term paralleled the weight increase of the mother, whereas Mollroy and Rodway (1937) reported that the weight of the infant was not directly dependent upon the mother's weight increase. The part played by heredity was claimed by Cummings (1934) to be the most important factor, although he thought that the state of health of the mother would be of some importance. Toombs (1931) concluded "that the size of the child at the time of delivery is determined by factors quite distinct from this consideration, and in most instances entirely beyond our control." Because of this controversy Beilly and Kurland (1945) felt it advisable to look at the problem again and subject their findings to statistical analysis. They found that there was a significant correlation between the weight gain of the mother and the weight of the baby at birth, although the degree of correlation was low. Beilly and Kurland also noted that the size of the mother influenced the weight gain during pregnancy - the light women showing a larger gain than the heavier women, but the heavier women tended to bear heavier offspring. Love and Kinch (1965) in a study of 2076 births reached a similar conclusion.

Eastman and Jackson (1968) discussed the effect of maternal weight gain and pre-pregnancy weight on birth weight in full time pregnancies. They stated that further knowledge about weight relationships in pregnancy is exceedingly important because of the major role played by low birth weight in neonatal mortality and in the aetiology of cerebral palsy. It is also known that superior mental development ensues in infants whose birth weight is above average, and that both mental and physical development improve with maternal weight gain (Churchill et al. 1966; Singer et al. 1968).

Eastman and Jackson (1968) found that increase in maternal weight gain was paralleled by progressive increase in mean birth weight and progressive decrease in the incidence of low-weight infants.

It has been shown that much of the estimated increase of body water in pregnancy can be accounted for by known components of weight gain, such as the fetus, placenta, liquor, added uterine muscle and mammary tissue, increase in plasma volume and red cell mass. Hytten et al. (1966) found that for women with little or no oedema there is remarkably good agreement between the estimated and the measured increases in water at 20 and 30 weeks. At term, however, the mean measured total body water content exceeds the estimated total body water by $1\frac{1}{2}$ to 2 litres. This discrepancy may be attributed to an increase in extracellular water. In women with oedema there appears to be a much larger increase in extracellular water and it would appear that a large increase in body water is required to produce a "good" pregnancy. In a study of the gain in total body water in women with normal pregnancies, Hytten et al. (1966) showed that the mean total body water in women with no clinical oedema rose by 6.84 Kg. body water between 10 and 38 weeks of pregnancy compared to the mean increase of 9.80 Kg. in women with generalised oedema during that period. The following year Thomson, Hytten and Billewicz (1967), from an epidemiological study, reported that women with oedema had a higher rate of weight gain than non-oedematous women, and that

babies born to oedematous women were heavier on average than those born to non-oedematous mothers.

In a study carried out by myself and others (Duffus, MacGillivray and Dennis, 1971), into the relationship between baby weight and changes in maternal weight, total body water, plasma volume, electrolytes and proteins and urinary oestriol excretion, it was found that in 20 patients with high weight gain defined as a weight gain of 1.4 pounds per week or more between the 20th and 30th weeks of gestation, 60 per cent of the babies were heavier than the 50th percentile and 10 per cent of the babies were lighter than the 10th percentile, i.e. normal distribution. In the 20 patients with low weight gain, however, (defined as a gain of less than 0.7 pounds per week between the 20th and 30th weeks of gestation), only 10 per cent of babies were heavier than the 50th percentile, and 35 per cent were lighter than the 10th percentile.

In this present study of perinatal mortality, it is obviously necessary to look at the patient's weight gain during pregnancy in order to determine whether there was any indication of the maternal response to her pregnancy not being as good as it should have been and to determine whether some change in obstetric management such as earlier intervention would have altered the outcome. This proved to be a very disappointing exercise, as maternal weight was not recorded in the domiciliary records; in the patients who received combined care the weight, if it was recorded by the general practitioner, was not recorded in the hospital records, and frequently the patients did not attend for their first antenatal visit until late in their pregnancy (see Chapter 10).

METHOD

Maternal weight for height was based on the measured weight at the 20th week of pregnancy and was expressed as a standard weight-for-height (Kemsley, Billewicz and Thomson, 1962).

Maternal weight gain was said to be high, normal or low.

High weight gain was defined as a weight gain of 1.4 pounds per week or more during the 10-week period 20 to 30 weeks gestation.

Normal weight gain was defined as a weight gain of 0.8 pounds to 1.2 pounds per week inclusive during the 10 week period 20 to 30 weeks gestation.

Low weight gain was defined as a weight gain of less than 0.7 pounds per week during the 10 week period 20 to 30 weeks gestation.

As factors such as gestation at birth and sex of the baby have an important bearing on the birth weight, it is necessary to use a standard which describes the distribution of birth weight at given gestational ages. Thomson, Billewicz and Mytten (1968) published an analysis of 52,004 legitimate single births which took place in the City of Aberdeen during the years 1948 to 1964. Their tables have been applied to the babies, where this was possible, standardising for maternal height, weight, parity, gestation length and sex of infant.

FINDINGS

In 281 patients the maternal weight/height ratio could be calculated. The distribution of weight/height ratio by clinical cause was said to be light, normal or heavy, and is shown in Table LXXVIII.

TABLE LXXVIII.

Maternal Weight/Height Ratio (281 patients)

Classif- ication.	No. of Patients	<u>Light.</u>		<u>Normal.</u>		<u>Heavy.</u>	
		No.	%	No.	%	No.	%
M.U.	29	3	10.3	11	37.9	15	51.7
P.U.	56	9	16.1	32	57.1	15	26.8
Tr.	18	3	16.7	6	33.3	9	50.0
Tox.	24	1	4.2	7	29.2	16	66.7
APH	50	5	10.0	21	42.0	24	48.0
M.D.	11	3	27.3	4	36.4	4	36.4
F.D.	74	6	8.1	32	43.2	36	48.6
Other	19	1	5.3	8	42.1	10	52.6
Total	281	31	11.0	121	43.1	129	45.9

As the weight/height ratio is not known in so many patients, it is only possible to make general conclusions, but it is of interest to note that 46 per cent of the patients were overweight and only 11 per cent were light for their height. This pattern was common to all women who had perinatal deaths apart from the premature - cause unknown group, where only 27 per cent were heavy for their height.

The patients' 20 to 30 week weight gain by clinical cause of perinatal death is shown in Table LXXIX.

TABLE LXXIX.

Maternal Weight Gain (20 - 30 weeks) (147 patients)

Classif- ication.	No. of Patients.	<u>Low.</u>		<u>Normal.</u>		<u>High.</u>	
		No.	%	No.	%	No.	%
M.U.	19	5	26.3	9	47.4	5	26.3
P.U.	23	7	30.4	12	52.2	4	17.4
Tr.	11	4	36.4	5	45.5	2	18.2
Tox.	18	3	16.7	6	33.3	9	50.0
APH	28	8	28.6	15	53.6	5	17.9
M.D.	6	2	33.3	4	66.7	0	0.0
F.D.	52	15	28.8	27	51.9	10	19.2
Other	10	1	10.0	6	60.0	3	30.0
Total	167	45	26.9	84	50.3	38	22.8

Fifty per cent of patients had a normal weight gain, 27 per cent had a low weight gain, and 23 per cent had a high weight gain. This general pattern was very different in the toxæmic patients where only 16.7 per cent of patients had a low weight gain, 33.3 per cent had a normal weight gain and 50 per cent had a high weight gain. The association of high weight gain and the development of pre-eclampsia is, of course, well recognised.

The percentile baby weights of the babies are shown in Table LXXX.

TABLE LXXX.

Percentile Weights by Clinical Cause (196 cases).

Classification	No. of Patients.	< 5th	< 10th	< 25th	< 50th	< 75th	< 90th	< 95th	> 95th
M.U.	25	4	5	4	5	3	4	0	0
P.U.	29	23	3	2	1	0	0	0	0
Tr.	16	3	1	3	2	2	3	1	1
Tox.	18	11	2	1	2	1	1	0	0
APH	35	14	5	4	5	4	1	2	0
M.D.	5	2	0	0	0	1	1	1	0
F.D.	58	35	7	6	4	2	4	0	0
Other	10	1	2	0	2	4	0	1	0
Total	196	93	25	20	21	17	14	5	1

The majority of babies where the percentile baby weight could be calculated were less than the 10th percentile, i.e. light for dates (118 out of 196). This was particularly noticeable in the premature- cause unknown group (26 out of 29), toxæmia (13 out of 18), and fetal deformity (42 out of 58).

DISCUSSION

It is unfortunate that the details of weight, weight gain and percentile baby weight were not known for all patients. One is left with the impression that the women who lost their babies were heavier than they ought to be for their height; 27 per cent of them failed to gain adequate weight and 60 per cent of the babies were growth retarded. The weight gain pattern of Glasgow women during pregnancy is not known. It would be exceedingly valuable if information about all Glasgow births could be obtained as Thomson, Billewicz and Hytten managed to collect for all Aberdeen births. This could be achieved if there was a standard antenatal record for all types of care, e.g. hospital, local authority, general practitioner or domiciliary care. If patients could always be seen at standard periods of gestation, e.g. 12, 20, 28, 30, 36, 40 weeks, and more frequently if required, the information would be easily obtained, collected, analysed, and made available for use by clinicians.

These findings may not be a true reflection of all the pregnancies that resulted in perinatal loss. For example, at first glance the high incidence (23 out of 29) of growth retarded babies in the premature - cause unknown group was most surprising. It must be remembered that a large number of patients in this group received no antenatal care, particularly these patients who delivered before 35 weeks gestation. It is possible that the group of patients where all the factors were known, i.e. weight, height, weight gain, etc., were in fact patients who delivered growth retarded babies and there were no details for those patients who delivered pre-term babies.

Hytten and Leitch (1971) have stated "that the best reproductive performance is associated with a weight gain a little less than 20 pounds

in the last half of pregnancy." It is known that the prematurity rate is influenced by the pre-pregnancy weight of the mother and by the mother's weight increase during pregnancy. Thomson and Billewicz (1963) have found that the prematurity rate (i.e. babies weighing 2500 g. or less) in the lightest 25 per cent of primigravidae was 9.6 per cent compared to the rate of 4.1 per cent in the heaviest 25 per cent. In his paper entitled "The Epidemiology of Prematurity," Baird (1964) showed that the women who gained a small amount of weight during pregnancy had high prematurity rates, whereas women who gained a large amount were more liable to develop pre-eclampsia, and for this reason tended to have an increased prematurity rate. The lowest prematurity rate was therefore found in association with a weight gain of about one pound per week between the 20th and the 36th weeks of pregnancy. This rate of weight increase was also associated with the lowest perinatal mortality.

Further information is required about weight and weight gain in pregnancy, particularly in high risk pregnancy. One had the impression that the association of maternal weight gain and fetal wellbeing, particularly towards the end of pregnancy, was not thought to be important. This was most obvious in the mothers who lost their babies in the mature - cause unknown group. To determine how important it is, further studies must be made. It would be interesting to know the normal weight patterns of parous patients and of patients who are light for their height who had a normal weight/height ratio and those who were obese before pregnancy began.

The impression one is left with, however, is that of the women who lost babies during the perinatal period, a substantial number were obese at the beginning of their pregnancy, and a substantial number of the babies were

growth retarded. This is an area of antenatal care where much more research is required. In the meantime, greater awareness of the importance of weight gain or failure to gain weight on the part of the obstetricians might lead to more active management and assessment of the intra-uterine state of the fetus if a patient's weight gain was poor. General practitioners, too, should be familiar with the normal pattern of weight gain in pregnancy. The patient ought to be weighed at each antenatal visit under standard conditions (stripped and wearing only a thin gown so that the weight of her clothes is eliminated), and if any faltering in her weight gain pattern occurs she ought to be referred to the hospital centre.

CHAPTER VII.

NEONATAL FINDINGS.

There is evidence that neonatal mortality can be reduced by the introduction of modern special care facilities for newborn babies. In a study quoted by Usher (1970) it was shown that a number of neonatal units had been operating for several years with adequate microchemistry, blood gas laboratory, respiratory and monitoring facilities. There was also full time medical staff for neonatal care. A number of hospitals were studied and it was found that neonatal mortality was lowest in the group having intramural intensive care facilities.

In the City of Glasgow in 1970 there were 207 special care cots for babies in eight maternity hospitals. Each of the two university centres had a Paediatrician whose major interest was in the field of neonatal paediatrics. In two other large centres general paediatricians looked after the neonatal unit and the other units were staffed by visiting paediatricians.

MATERIAL

During 1970 in the City of Glasgow there were 175 neonatal deaths, 103 of whom occurred during the first day of life (Table LXXXI).

TABLE LXXXI

Age at Death by Clinical Classification

	No.	0	1	2	3	4	5	6	7	N/S
M.U.	50	42	4	3	1	0	0	0	0	0
P.U.	101	45	36	7	4	3	3	1	2	0
Tr.	23	16	5	1	0	1	0	0	0	0
Tox.	29	19	4	3	1	0	1	1	0	0
AEH	94	62	23	3	1	2	2	0	0	1
M.D.	12	5	3	0	0	3	1	0	0	0
F.D.	105	59	25	7	4	6	3	1	0	0
Other	23	14	3	2	4	0	0	0	0	0
Total	437	262	103	26	15	15	10	3	2	1
%		60.0	23.6	5.9	3.4	3.4	2.2	0.7	0.5	0.2

FINDINGS

The paediatric records of the 175 babies who died during the neonatal period were studied in detail and the findings are discussed below.

Information about the condition of the babies at birth as judged by the Apgar score was available for 74 per cent of the babies. The first score was taken at 1 minute in some hospitals and 2 minutes in others. The Apgar score is a method of scoring devised by Virginia Apgar (1953) to evaluate the condition of the newborn infant. The following five objective signs, heart rate, respiratory effort, muscle tone, response to catheter in nose, colour, are evaluated and each given a score of 0, 1 or 2. A score of 10 indicates an infant in the best possible condition.

The details of the Apgar score by clinical classification are shown in Table LXXXII.

TABLE LXXXII.

First Apgar Score by Clinical Classification

	No.	<u>Apgar Score.</u>											N/S
		0	1	2	3	4	5	6	7	8	9	10	
M.U.	8	0	2	2	1	0	0	1	0	0	0	0	2
P.U.	56	1	8	14	3	3	4	2	3	2	0	0	16
Tr.	7	0	4	0	1	0	0	0	0	0	0	0	2
Tox.	10	0	2	1	0	1	2	0	0	2	1	0	1
APH	32	0	5	2	3	2	2	1	1	0	0	0	16
M.D.	7	1	0	0	0	2	0	1	0	0	0	0	3
F.D.	46	1	2	8	3	2	0	2	4	2	1	0	21
Other	9	0	1	1	1	1	1	0	2	0	0	0	2
Total	175	3	24	28	12	11	9	7	10	6	2	0	63
%		1.7	13.7	16.0	6.9	6.3	5.1	4.0	5.7	3.4	1.1	0.0	36.0

For the 119 patients in whom the Apgar score was known, the percentage with a score of less than 5 was calculated (Table LXXXIII).

TABLE LXXXIII.

Apgar Score less than 5 by Clinical Classification

	Score	5	
M.U.	(6)	5	(83.3 per cent)
P.U.	(40)	29	(72.5 ")
Tr.	(5)	5	(100.0 ")
Tox.	(9)	4	(44.4 ")
APH	(16)	12	(75.0 ")
M.D.	(4)	3	(75.0 ")
F.D.	(25)	16	(64.0 ")
Other	(7)	4	(57.1 ")
Total	(119)	78	(65.5 ")

All the babies in the Trauma group and the majority of the babies in the Mature - Cause Unknown, Antepartum Haemorrhage, Maternal Disease and Premature - Cause Unknown groups were in poor condition at birth.

Details of methods of resuscitation were known in 82 per cent of cases. These are described in Table LXXXIV.

TABLE LXXXIV.

Methods of Resuscitation by Clinical Cause

	No.	<u>Type of Resuscitation</u>					N/S
		None.	IPPR Intubation	IPPR Face Mask	IPPR + Respiratory Stimulant.	Other	
M.U.	8	1	2	2	2	0	1
P.U.	56	13	6	16	11	5	5
Tx.	7	0	1	0	3	1	2
Tox.	10	2	2	4	2	0	0
APH	32	4	8	6	7	1	6
M.D.	7	3	1	0	0	0	3
F.D.	46	19	3	5	4	1	14
Other	9	1	0	5	0	2	1
Total	175	43	23	38	29	10	32
%		24.6	13.1	21.7	16.6	5.7	18.3

IPPR = Intermittent Positive Pressure Respiration.

Resuscitation was apparently not required in 43 babies (24.6 per cent of all neonatal deaths.

Nineteen of these cases were in fact in the fetal deformity category so perhaps the decision was made not to resuscitate the baby irrespective of its condition at birth. In the premature - cause unknown group, 13 babies did not require resuscitation. The most common methods of resuscitation were by intermittent positive pressure respiration, either by face mask or intubation. A further group also received a respiratory stimulant. Three babies were put in the hyperbaric oxygen chamber.

A number of babies had problems with respiration, due to a number of causes, and in some cases there was more than one cause for the respiratory distress. The details are given in Table LXXXV.

TABLE LXXXV.

Respiratory Distress by Clinical Cause.

	No.	All Causes	R.D.S.	Infection	Aspiration	Pneumothorax	Other/ Immaturity
M.U.	8	6	2	1	5	0	0
P.U.	56	39	16	3	0	0	21
Tr.	7	1	0	0	0	1	0
Tox.	10	8	4	2	1	0	4
APH	32	22	6	0	0	0	16
M.D.	7	5	4	2	0	1	2
F.D.	46	17	1	2	2	0	13
Other	9	2	2	0	0	0	0
Total	175	100 (57.1%)					

There were 100 babies who had problems with respiration. The common problems were immaturity of the lungs and respiratory distress syndrome per se. Five of the six babies with respiratory distress in the mature - cause unknown group suffered from aspiration syndrome.

Forty-three babies were known to have a cerebral haemorrhage. An attempt was made to classify these into the various types, and this was possible where postmortem examination had been carried out. These details are given in Table LXXXVI.

TABLE LXXXVI.

Birth Injury.

	All Haemorrhages.	Subdural.	Sub- arachnoid.	Intra- ventricular.	Tear.	
					Falx.	Tentorium.
M.U.	1	0	0	0	0	1
P.U.	14	1	3	11	0	1
Tr.	4	0	1	0	0	3
Tox.	4	0	0	4	0	0
APH	10	0	0	8	0	2
M.D.	4	0	0	4	0	0
F.D.	4	0	0	1	0	3
Other	2	0	1	2	0	0
Total	43	1	5	30	0	10

Some babies had more than one type of haemorrhage. In ten cases a tentorial tear was found at postmortem. This sign of traumatic delivery would be expected in the trauma group, but not in the other categories. Thirty babies had an intraventricular haemorrhage which commonly occurs in babies suffering from anoxia.

Another important aspect of paediatric care is the transfer of babies in the neonatal period. During the antenatal period 16 mothers were admitted from home to hospital by the Flying Squad, eleven of these because of antepartum haemorrhage. Eight patients were transferred from one hospital to another, three when they were in labour. After delivery, 10 babies were transferred from home to hospital, 19 babies were transferred from one hospital to another, and two babies had two transfers. The reasons for the transfer in the neonatal period was either because the hospital where the baby was born had no special care facilities or that operation was required in the neonatal period and the baby was being transferred to the Surgical Department of the Children's Hospital.

DISCUSSION

The hospital records kept during the neonatal period did not contain nearly enough information for retrospective analysis. It was therefore impossible to obtain an accurate picture of the few hours of the babies' lives. Seventy-eight babies were known to have an Apgar score of less than 5 at the first count, 100 babies suffered from some form of respiratory distress, and 43 babies suffered from an intracranial haemorrhage. The intra-uterine environment for these babies had obviously been less than satisfactory.

In the Report of the Expert Group on Special Care for Babies (1971), it is stated that "The baby at risk travels best in utero. When difficulty can be anticipated, delivery should be at a maternity department with an associated special care unit which can deal with the baby." Earlier appreciation of problems during pregnancy and transfer of the mother before delivery to a special unit might have prevented a number of perinatal deaths in this study. The same report in a paragraph on medical records and exchange of clinical information states that "the history is of vital importance in the clinical management of the baby newly admitted to a special care nursery. The receiving nursery should make available to those who are likely to call on its services a short and simple proforma to be completed in advance and to accompany the baby to be admitted." The lack of information was very obvious when studying the records of the Glasgow perinatal deaths. When a baby was admitted to a paediatric department it was usually seen as an emergency and often a failure of communication between the labour record and the paediatric chart was revealed. Details about the baby frequently did not match up and this was particularly so when the baby was transferred from home or another hospital. The Report of the Expert Group on Special Care of Babies goes on to say that "the diagnostic information available from the records in each nursery should allow a comparative analysis of the work of special care nurseries. This creates a valuable opportunity to get information

on the range of work in the nurseries including variation between them."

At a meeting held at the Scottish Hospital Centre in February, 1973, the problem of neonatal information services was discussed. The importance of collecting neonatal statistics on a national basis was generally agreed, but the problem was deciding how much information should be collected. It was felt that it was important to ensure that (i) the collection of data was purposeful, (ii) the information to be collected was of a type which could be recorded accurately and completely, and (iii) the data could be collected during the routine care of the infant and not as an exception. From the problems encountered in collecting the perinatal mortality data in Glasgow it was suggested that a limited amount of accurate information should be collected on a national basis. Another ambitious scheme was proposed whereby there was one document for both the clinical case sheet and for transmission to the computer centre for processing.

Butler (1967) has stated that "medical details included in vital statistics systems for first week deaths are very limited and are likely to remain so as long as they are recorded on the same certificates as deaths at other ages." An improvement in the standards of record keeping is obviously required, and "a special care service for newborn babies should comprise anticipation and prevention of damage to the baby before and during birth, specialised observation and treatment for newborn babies, teaching and training of professional staff, follow-up and research" (Report of the Expert Group on Special Care of Babies, 1971).

The Report "Paediatrics in the Seventies," Court and Jackson (1972) was edited on behalf of the British Paediatric Association. In the section of Perinatal Paediatrics it is suggested that intensive care nurseries will most likely be found in the University hospitals, and in such a centre "the perinatal paediatrician's primary function is to develop the intensive care facilities, including equipment, techniques and suitably trained staff, sharing the consultant clinical responsibilities with one or more general paediatricians who have a special interest in the newborn.

"Treatment in the intensive care nursery would be available to vulnerable babies from a wide area; they would be transferred to the associated maternity unit in utero, or after birth to the intensive care nursery in a specially equipped ambulance staffed by nursery personnel. Babies requiring neonatal surgery are also suitable candidates for an intensive care nursery if appropriate geographical and professional relationships can be achieved."

Recommendations from this part of the Report are -

- (1) "The clinical care of the majority of newborn infants should remain in the hands of the general paediatricians.
- (2) "In addition there should be at least one specialist paediatrician working in each University centre, giving all or most of his time to perinatal paediatrics."

A Report "Towards an Integrated Child Health Service" (1973) by a subgroup of the Child Health Service from the Joint Working Party on the Integration of Medical Work for the Scottish Home and Health Department states that "All specialist maternity units will have special care nurseries but intensive neonatal care facilities will only be provided in a number of the larger centres where the most difficult obstetric and "at risk" deliveries take place. These intensive care centres should be sited within or close to large hospital complexes where essential services such as micro-chemistry, radiology, haematology and blood transfusion are available on a 24-hour basis, and where frequent and easy consultation with specialists such as cardiologists, surgeons and pathologists is possible."

In a City the size of Glasgow where in 1970 there were 16,748 deliveries, three such specialist centres responsible for 5,000 deliveries per annum would be required. Obstetric high risk patients should be transferred from other maternity units before delivery. This always poses a problem, as the consultant in charge of the patient feels his patient is being taken from him. Arrangements could be made for him to deliver the patient himself in the specialist maternity

unit if he so desired.

A high standard of antenatal care is required so that high risk patients are detected early and arrangements can be made for their delivery at a specialist maternity centre.

CHAPTER VIII.

MULTIPLE BIRTHS

When a survey of births is carried out the inclusion of multiple births presents a number of problems. Firstly, the mother is the same for two or more babies, complicating the analysis of maternal characteristics. Secondly, the problems relevant to multiple births differ from those of single pregnancies, i.e. the high preponderance of small babies, the classified cause of death being premature - cause unknown, tends to confuse the picture.

As this study was of all perinatal deaths, however, it was felt essential to include twin pregnancies in the analysis, but this chapter is included as it was felt that it is also of interest to analyse the deaths in multiple pregnancies separately.

Bulmer (1970) in his book "The Biology of Twinning in Man" shows from various studies that the incidence of twinning varies with race, with age and with parity (dizygotic twins only). "The monozygotic twinning rate is nearly constant at 3.5/1000 in all races. Dizygotic twinning rate varies widely, being 8/1000 in Caucasoids, twice that rate in Negro populations, and less than half that in Mongoloids. The rate of dizygotic twinning increases from a rate of zero at the menarche to a maximum at 37 years, it then falls sharply to zero at the time of the menopause." This increase of rate with age is thought to be due to an increase in gonadotrophin.

In a recent study from Dundee (Daw, 1974), a peak incidence of monochorionic twins was found in mothers aged 21 to 25 years and a peak incidence of dichorionic

twins in mothers aged 21 to 30 years. Daw states that this is significant when compared with previous reports showing a peak in the age group 35 to 39 years (Waterhouse, 1950), and confirms Guttmacher's view that the age of twin reproduction is falling. The Dundee figures are reflected in the latest Scottish National figures (Registrar General's Report, 1969).

The Dundee study also showed that primigravidae with twin pregnancies have a high incidence of premature labour in spite of hospital admission for rest.

Complications, such as death of one fetus, transfusion of blood from one twin to the other, abnormality and a higher incidence of hydramnios, are more common in monochorionic twins. There is also a greater perinatal loss in this group.

FINDINGS

During 1970, 33 perinatal deaths occurred in multiple (all twin) pregnancies. In 10 pregnancies both babies died, and in 13 further pregnancies one baby died. One in 14 perinatal deaths therefore occurred in a multiple birth during 1970, showing that the perinatal loss in twin pregnancies is considerably greater than in single births.

Table LXXXVII shows the clinical classification of these perinatal deaths.

TABLE LXXXVII.

(a) 20 perinatal deaths where both babies in 10 pregnancies died.

P.U.	15
APH	2
M.D.	2 - mother had haematemesis at 36 weeks gestation.
F.D.	1 - anencephalic.

(b) 13 perinatal deaths where one baby in a twin pregnancy died.

P.U.	10
APH	1 - hypertension -- abruption.
F.D.	1 - hydrocephalus.
Other (Rh.)	1

The patient with Rhesus incompatibility was admitted to hospital, because of this problem, so that amniocentesis could be carried out. A twin pregnancy was not diagnosed. This patient was para 3 and had two previous stillbirths. The membranes ruptured spontaneously at 34 weeks gestation and because of the presence of meconium stained liquor and her previous history, a Caesarean Section was carried out. One baby was macerated but the other survived.

Table LXXXVIII illustrates the age, parity and social class characteristics of the 23 mothers.

TABLE LXXXVIII.

Maternal Characteristics.

	<u>Age.</u>		<u>Parity.</u>		<u>Social Class.</u>			
	1	2	1	2	1	2		
< 20 years	2	2	0	5	3	I	0	0
20-29 years	8	4	1	4	3	II	0	0
30-39 years	0	5	2	0	4	III	3	3
40 & over	0	2	3+	1	3	IV	3	5
						V	4	3
						N/S	0	2

1 = Both babies died.
2 = One baby died.

The mothers who lost both babies were younger and of lower parity than the mothers who lost one baby.

There was also a difference in the gestation length of the pregnancies (Table LXXXIX).

TABLE LXXXIX.

Gestation Length of Twin Pregnancies.

	- 30 weeks.	31-33 wks.	34-36 wks.	37-39 wks.	40 wks +
1.	7	2	1	0	0
2.	0	2	1	6	2

1 = Both babies died,
2 = One baby died.

Table LXXXIX shows that the gestation length is much shorter in the twin pregnancies where both babies die.

Table XC illustrates the antenatal complications by clinical cause.

TABLE XC.

Antenatal Complications

	P.U.	APH	M.D.	F.D.	Other
Antenatal Admission	12	2	1	2	1
Hydramnios	6	0	0	1	0
Premature Rupture of Membranes	0	0	0	1	1

In 12 of the 18 multiple pregnancies where the main cause of death was premature - cause unknown, the patients were admitted to hospital because twin pregnancy was suspected. Six patients had hydramnios. It is interesting to note that in none of these 18 patients did spontaneous rupture of membranes occur.

In the 13 cases where only one twin died, in 6 cases it was the first twin and in 7 cases the second twin died.

The time of death of the twins is shown in Table XCI.

TABLE XXI.

Time of Perinatal Death

(a) One Baby Died.

<u>Twin I.</u>	<u>Twin II.</u>
1. S.B. Macerated.	1. S.B. Macerated.
2. S.B. Macerated.	2. S.B. Macerated.
3. S.B. Macerated. Rh incompatibility.	3. S.B. Macerated.
4. S.B. F.H. stopped in 2nd stage.	4. S.B. Macerated.
5. 2nd day Asphyxia. APH.	5. S.B. Macerated.
6. 6th day - Kernicterus.	6. S.B. Anencephalic.
	7. 7th day - Respiratory Distress, Septic Portal Vein Thrombosis.

(b) Both Babies Died.

	<u>Twin I.</u>	<u>Sex.</u>		<u>Twin II.</u>	<u>Sex.</u>
1.	1st Day	F.		1st Day	F
2.	1st Day	F		1st Day	F
3.	1st Day	F		5th Day	F
4.	S.B.	M		S.B.	M
5.	S.B.	M		3rd Day	M
6.	1st Day	M		S.B. Macerated	M
7.	1st Day	M		S.B.	M
8.	1st Day	M		S.B.	M
9.	1st Day	M		1st Day	M
10.	1st Day	M		1st Day	M

DISCUSSION

The findings from this small series of perinatal deaths in multiple pregnancies are very similar to those in the British Perinatal Survey in most respects. The twin deaths fell into two well-defined categories (a) where both babies die, and (b) where only one baby dies. Much has been written about the problems of the second twin and how mortality is greater in the second twin (Guttmacher and Kohl, 1958; Law, 1967; Butler and Alberman, 1969). All these studies included a large number of cases and this finding was not borne out in

this small group (Table XCI). Of the 13 babies that died, 6 were the first twin and 7 the second twin.

In both groups of twin pregnancy, by far the commonest cause of perinatal death was premature - cause unknown. The mothers who lost both babies delivered early in the third trimester and were younger and of lower parity than those who lost one baby (Tables LXXXVIII and LXXXIX).

The Editorial Team in "Perinatal Problems" (1969) state in their discussion on twin deliveries where both babies die "It is, however, unfortunate that the factors which seem to contribute to this risk, namely low maternal age, monozygosity, and particularly male:male combinations, are also those which can least be remedied." In this small series where both twins died they were each of the same sex and there were more male pairs than female pairs (7 to 3). The importance of rest is stressed in the young primipara in an attempt to prevent premature labour. In the 10 patients who lost both babies, only one patient was seen at the hospital antenatal clinic before 12 weeks, two before 20 weeks, 6 were seen after 20 weeks, and one received no care at all.

In the 13 patients who lost one twin, the fact that they were having twins was not diagnosed in two patients, one patient had a Caesarean Section because of Rhesus incompatibility, and the other was X-rayed in labour because of hydramnios and the twins were then discovered. There were 10 stillbirths in this group, 9 being macerated (Table XCI). Only 6 patients were admitted to hospital for rest because they had a twin pregnancy. One was a primigravida and two women were having their fourth or subsequent child. They were not, however, admitted until the 32nd week of pregnancy. If bed rest is going to be effective at all, it is important that the patient be admitted at 28 weeks gestation when the risk of premature labour is high.

Diagnosis of a twin pregnancy can now be made very early in pregnancy, with

the help of ultrasound. As soon as this is suspected, therefore, ultrasonic examination ought to be made and if the suspicion of twin pregnancy is confirmed, close watch can be kept on the patient throughout her pregnancy.

Early diagnosis of twin pregnancy, close supervision during pregnancy, and an attempt to prevent premature labour ought to improve the perinatal outcome in twin pregnancies.

CHAPTER IX.

DOMICILIARY DELIVERIES.

One of the findings of the British Perinatal Mortality Survey was that the evidence there presented seemed to show beyond doubt that many perinatal deaths could be prevented by a higher standard of obstetric care, meaning ready access to specialist care, and more beds in specialist hospitals. It is now possible for all patients in the City of Glasgow to be delivered in hospital if they so desired, not because more obstetric beds have been created but because the length of time the patient stays in hospital has been reduced. Illsley (1967) has pointed out, however, that the differential distribution of obstetric care is influenced by many factors other than the existence of hospital beds, and regrettably, in some parts of this country and other countries the ability of the richer sections of the community to pay for additional care and skill. He states that "many factors combine to prevent an allocation of resources ideally suited to obstetric needs; continuous redistribution of population, the slowness of building programmes in adapting to change or newly perceived needs, the concentration of services in highly urbanised areas and teaching hospitals and the inability of isolated and poor communities to attract the requisite quantity and quality of professional staff. However, the high proportion of domiciliary confinements among lower social groups and high parity mothers often represents not external medical selection but patient preference stemming from tradition." This final sentence is very relevant to the domiciliary

deliveries in the City of Glasgow. Smith and Macdonald (1965) showed that there was not a very efficient selection of cases for hospital delivery and a number of primigravid patients and those of high parity were being confined at home, along with a considerable number of patients with a poor obstetric history. Richards et al. (1979), in a study of the use of maternity care in Glasgow, found from an analysis of all births in 1967 that the overall hospital confinement rate was 84.5 per cent. Ninety-four per cent of primigravid patients were delivered in hospital, whereas only 75.5 per cent of women having a fourth or subsequent baby were. This latter figure had risen to 81 per cent two years later (M.O.H. Report, City of Glasgow, 1970). It is, however, obvious that a number of patients still prefer to be delivered at home. If this is to take place, selection of patients suitable for domiciliary delivery must be at a very high level. The following risk categories have been suggested by the Montgomery Report (1959):

- (1) Low Risk: Age 20 to 30 years with 2nd or 3rd pregnancy and no previous obstetric complication.
- (2) Medium Risk: Age under 20 years, age 20 to 34 years with previous obstetric complication. Primigravida under 30 years.
- (3) High Risk: 4th or subsequent pregnancy. Age 35+ years. Primigravida aged 30+ years. All cases with a bad obstetric history (e.g. AFI or stillbirth) or neonatal death.

Using these risk categories a study of the domiciliary deliveries occurring during the first six months of 1970 was carried out by the Social Paediatric Research Unit and the findings were reported in the Report of the Medical Officer of Health for the City of Glasgow for the year 1970.

They found that during that six-month period 54 per cent of the 361 patients booked for home confinement fell into the high risk category. Selection of cases suitable for delivery at home, therefore, remains unsatisfactory.

Details of Domiciliary Perinatal Deaths from the Survey:

Details of Domiciliary Perinatal Deaths from the Survey:

During 1970 there were 913 domiciliary deliveries in the City of Glasgow.

Thirty-four of these babies were either stillborn or died during the first week of life, giving a perinatal mortality of 37.3 per thousand total births. Of these 34 births, only 9 were actually booked for home confinement, 13 were booked for hospital delivery, and 12 received no care and were therefore unbooked.

Table XCII illustrates the number of deaths by clinical cause, and Table XCIII illustrates the age, height, parity and social class distribution of the mothers.

TABLE XCII.

Domiciliary Perinatal Deaths
by Clinical Cause

<u>Classif- ication.</u>	<u>No.</u>	<u>Booked for Home.</u>	<u>Booked for Hospital.</u>	<u>Unbooked.</u>
M.U.	7	5	0	2
P.U.	10	0	4	6*
Tx.	3	0	1	2
Tox.	0	0	0	0
APH	1	0	1	0
M.D.	0	0	0	0
F.D.	12	4	7	1
Other	1	0	0	1
Total	34	9	13	12

* = One set of twins.

TABLE XCIII.

Domiciliary Perinatal Deaths - Maternal
Characteristics

		Booked for Home.	Booked for Hospital	Unbooked.
<u>Age:</u>				
< 20 yrs.	3	0	1	2
20-29 years	19	5	6	8*
30-39 years	9	3	5	1
40 & over	2	1	0	1
Not stated	1	0	1	0
Total	34	9	13	12
<u>Height:</u>				
0 - 5'1"	12	3	5	4
5'2" - 5'4"	16	6	7	3*
5'4" & over	4	0	1	3
Not stated	2	0	0	2
Total	34	9	13	12
<u>Parity:</u>				
0	7	0	1	6*
1	6	1	4	1
2	5	2	3	0
3+	16	6	5	5
Not stated	0	0	0	0
Total	34	9	13	12
<u>Social Class:</u>				
I	1	1	0	0
II	1	0	1	0
III	18	7	5	6
IV	9	0	5	4*
V	4	1	2	1
Not stated	1	0	0	1
Total	34	9	13	12

* = One set of twins.

As the number of patients in each group is small, only general points can be made.

Booked for Home Confinement:

Of the 9 patients booked for confinement at home, only three would be considered suitable by the age, parity, height and previous history criteria. It is of interest that there are only two causes of death in this group - fetal deformity (4 cases) and mature - cause unknown (5 cases). The details of the patients are given in Table XCIV.

TABLE XCIV.

Details of Patients booked for Home Confinement.

No.	Classification of Death.	Age.	Parity.	Social Class.	Gestn. Length.	Birth Weight.
1.	F.D. (Anencephalic)	25	3+1	III	34	1474
2.	F.D. (Anencephalic)	27	1+0	V	34	1814
3.	F.D. (Spina Bifida + Hydrocephalus)	40	9+1	III	37	3629
4.	F.D. (Congenital Heart Disease)	26	2+0	III	41	3629
5.	M.U.	31	4+0	III	37	3402
6.	M.U.	32	2+2	III	39	2722
7.	M.U.	34	5+0	III	40	3157
8.	M.U.	29	2+0	I	40	4082
9.	M.U.	29	3+0	III	40	4309

At the present time fetal deformity cannot be detected early in pregnancy by a routine procedure, so these tragedies will continue to occur in domiciliary as in hospital practice. One patient had had an X-ray because of hydramnios three days prior to delivery of an anencephalic fetus at home. The delivery took place before the result of the X-ray was available. Two other babies had gross central nervous system abnormalities and the fourth child had inoperable congenital heart disease, dying a few days after delivery in a neonatal paediatric unit.

All the women who lost their babies from the classification mature - cause unknown, were under 35 years of age. None of the pregnancies were more

than 40 weeks gestation, but three of the women were having a fourth or subsequent child, which ought to have been an indication for hospital delivery (for one woman it was a particularly important pregnancy as her first four children had been burned in a house fire, but she refused hospital confinement). Another patient had suffered from "depression" during her pregnancy requiring treatment with Librium.

From the midwives' notes, which are not very detailed and maternal weight was not recorded, there was no indication that anything was going wrong with these five pregnancies before fetal death occurred except in one case where the membranes ruptured 30 minutes before delivery and there was meconium stained liquor. The fetal heart rate was said to be satisfactory.

Booked for Hospital Delivery:

Of the patients who were booked for delivery in hospital, but in fact were confined at home, the main cause of fetal death was due to fetal deformity (7 cases). There were 4 deaths due to premature - cause unknown, and one each in the trauma and antepartum haemorrhage groups. All the patients had attended their general practitioners and the hospital for antenatal care, although the gestation at which they first attended is not known as this fact was not recorded on the domiciliary midwife's records. The reason why the patients did not reach hospital is not known. The length of labour was not recorded.

The relevant details of these patients are shown in Table XCV.

TABLE XCV.

Details of Patients Booked for Hospital Confinement

No.	Classification.	Social Class.	Parity.	Gestn. Length.	Birth Weight.
1.	F.D. (Anencephalic)	V	1+0	38	907
2.	F.D. (Anencephalic)	V	1+1	?	Unknown
3.	F.D. (Alimentary)	IV	2+0	33	2070
4.	F.D. (Cardiac)	IV	1+0	41	3160
5.	F.D. (C.N.S.)	IV	2+0	36	450
6.	F.D. (Anencephalic)	III	1+0	28	907
7.	F.D. (C.N.S.)	II	0+0	34	1400
8.	P.U.	IV	3+	28	Unknown
9.	P.U. (5 lb. Death due to inadequate resuscitation)	III	1+	34	2268
10.	P.U. (2041 g. Noted to be "small-for-dates" at hospital antenatal clinic)	III	7+	38	2041
11.	P.U. (Previous babies all weighed less than 5 lb.)	III	5+	28	1220
12.	Tr. (Breech)	IV	3+0	40	2835
13.	APH (Other)	III	7+4	32	1334

In five of the 7 cases where death was due to fetal deformity the abnormality was in the central nervous system. Six of the cases of deformity occurred in women of Social Classes IV and V and they all occurred in women of low parity.

A breech delivery occurred in a full term infant booked for hospital delivery, but labour was so rapid that transfer to hospital could not be carried out in time. This was classified as a traumatic delivery.

Two of the four patients who delivered babies whose deaths were classified as premature - cause unknown, had babies that by their weight at least ought to have survived. The uterine size in one of the patients was noted on the hospital antenatal record to be "small for dates," but no

further action was taken.

The membranes were stated to have ruptured five days before delivery of a stillborn child showing signs of early maceration. Another patient whose five previous babies all weighed less than 2500 g. had been seen by her doctor the day before delivery at 28 weeks gestation, because of abdominal pain, which was thought to be due to a urinary tract infection. She was treated with an antibiotic and delivered at home a few hours later.

Unbooked Patients:

Thirteen babies (including one set of twins) were born to mothers in this group. Two patients had attended their general practitioners but no arrangements had been made for their delivery. The other ten patients received no antenatal care.

Table XCVI illustrates the relevant details of these babies.

TABLE XCVI.
Details of Unbooked Patients.

No.	Classification.	Marital Status.	Parity.	Gestation Length.	Birth Weight.	Remarks.
1&2	P.U.	P.N.C. Married 6 wks. before delivery.	0+0	31	850 1190	Twins.
3.	P.U.	Single	0+0	28	1106	
4.	P.U.	Single	0+0	28	1474	
5.	P.U.	Single	4+0	?	1361	Previous LUSCS.
6.	P.U.	Married	4+0	34		
7.	M.U.	Single	0+0	40	2500	Delivered by mother.
8.	M.U.	Married	3+0	?35	2722	Requested termination at 13 weeks - refused.
9.	Tr.	Single	0+0	?	Unknown	Breech.
10.	Tr.	Single	1+0	32	2044	Breech.
11.	F.D.	Married	7+0	39	Unknown	
12.	Rh Incompatibility	Separated	4+0	38	2155	Two previous stillbirths.
13.	Other (Cause Unknown)	Single	?	?	820	

The most striking common factor in this group of patients is their marital status. Seven patients were single, one was separated, and the mother of the twins was married six weeks before the babies were born. The three married women had large families.

In theory a number of these deaths would not have occurred if the mothers had received adequate antenatal care. The two mature - cause unknown deaths occurred in good sized babies, but there was no trained person present at their deliveries. One of the mothers sought a termination of pregnancy, but this was not recommended by the Psychiatrist she saw during the first trimester. She did not come for or receive ^{any} antenatal care.

The small babies and those delivered by breech would have had a greater chance of survival if they had been born in hospital with good neonatal facilities available.

One patient had had two stillbirths because of Rhesus incompatibility and yet did not attend for antenatal care. She was separated from her husband and presumably her social problems contributed to her lack of care.

DISCUSSION

The patients who were confined at home were a heterogeneous group. Those patients who were booked for home confinement and who lost their babies were nearly all "high risk" patients and should not have been delivered at home. Those patients who were booked for hospital delivery were only confined at home by "accident." Apart from giving the patients greater understanding of labour and thereby ensuring their coming into hospital as soon as the contractions started, these tragedies at home cannot be prevented.

The unbooked patient presents a much more serious problem. She usually has immense social problems, frequently is single, and one may presume she is often reluctant to face the reality of her pregnancy to the extent required to make

preparation for her confinement. It is probably the complete lack of care during her pregnancy that contributes to the perinatal loss as much as being delivered at home. Illsley (1967), discussing the problems of illegitimate pregnancy, writes that "it is clear that the social influences operating in an out-of-wedlock conception are not only complex but relevant to the clinical course of the pregnancy. Maternal social status and physical stature, stress, possible attempts to abort, delayed antenatal care, poor living conditions, etc. add up to a complete aetiological situation which requires equally complex analytical methods."

Beard (1969), writing in "Perinatal Problems," points out that although differences of opinion still exist about the advantages of a hospital confinement there is no doubt that the risk of perinatal death is lower in a well equipped and well staffed hospital than at home. He has shown that although no direct attempt has been made to persuade women to have their babies in hospital except where there were obvious medical indications, almost a hundred per cent hospital delivery rate has been reached and good perinatal results have been achieved in Aberdeen by a high standard of obstetric care, although the women are not so tall and so healthy as the women in other areas of the country. He states that "the high technical standards available in hospital have resulted in a perinatal death rate well below the national average. It is for society to decide how far it wishes to go in ensuring that the risks are reduced to a minimum."

The excellent results obtained in Holland where the domiciliary delivery rate is very high are frequently quoted in the argument that the high incidence of hospital confinements is not necessary. The risks of a complicated delivery are, of course, less where mothers are well grown and healthy, and this is why a maternity service based primarily on domiciliary confinement is so successful in Holland (Beard and Thomson, 1969). The incidence of fetal deformity is lower in Holland than Scotland, so perhaps if more patients were delivered in hospital the

perinatal mortality rate might even be lower than it is.

While women must still be left with the right to have a domiciliary delivery if they so wish, rigorous attention must be paid to the risk factor in assessing whether a woman is suitable for domiciliary confinement, and proper advice must be given and risks explained to the women in high risk categories who wish to be confined at home. General practitioners who undertake obstetric practice must be properly trained to deal with all types of obstetric emergency and carry emergency equipment with them at all times.

Proper selection of patients for domiciliary delivery and quick referral to hospital as soon as complications develop would help to reduce the number of perinatal deaths in this group of women.

CHAPTER X.

PERINATAL MORTALITY RATE BY PLACE OF CONFINEMENT.

Owing to the size of the City of Glasgow there are a number of centres where patients may be delivered. There are 9 maternity hospitals, 3 general practitioner units, and 2 nursing homes. The problems encountered by each unit are different, depending partly on which area it draws its patients from and partly on what the particular interests of the obstetricians are. During the early days of intra-uterine transfusion, for example, the procedure was only undertaken by a few obstetricians. The severe cases of Rhesus isoimmunisation were sent to these obstetricians and, not surprisingly, the perinatal mortality due to Rhesus isoimmunisation tended to be greater in their hospitals owing to the number of severe cases they saw.

Because of these differences it is not possible to compare directly one hospital's perinatal outcome with that of another hospital. What is important, however, is for each hospital to look at its own perinatal mortality, to see where its specific problems lie in the area it serves, and to see what improvements could be made. The object of looking at the perinatal mortality rates by place of confinement was therefore to illustrate the different problems that the different maternity centres face. The time interval between the patient being referred to hospital and being seen at the clinic is also relevant as it illustrates (1) the type of patient attending for care, whether she comes early in her pregnancy or not, and

(2) the hospital delay time before the patient is seen.

ILLUSTRATIONS

The information obtained for the perinatal deaths only covers hospital and domiciliary deliveries. Table XVII illustrates the number and percentage of perinatal deaths by clinical cause and place of confinement.

TABLE XVII.

Perinatal Deaths by Place of Confinement and Clinical Cause.
(436 cases and 1 unknown)

Classification.	Domiciliary Delivery	Hospitals								
		1	2	3	4	5	6	7	8	9
<u>Number of Deaths</u>										
H.U. (50)	7	2	4	2	4	12	1	2	8	9
P.U. (101)	10	4	7	11	4	16	0	22	13	14
Tr. (23)	3	0	0	4	1	3	0	9	3	0
Tox. (29)	0	0	1	6	0	2	0	12	6	2
APH (94)	1	1	11	11	2	11	0	35	9	13
M.L. (12)	0	1	0	1	1	0	0	6	1	2
F.L. (105)	12	1	7	9	8	6	0	30	14	18
Other (22)	1	0	7	5	1	1	0	7	0	0
Total (437)	34	9	37	49	21	51	1	123	54	57
<u>Percentage Deaths by Place of Confinement</u>										
H.U. (50)	20.6	22.2	10.8	4.1	19.0	23.5	100.0	1.6	14.8	14.0
P.U. (101)	29.4	44.4	13.9	22.4	19.0	31.4	0.0	17.9	24.1	24.0
Tr. (23)	8.8	0.0	0.0	8.2	4.8	35.9	0.0	7.3	5.6	0.0
Tox. (29)	0.0	0.0	2.7	12.2	0.0	3.9	0.0	9.8	11.1	3.5
APH (94)	2.9	11.1	29.7	22.4	9.5	21.6	0.0	28.5	16.7	22.3
M.L. (12)	0.0	11.1	0.0	2.0	4.8	0.0	0.0	4.9	1.9	3.5
F.L. (105)	35.3	11.1	13.9	13.4	33.1	11.8	0.0	24.4	25.9	31.0
Other (22)	2.9	0.0	13.9	10.2	4.8	2.0	0.0	5.7	0.0	0.0
Total (436)	99.9	99.9	99.9	99.9	100.0	100.1	100.0	100.1	100.1	100.0

Tables XCVIII and XCIX illustrate the gestation length at the time of referral, and at the first hospital visit, by each clinical cause in hospital deliveries only.

TABLE XCVIII.

Gestation Length at Referral (Hospital deliveries only, 395* patients)

	Total.	< 10 wks.	10-19 wks.	20-29 wks.	30+ wks.	N/S
M.U.	43	5	11	7	2	18
P.U.	85	7	24	8	1	45
Tr.	20	4	9	1	0	6
Tox.	29	7	13	2	0	7
APH	92	8	21	13	1	49
M.D.	11	1	4	1	0	5
F.D.	93	15	38	10	0	30
Other	22	6	6	2	1	7
Total	395	53 (13.4%)	126 (31.9%)	44 (11.1%)	5 (1.3%)	167(42.3%)

TABLE XCIX.

Gestation Length at 1st Antenatal Visit (395 patients).

	Total.	< 10 wks.	10-19 wks.	20-29 wks.	30+ wks.	N/S.
M.U.	43	2	11	17	4	9
P.U.	85	4	24	26	5	26
Tr.	20	2	9	5	1	3
Tox.	29	5	11	10	0	3
APH	92	2	21	20	8	41
M.D.	11	1	3	4	1	2
F.D.	93	1	41	23	6	22
Other	22	1	9	9	2	1
Total	395	18 (4.6%)	129 (32.7%)	114 (28.9%)	27 (6.8%)	107 (27.1%)

* This number = total number of patients - 34 domiciliary deliveries + 8 (where both twins died of same clinical cause).

Table C shows the time interval before the patient is first seen at hospital (single births only).

TABLE C.

Time Interval from Referral to First Antenatal Visit by Hospital of Delivery. (369 cases).

Hospitals.	No.	<3 wks.	3-4 wks.	5-6 wks.	>6 wks.	N/K.
1.	8	1	0	0	1	6
2.	34	1	0	0	16 (47.1%)	17
3.	49	9	25	1	0	14
4.	20	0	4	4	7 (35.0%)	5
5.	48	7	14	7	0	20
6.	1	0	0	0	1	0
7.	107	5	20	12	25 (23.4%)	45
8.	49	5	6	11	3 (6.1%)	24
9.	53	4	15	7	4 (7.5%)	23
Total	369	32 (8.7%)	84 (22.8%)	42 (11.4%)	57 (15.4%)	154 (41.7%)

Hospitals 3 and 7 contain the Departments of Midwifery and of Obstetrics of the University of Glasgow (Table XCVII). It is very interesting to note that in these two hospitals the perinatal mortality from the cause, mature - unknown, is very much lower than that in other hospitals. In hospital 5 there were 12 deaths in the mature - cause unknown group. These cases are discussed in detail in Chapter V(a), but it is worth while noting that this hospital serves a very poor area of the city, and it is situated some distance from the housing schemes which it serves. A number of these patients were of high parity, attended infrequently for antenatal care, and intra-uterine death occurred before term. These findings point to the fact that these patients require intensive supervision during their pregnancy. In an attempt to improve the antenatal care and hopefully the perinatal outcome in this area, an antenatal clinic is being started in the heart of the housing scheme in a tenement building. It is hoped that by being near the patients' homes the patients will attend more regularly and earlier in their pregnancies.

It is proposed to hold Child Welfare clinics in conjunction with the antenatal clinic.

Hospitals 1, 2, 4, 5 and 6 do not have intensive care paediatric units attached to them. This may contribute to the differing rates in the premature - cause unknown deaths, although this is a complex group, and there is a large environmental component. The varying percentage of deaths from antepartum haemorrhage depends partly on the type of hospital (1 and 6 are attached to 2 and 7 and tend only to have normal deliveries) and partly on whether the hospital gives Flying Squad service.

Twelve per cent of patients were not referred to the hospital antenatal clinic until the second half of pregnancy (Table XCVIII), and 36 per cent of patients were not seen at hospital until 20 weeks or later (Table XCIX). This delay in antenatal attendance is in some cases due to poor administration at the booking clinic of some hospitals. This fact is illustrated in Table C. In a number of patients the referral time is not known (this group includes emergency admissions with no record of prior antenatal attendance), so that statistical analysis is not meaningful. Trends, however, can be seen and it is quite incredible that whereas one hospital saw 34 of its 35 patients within the first month of booking, another hospital saw 16 of its 17 patients where time of referral was known after six weeks' delay.

DISCUSSION

During the period of study it would appear that there is considerable room for improvement in the organisation of appointments in antenatal clinics. General practitioners are not going to be encouraged to refer their patients early in pregnancy, neither are patients going to be eager to attend, if there is this long delay. With the problems of assessing gestation length it is

frequently stressed how important it is to see the patient during the first trimester. If the obstetricians believe this to be true, they must see that patients are seen then, and do not have to wait weeks for an appointment.

There are a number of factors already mentioned which contribute to differing perinatal mortality rates from hospital to hospital. It is possible, too, however, that differing standards of obstetric practice exist and perhaps the findings shown in Table C indicate this.

CHAPTER XI.

HOUSING CONDITIONS.

Environmental influences affect perinatal outcome in two ways.

Firstly, the environment in which the mother grew up will determine whether she reaches her genetic potential, thus determining whether or not her physiological response to pregnancy will be impaired. The more immediate environment in which the mother lives during her pregnancy also influences the outcome of the pregnancy.

As Baird (1969) points out in the opening chapters of "Perinatal Problems," "A poorly educated mother, brought up in a poor environment, is likely to marry and begin childbearing at a relatively early age, and to have more and more closely-spaced children than one whose upbringing leads her to plan her family with an eye to economic security in the future."

The following chapter deals in some detail with the geographical distribution of perinatal deaths in Glasgow, showing the problems of poor housing in the city and tracing the historic reasons for this. This chapter will deal with the ethnic origin of the women losing their babies and the actual conditions in the home.

Society is becoming increasingly aware of the existence and problems of social deprivation (Wedge and Prosser, 1973; Townsend, 1972). Although heredity is obviously important, it is becoming increasingly apparent that the beginnings of social deprivation lie in environmental influences both within the

family and outside it. These influences play a part in the development of a child, his attainments, personality and behaviour. In this study of perinatal deaths these facts are obviously not all relevant as only the child in utero will be affected, but these perinatal deaths are only the tip of the iceberg and there is considerable morbidity due to environmental influences in the surviving children.

Butler (1974), reporting on the late postnatal consequences of fetal malnutrition, summarises the findings as follows:

"(1) In a National Survey of 17,000 births, important maternal determinants of low birth weight and thus of fetal nutrition were severe pre-eclampsia, multiparity, cigarette smoking after 20 weeks and low stature (itself an index of maternal pre-pregnancy nutrition). These four factors also explained the lower birth weight found with adverse social class and maternal age.

"(2) Smoking during the second half of pregnancy lowered mean birth weight by 180 g., increased perinatal mortality by 30 per cent, and on follow-up at age 7 years "depressed" reading ability, height, and social adjustment by a small but significant amount; all these adverse effects persisted after allowance for age, parity, social class, and other environmental factors.

"(3) Infants with birth weights below the 5th percentile for week of gestation, regardless of cause, showed poorer overall performance in a number of educational and physical tests at 7 years of age. The degree of impairment was low in first born children of professional parents but high in later born children of low class."

In his report "Family Circumstance and Location of the Socially Deprived," Res (1974) states that "Depriving or inadequate housing conditions such as the

lack of amenity in the home are largely relative, dependent on what national housing standards are prevalent at one point in time, others are perhaps more social and economic in content. The stability of the structure of the house, the freedom from damp, adequate lighting and ventilation, the supply of hot water, the exclusive use of a W.C. etc., are measured against standards laid down in the Housing (Scotland) Act that are considered to be tolerable. None of the Corporation housing stock should be lacking in these basic amenities, although the standard of fittings in some categories of the older houses may be less than in a reasonable condition. If social deprivations exist in Corporation housing they are more likely to be found in the social/economic effects of overcrowding, poor general external environment, and in the "enforced" resettlement of families as a result of demolition or clearance orders, rather than the physical conditions within the home."

As there are no accepted formal conditions, the social/economic factors in housing leading to deprivation are difficult to measure. Overcrowding, however, can be measured. There are two measures of overcrowding covering Britain as a whole which are applicable to Scotland, statutory overcrowding and census overcrowding. Rae describes this in detail in his report. Two tables are used in the 1944 housing standard in assessing the number of habitable rooms; one balances the permitted number of persons against the number of habitable rooms, the other balances the permitted number of persons against the floor area of rooms. The relevant table is the one which results in the lower number of occupants. Adults and children of ten years and older are counted as one, children between one and ten years are counted as half, and children below the age of one year are not counted. The permitted number of persons per room ranges from three persons in a two apartment house and seven and a half persons in a four apartment house. Census overcrowding is accepted as more than 1.5

persons per room. The definition of overcrowding found in the Letting Regulations of the Housing Management Department of the Corporation falls between the two. The definition of overcrowding used in the Social Paediatric and Obstetric Research Unit for all Glasgow births is that of census overcrowding. Fifty point four per cent of all births in Glasgow during 1970 were to women living in overcrowded conditions (Sweeney, personal communication).

FINDINGS:

The number of occupants per room and the percentage distribution is shown in Table CI.

TABLE CI.

Occupants per Room by Clinical Cause (Single Births only)

	No.	Occupants per Room								N/S
		0.5	0.5 - 0.75	0.75 - 1	1 - 1.25	1.25 - 1.5	1.5 - 1.75	1.75 - 2	2	
M.U.	50	1	3	2	10	13	4	1	10	6
P.U.	76	0	7	2	13	8	12	3	18	13
Tr.	23	0	2	0	8	2	3	0	6	2
Tox.	29	0	6	1	7	2	2	1	5	5
APH	91	1	4	0	15	18	17	2	20	14
M.D.	10	0	0	0	1	2	3	0	3	1
F.D.	103	1	9	0	25	13	19	4	16	16
Other	22	0	1	1	6	3	4	1	5	1
Total	404	3	32	6	85	61	64	12	83	58

~~~~~  
Overcrowding.

Percentage

|              |            |            |            |             |             |             |            |             |             |
|--------------|------------|------------|------------|-------------|-------------|-------------|------------|-------------|-------------|
| M.U.         | 2.0        | 6.0        | 4.0        | 20.0        | 26.0        | 8.0         | 2.0        | 20.0        | 12.0        |
| P.U.         | 0.0        | 9.2        | 2.3        | 17.1        | 10.5        | 15.8        | 3.9        | 23.7        | 17.1        |
| Tr.          | 0.0        | 8.7        | 0.0        | 34.8        | 8.7         | 13.0        | 0.0        | 26.1        | 8.7         |
| Tox.         | 0.0        | 20.7       | 3.4        | 24.1        | 6.9         | 6.9         | 3.4        | 17.2        | 17.2        |
| APH          | 1.1        | 4.4        | 0.0        | 16.5        | 19.8        | 18.7        | 2.2        | 22.0        | 15.4        |
| M.D.         | 0.0        | 0.0        | 0.0        | 10.0        | 20.0        | 30.0        | 0.0        | 30.6        | 10.0        |
| F.D.         | 1.0        | 8.7        | 0.0        | 24.3        | 12.6        | 18.4        | 3.9        | 15.5        | 15.5        |
| Other        | 0.0        | 4.5        | 4.5        | 27.3        | 13.6        | 18.2        | 4.5        | 22.7        | 4.5         |
| <b>Total</b> | <b>0.7</b> | <b>7.9</b> | <b>1.5</b> | <b>21.0</b> | <b>15.1</b> | <b>15.8</b> | <b>3.0</b> | <b>20.5</b> | <b>14.4</b> |

The overall overcrowding percentage was 59 per cent of all perinatal deaths.

This is very similar to the percentage of 50.4 for all Glasgow deliveries.

The percentage overcrowding varies with clinical cause (Table CII).

TABLE CII.

Overcrowding by Clinical Cause

|              | No.        | Percent.    |
|--------------|------------|-------------|
| M.U.         | 15         | 30.0        |
| P.U.         | 33         | 43.4        |
| Tr.          | 9          | 39.1        |
| Tox.         | 8          | 27.5        |
| APH          | 39         | 42.9        |
| M.D.         | 6          | 60.6        |
| F.D.         | 39         | 37.8        |
| Other        | 10         | 45.4        |
| <b>Total</b> | <b>159</b> | <b>39.3</b> |

Although the lack of facilities is common to many Glasgow households, conditions were worse in women who lost their babies (Table CIII).

TABLE CIII.  
Housing Conditions.

|                          | Prims. | Multip. | Total. | All Glasgow<br>(from Registrar-<br>General's<br>Census, 1972) |
|--------------------------|--------|---------|--------|---------------------------------------------------------------|
| No Hot Water             | 29.3%  | 24.0%   | 25.9%  | 15.9%                                                         |
| No W.C.                  | 20.6%  | 21.6%   | 21.3%  | 11.7%                                                         |
| No Bath                  | 38.0%  | 32.1%   | 34.1%  | 22.3%                                                         |
| No Cooking<br>Facilities | 2.7%   | 2.4%    | 2.5%   |                                                               |

Table CIV shows the ethnic groups. In Glasgow during the 1971 census there were 2,105 women from India and Pakistan living in Glasgow, making up 0.5 per cent of the female population. The perinatal deaths in this group accounted for 1.8 per cent.

TABLE CIV.  
Ethnic Groups.  
(percentage of total in brackets)

|                | 0              | 1           | 2           | 3           | 4           | 5           | 6        | 7           | 8        | N/S           |
|----------------|----------------|-------------|-------------|-------------|-------------|-------------|----------|-------------|----------|---------------|
| M.U.           | 42             | 0           | 1           | 0           | 1           | 0           | 0        | 0           | 0        | 6             |
| P.U.           | 79             | 3           | 1           | 1           | 0           | 2           | 0        | 1           | 0        | 14            |
| Tr.            | 20             | 0           | 0           | 0           | 0           | 0           | 0        | 0           | 0        | 3             |
| Tox.           | 20             | 1           | 0           | 2           | 0           | 1           | 0        | 0           | 0        | 5             |
| APH            | 79             | 1           | 1           | 1           | 0           | 0           | 0        | 0           | 0        | 12            |
| M.D.           | 9              | 1           | 0           | 0           | 0           | 0           | 0        | 0           | 0        | 2             |
| F.D.           | 83             | 1           | 1           | 2           | 0           | 5           | 0        | 0           | 0        | 13            |
| Other          | 20             | 0           | 0           | 0           | 0           | 0           | 0        | 0           | 0        | 3             |
| Total<br>(437) | 352<br>(80.5%) | 7<br>(1.6%) | 4<br>(0.9%) | 6<br>(1.4%) | 1<br>(0.2%) | 8<br>(1.8%) | 0<br>(0) | 1<br>(0.2%) | 0<br>(0) | 58<br>(13.3%) |

- |                       |                    |
|-----------------------|--------------------|
| 0 = Scotland          | 5 = India/Pakistan |
| 1 = England and Wales | 6 = Other Asian    |
| 2 = Ireland           | 7 = Africa         |
| 3 = Other European    | 8 = Other          |
| 4 = Other White       |                    |

## DISCUSSION

The overall findings are that housing conditions in Glasgow are exceedingly poor, particularly with regard to overcrowding where there is a baby, and it has been shown that social deprivation adversely affects the development of the child. The National Child Development Study, unique in the world, is following the progress from birth to maturity of all children in England, Scotland and Wales who were born in the week 3rd to 9th March, 1958. "Born to Fail?" (Wedge and Prosser, 1973) is a report on the striking differences in the lives of British children. It reports on the National Children's Bureau's study of socially disadvantaged children in Britain. The social conditions in which 10,000 national representative eleven-year-olds were growing up and then compared the way of life of the two groups of children, the "disadvantaged" and the "ordinary."

"Disadvantaged" was defined as children who came from a family which had only one parent figure or contained at least five children and had lived in "poor housing" and had a "low income."

The report (Wedge and Prosser, 1973) stated that "One child in 16 was the proportion of disadvantaged among all the children in Britain, but in individual regions the prevalence varied. In Southern England there was only 1 in 47 children. In Wales and Northern England there was 1 in every 12.

"But the most disturbing proportion was found in Scotland, where one in every 10 children was disadvantaged."

Talking about "Born to Fail?" in Glasgow in October, 1974, Wedge said that the proportion of "disadvantaged" children in the Strath Clyde region was about twice that found in England and Wales; Glasgow had between two and four times the prevalence of England and Wales, and a greater prevalence than the rest of Scotland.



1. Victorian tenement block - still inhabited.



2. Rehousing - housing tenement block - with a number of uninhabited flats.

The four photographs included in this chapter illustrate some of the types of houses in Glasgow.

What can be done to improve the situation for the large number of Glasgow citizens living in socially deprived conditions? Rae (1974) suggests in the conclusion to his report "Family Circumstance and the Location of the Socially Deprived" that a working party of representatives of Corporation Departments of Education, Social Work, Housing Management, Planning, the Health Board, and other agencies be formed as a standing "Case Conference" to oversee arrangements for "managing" the resettlement of families at risk. After discussing which cases ought to be studied first, and illustrating lines of research, Rae (1974) concludes by saying "These recommendations have purposely been kept open, if a working party were to be set up much would depend on what could be agreed between the participants. Much would also depend upon willingness. The willingness to accept that the problems of low school attainment, poor health, social inadequacy, emotional poverty, maladjustment, personality disorders, truancy, delinquency and of bad social conditions generally stem from the same background of families deprived of income, of good housing conditions (in the widest sense), and by family size and composition, located in the same well defined areas of physical and social environmental impoverishment, and that it is only when these multiple difficulties are tackled in a collective willingly collaborative way will there be any lasting improvement in the social health of the City."

The problem of poor housing and social deprivation is a multifaceted problem requiring a multidisciplinary approach to improve the present situation in Glasgow. At this time of reorganisation of the health services and of local government, surely the time is ripe for such an approach to be made.



3. **Between-the-wars tenement blocks and a number of modern tower blocks in the distance with an area of waste ground separating the two schemes.**



4. **Post-war tenement block, unkept with broken windows and graffiti in the stairway.**

CHAPTER XII

GEOGRAPHICAL DISTRIBUTION OF PERINATAL DEATHS.

Glasgow, the industrial heart of Scotland and her largest city, stands on the banks of the River Clyde to which it owes part of its development and prosperity. The area of the city is 60.5 square miles and during 1970 the population was 907,672 (M.O.H. Report, City of Glasgow, 1970). At the time of the study the city was divided into 37 municipal wards.

Homelessness and living conditions which destroy or gravely endanger health are very much problems of the medical and social services. Glasgow has severe housing problems and it therefore seems relevant at this point to trace the historic development of housing in Glasgow.

In his "Atlas of Glasgow and the West Region of Scotland," Professor Melvin Howe (1972) of the University of Strathclyde describes the growth of the city, which was determined partly by the course of the River Clyde and partly by its very hilly site. It was between 1660 and 1750 that Glasgow's fortune as a trading city was firmly established. Although the population of the city began to increase with an influx of people from the crofting communities, it was not until the middle of the 18th century that the city extended through its mediaeval boundaries and planned expansion was undertaken. From 1780 to 1830 the population increased five-fold from 40,000 to 200,000 and the main direction of urban development was westward from the old town situated round

the Cathedral. Professor Howe goes on to describe how this phase of rapid growth of the city was governed by the supply of land<sup>and</sup> the attitudes of the landowners. George Square, which is now regarded as the centre of the city, with the City Chambers occupying one side of it, was originally laid out as an area of elegant homes. Further west, the low-priced agricultural land was transformed by a regular pattern of streets. South of the river, similar development took place. Part of the Gorbals at that time was considered to be one of the most desirable dwelling places in the city.

The city boundaries continued to extend. In the first half of the nineteenth century the extensions were mainly to the east and west, whereas in the second half of that century they were to the north and south. Difficulties arose in an acute form in Glasgow through this rapid expansion of population. For while this population, considered geographically, formed one community, it was divided for administrative purposes into Glasgow and its suburbs which were themselves divided<sup>until 1846</sup> into burghs, now represented by the districts of Anderston, Calton, and Gorbals, so at that early period there were four independent local authorities.

During the nineteenth century, the conception that environment could play a considerable part in either causing disease or assisting its spread was only slowly gaining acceptance. In a memorial volume of the writings of J.B. Russell, Medical Officer of Health for the City of Glasgow from 1872 to 1898, entitled "Public Health Administration in Glasgow," a number of statements made by eminent men illustrated the sorry tale of Glasgow during the nineteenth century.

In 1837, Dr. Cowan, Professor of Medical Jurisprudence in the University, wrote that "Many of the causes and the production and propagation of fever must be ascribed to the habits of our population; to the total want of cleanliness among the lower orders of the community; to the absence of ventilation in the more densely peopled districts; and to the accumulation, for weeks or months

together, of filth of every description in our private and public dunghills; to the overcrowded state of the lodging houses resorted to by the lowest classes; and to many other circumstances unnecessary to mention."

In Reports from Assistant Handloom Weavers Commissioners, Parliamentary Paper issued 27th March, 1839, J.C. Symons wrote "These districts (the low districts of Glasgow) contain a motley population, consisting in almost all the lower branches of occupation but chiefly of a community whose sole means of subsistence consists in plunder and prostitution. Under the escort of that vigilant officer, Captain Miller, the Superintendent of the Glasgow Police, I have four times visited these districts, once in the morning and three times at night; I have seen human degradation in some of its worst phases, both in England and abroad, but I can advisedly say that I did not believe until I visited the wynds of Glasgow, that so large an amount of filth, crime, misery and disease existed on one spot in any civilised country. The wynds consist of long lanes, so narrow that a cart could with difficulty pass along them; out of these open the "closes" which are courts about fifteen or twenty feet square, round which the houses, mostly of three storeys high, are built; the centre of the court is the dunghill which probably is the most lucrative part of the estate to the laird in most instances, and which it would consequently be esteemed an invasion of the rights of property to remove. .... In the lower lodging houses, ten, twelve and sometimes twenty persons, of both sexes and all ages, sleep promiscuously on the floor in different degrees of nakedness. These places are generally, as regards dirt, damp, and decay, such as no person of common humanity would stable his horse in.

"Many of the worst houses are dilapidated and in a dangerous state, and are condemned by the Dean of Guild Court, a sentence of which the execution appears to be generally postponed, and which renders these abodes doubly desirable to the occupants as the passing of sentence prevents the levy of rent.

\* \* \* \* \*

"I have visited the parts of Edinburgh likewise, where the lowest portion of the community reside, but not which can be for a moment compared with the wynds of Glasgow exists there. It is my firm belief that penury, dirt, misery, drunkenness, disease and crime culminate in Glasgow to a pitch unparalleled in Great Britain."

These dreadful remarks were followed three years later by some constructive suggestions in 1840 by Dr. Neil Arnott in the Official Report to Poor Law Commissioners.

"Several intelligent inhabitants of Glasgow stated that they were persuaded, if any capitalists would buy the ground of these wynds, and pull down the houses to substitute better houses in wide streets, with good drainage, the increased rental would make the speculation even to them very profitable, while the saving to the community of the cost of supporting the wretched widows and orphans of men who died of the disease generated in the place would exceed the amount of any rent which the property could produce."

By the end of the nineteenth century building regulations had been devised to control the erection of houses so that the primary requirements of lighting and ventilation should be met and the evils arising from the unsuitability and impurity of site and surroundings prevented.

In the excellent paper "Family Circumstance and Location of the Socially Deprived," J.H.Rae describes Local Authority housing policy in Glasgow. He describes how the various Acts of Housing and Town Planning from 1919 onwards were designed to meet the general housing needs of the community, and later specific policies to meet the needs of families made homeless by slum clearance and to house families of four or more people on a limited income. This diversion of housing provision was reflected by a differential rent structure. He states that "ordinary" housing for general housing purposes were let at near "economic" rents, whilst "rehousing" houses were let at a particularly low rental made possible

by the higher rent subsidies available under slum clearance legislation. The third "intermediate" category was introduced for applicants who did not qualify for "rehousing" houses and who could not afford the rents charged for "ordinary" houses. This system of paying different rents for "ordinary," "rehousing" and "intermediate" housing was continued after the war, each being further divided into three, making nine rental groups in all. In 1969, Corporation "scheme" houses were re-allocated for rental purposes into eight Amenity groups and rents divided within each group as to whether a house has been built prior to or after the war. The new re-categorised rent structure followed roughly the old inasmuch as the better "ordinary" housing stock was placed in Amenity Groups 1, 2, 3, and 4, "intermediate" housing spread between Groups 4, 5, and 6, and "rehousing" shared between Groups 7 and 8.

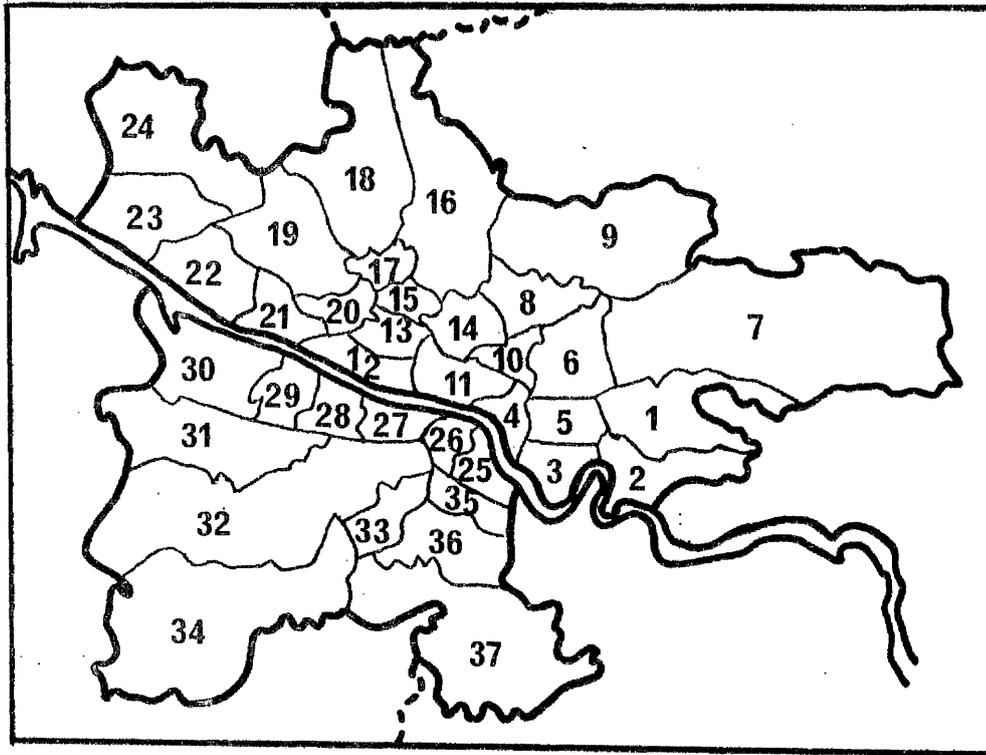
In a previous paper "Housing and Social Deprivation" by Mansley (1973), the instant growth of the east end of the City of Glasgow is analysed. It describes how the area studied could be divided into three zones. These three zones apply to the city as a whole.

(1) The Inner Zone is situated between the city's business centre and the suburbs. This area has been subjected to considerable structural renewal and change and now shows the fragmented mixture of ageing functional, mainly residential, industrial and commercial. This zone contains the bulk of the cheapest, privately owned and privately rented housing correlating with obsolescence and lack of amenity in the home. There is also Corporation housing in the lower reaches of the Rent and Amenity Groups of Housing Management.

(2) The Middle Zone consists mainly of low density pre-war Corporation housing estates of cottages and flatted houses in the higher rent and amenity categories.

(3) The Outer Zone consists of suburbs of higher density post-war Corporation housing, mainly tenements.

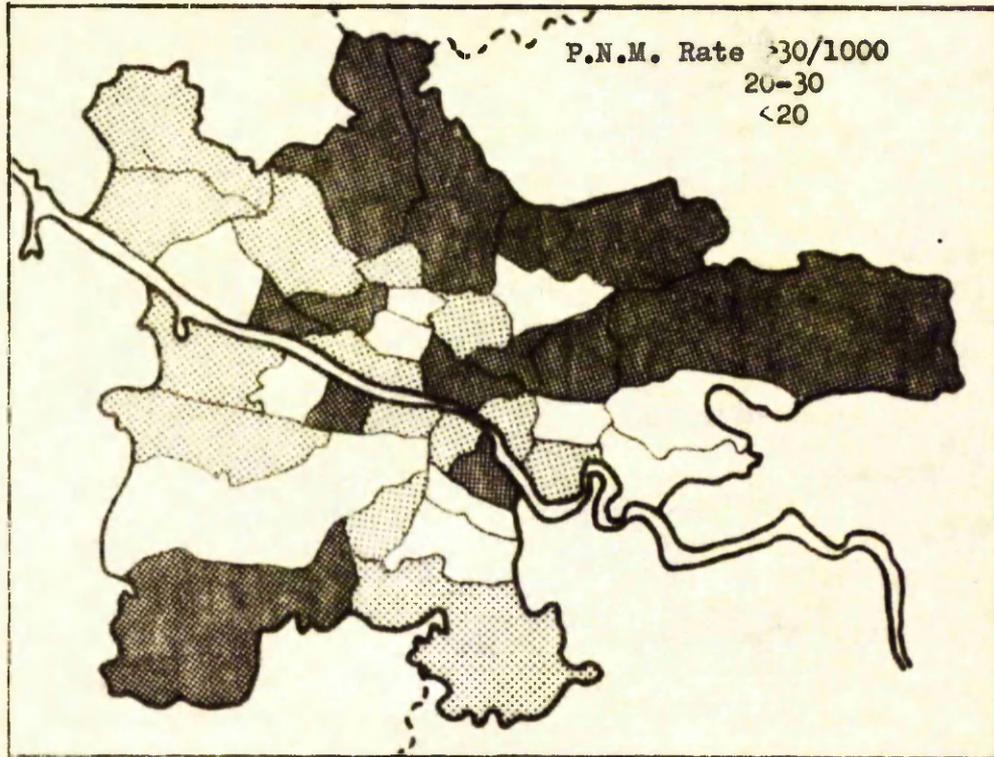
GLASGOW CITY WARDS.



| <u>Name.</u>              | <u>No. Inhab.</u> | <u>Name.</u>      | <u>No. Inhab.</u> |
|---------------------------|-------------------|-------------------|-------------------|
| 1. Shettleston & Tolcross | 676               | 20. Ebbick West   | 310               |
| 2. Parkhead               | 241               | 21. Parkhead West | 317               |
| 3. Dalmeconck             | 615               | 22. Whiteinch     | 269               |
| 4. Calton                 | 304               | 23. Yoker         | 336               |
| 5. Mile-End               | 603               | 24. Knightwood    | 703               |
| 6. Dennistoun             | 444               | 25. Hutchesontown | 281               |
| 7. Provan                 | 1228              | 26. Corbals       | 168               |
| 8. Cowcaddens             | 628               | 27. Kingston      | 235               |
| 9. Springburn             | 481               | 28. Muning Park   | 362               |
| 10. Tomhead               | 263               | 29. Govan         | 504               |
| 11. Buchanan              | 77                | 30. Paisley       | 382               |
| 12. Anderson              | 220               | 31. Craigton      | 347               |
| 13. Park                  | 276               | 32. Pollokshields | 492               |
| 14. Cowcaddens            | 233               | 33. Camphill      | 323               |
| 15. Woodside              | 317               | 34. Pollokshaws   | 715               |
| 16. Rushill               | 593               | 35. Govanhill     | 600               |
| 17. North Kelvin          | 597               | 36. Langside      | 389               |
| 18. Maryhill              | 491               | 37. Galloway      | 895               |
| 19. Kelvinside            | 295               |                   |                   |

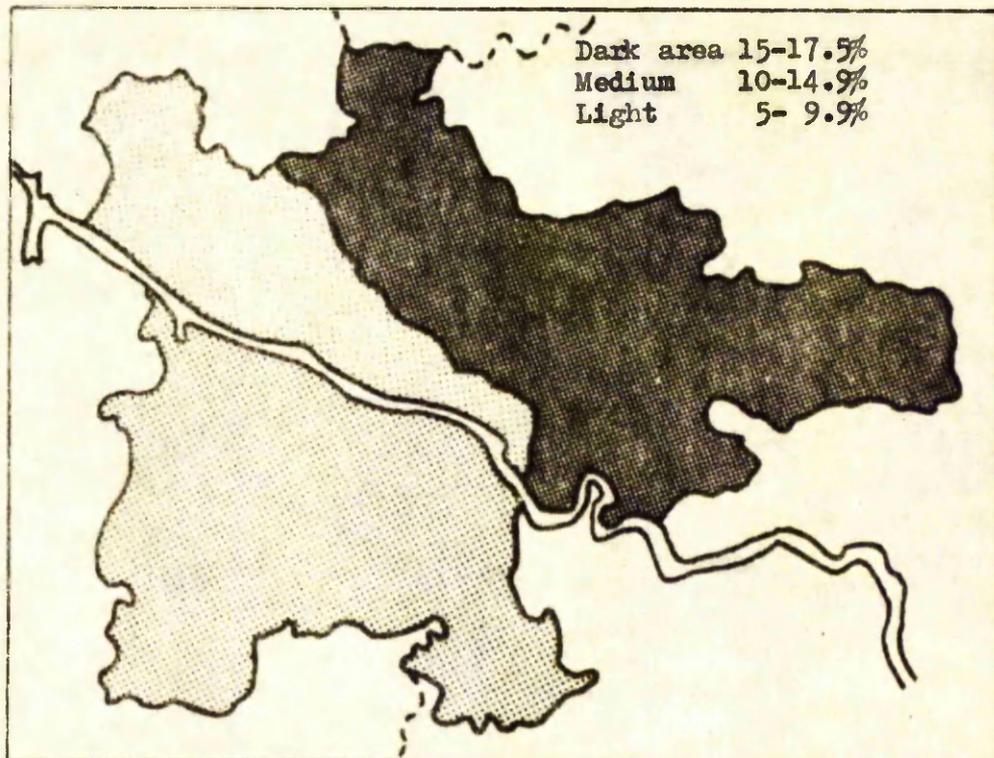
MAP II.

Perinatal Mortality by Ward (City of Glasgow, 1970).



MAP III.\*

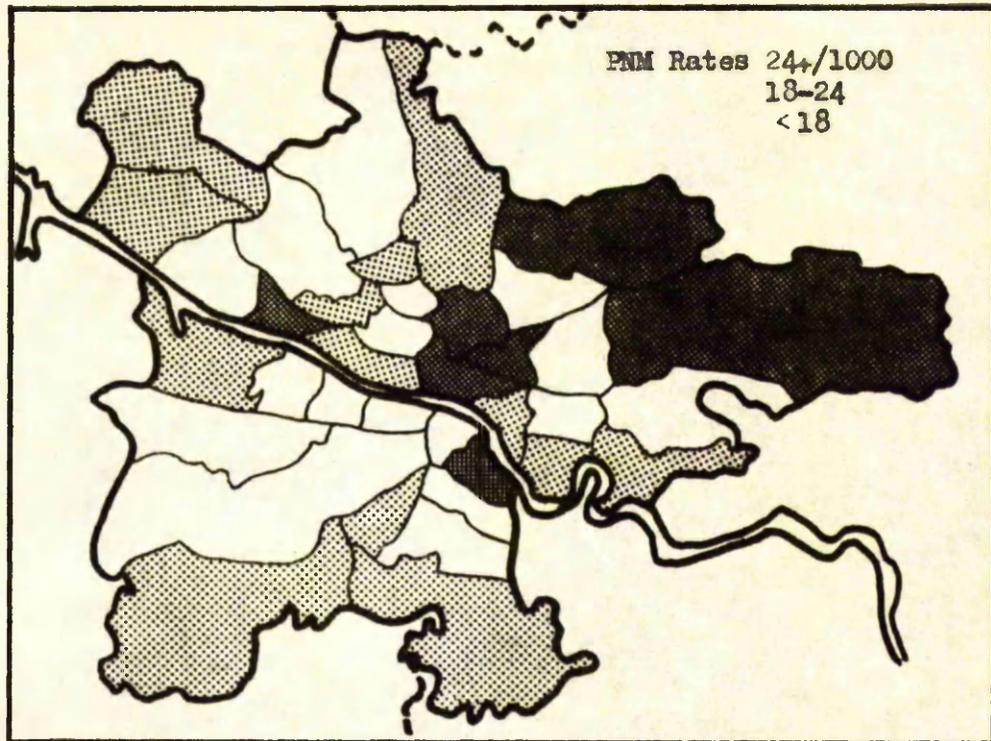
Percentage Resident Male Population in Socio-Economic Class V (1966).



\*This map is based on that of Howe (1972). Atlas of Glasgow and the West Region of Scotland. Holmes McDougall, Edinburgh. p. 25

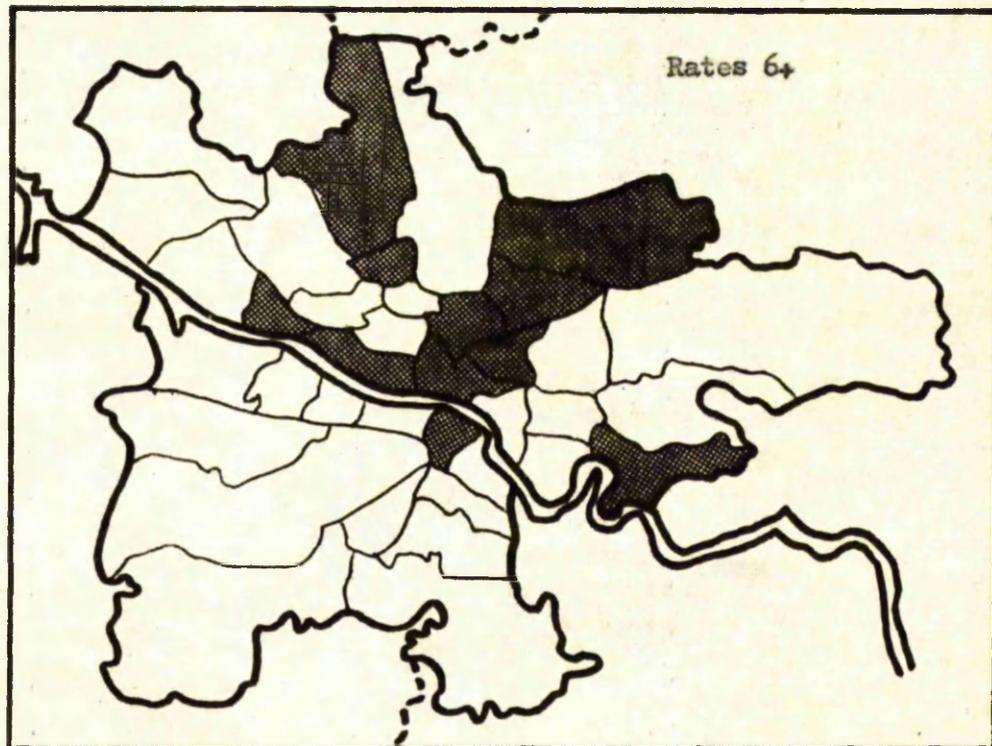
MAP IV.

Area Distribution of Environmental Deaths (1970).



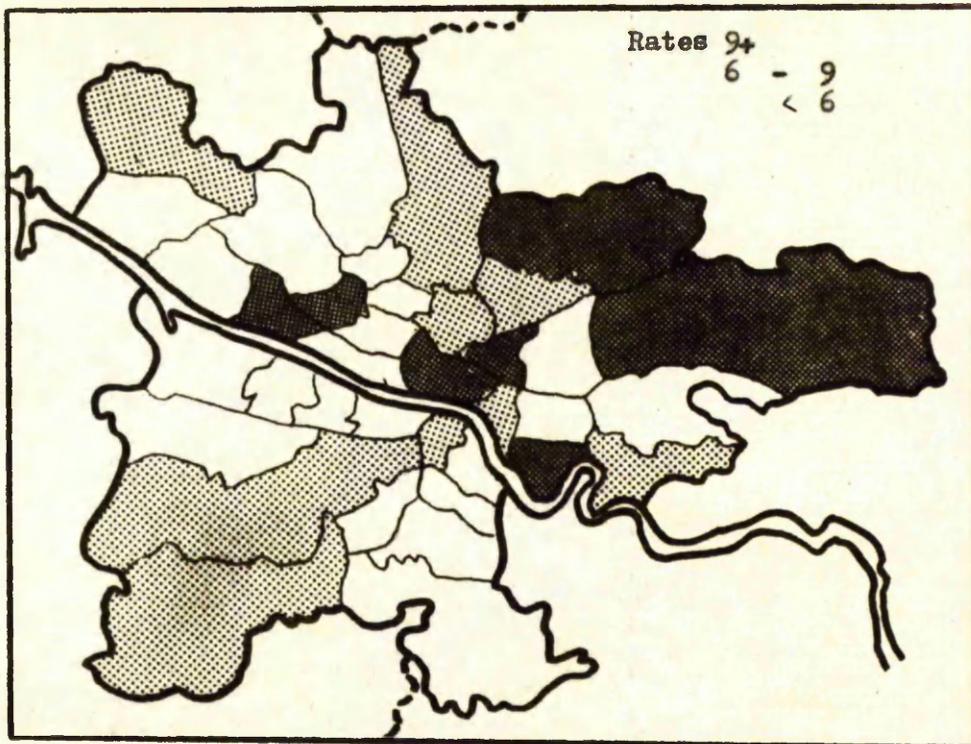
MAP V.

Area Distribution of C.N.S. Deaths (1970)



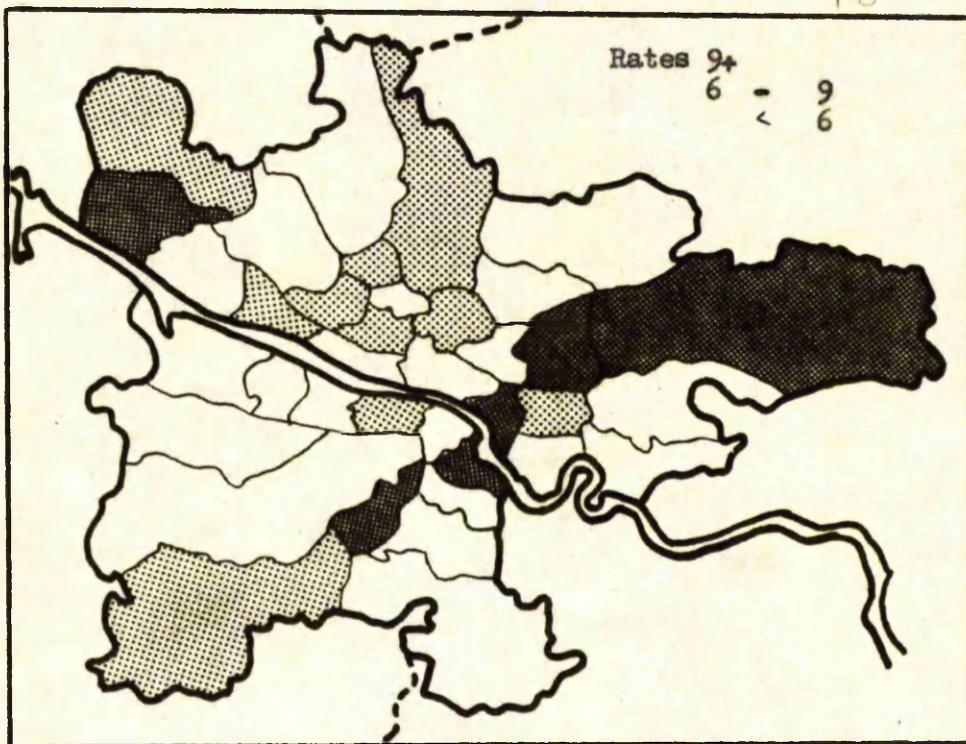
MAP VI.

Area Distribution of APH Deaths (1970)



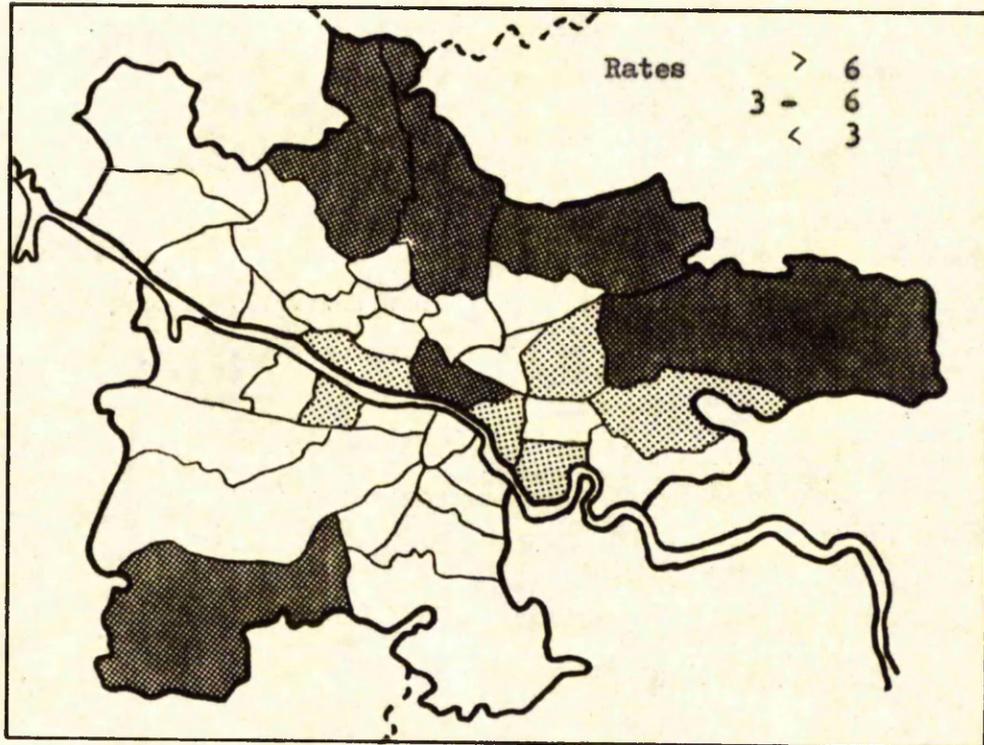
MAP VII.

Area Distribution of P.U. Deaths (1970)



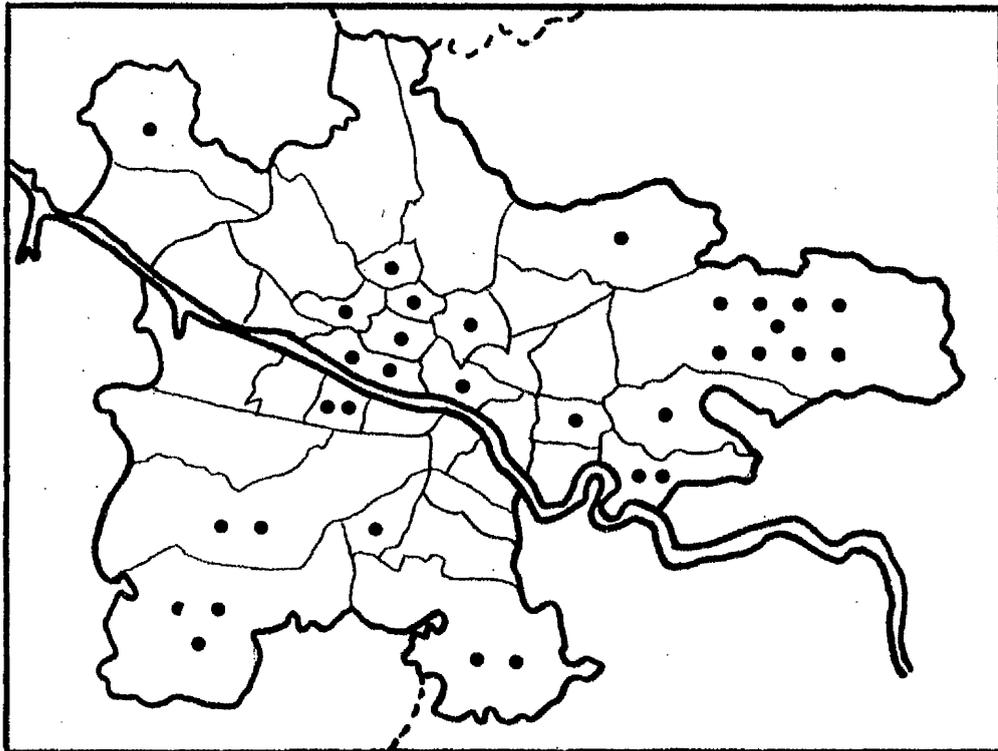
MAP VIII.

Area Distribution of M.U. Deaths (1970)



MAP IX.

Domiciliary Deaths (1970)



## FINDINGS

The perinatal mortality rate by ward of residence was calculated for the 428 mothers who lost their babies during 1970 (i.e. 437 perinatal deaths - 9 perinatal deaths where both twins died from the same clinical cause). Map I illustrates the wards of the City of Glasgow. The dark areas show the highest mortality rates and the light areas the lowest. This is so in all the subsequent maps apart from Map III. Map II illustrates the perinatal death rate by ward. The rates in Wards 7, 11 and 16 were significantly high and in Ward 5 significantly low,  $p < 0.05$ .

Map III shows the percentage resident male population in socio-economic Class V based on the 1966 sample census. This presents a similar pattern to the perinatal death map in that the greatest percentage of Social Class V males live to the north and east of the city, and the highest perinatal mortality rates are in this area. The area distribution of environmental deaths is illustrated in Map IV (the rate in Ward 7 being significantly high and Ward 36 significantly low  $p < 0.05$ ). Map V shows the area distribution of central nervous system abnormality deaths. This presents a different picture with the high rates occurring in the north - central area of the city. The numbers are relatively small, however, and the differences do not reach statistical significance. The area distribution of antepartum haemorrhage is shown in Map VI. A highly significant rate was found in Ward 7 ( $p < 0.01$ ) and significantly high rates were found in Wards 3 and 10 ( $p < 0.05$ ).

The premature - cause unknown deaths are illustrated by ward in Map VII. Again high rates are found in the east side of the city although they do not reach statistical significance. Map VIII illustrates the area distribution of mature - cause unknown deaths with highly significant rates being found in Wards 11 and 16 ( $p < 0.01$ ) and a significantly high rate in Ward 7 ( $p < 0.05$ ). The area of residence of the 33 mothers who had a domiciliary confinement is shown in

Map IX. Thirteen deaths occurred in the peripheral estates of the east side of the city.

DISCUSSION

By plotting out the area of residence of the mothers of the babies who died in the perinatal period, it is possible to pin-point the areas of greatest need, and where medical and obstetric help should be concentrated. The perinatal mortality rate is one of many yardsticks which can be used to determine poor environmental areas. Area 7, for example, Provan, had a high perinatal mortality rate by each clinical cause examined except central nervous system abnormality. This area is known to be very socially deprived, and yet there is no co-ordination of medical and social help. There is not even a Health Centre in the area, and there is a great shortage of Health Visitors. Concentration of resources, both man-power and money, would reap enormous benefits for the population in that area.

CHAPTER XIII.

COMPARISON OF CLINICAL AND PATHOLOGICAL CLASSIFICATIONS.

The main reason for classifying perinatal deaths is to analyse the deaths and to determine what can be done to prevent similar deaths occurring in the future. All perinatal deaths are recorded for the Registrar General, the cause of death being assessed by the attending physician using information from post-mortem examination if it has been carried out. The deaths are normally classified using the International Classification of Diseases.

While this study was being carried out, it was felt that it would be beneficial to compare the registered cause of death with the clinical cause to determine which was more meaningful.

The early history of the study of disease is described in the Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death (1967). The statistical study of disease began in the seventeenth century with work on the London Bills of Mortality by John Graunt, examples of which are shown in the following table (Table CV).

TABLE CV.

Causes of Death from Bills of Mortality,  
London - August 15-22, 1665 (A - L)

|             |     |                |    |
|-------------|-----|----------------|----|
| Abortive    | 6   | Flux           | 2  |
| Aged        | 54  | Dead in Street |    |
| Apoplexie   | 1   | at St.         |    |
| Bedridden   | 1   | Bartholomew    | 1  |
| Cancer      | 2   | Frighted       | 1  |
| Childbead   | 23  | Gangrene       | 1  |
| Chrisomer   | 15  | Gowt           | 1  |
| Collick     | 1   | Grief          | 1  |
| Consumption | 174 | Gripping in    |    |
| Convulsion  | 88  | the Gut        | 74 |
| Dropsy      | 40  | Jaundies       | 3  |
| Drowned     | 2   | Imposthume     | 18 |
| Feaver      | 353 | Infants        | 21 |
| Fistula     | 1   | Fell Down-     |    |
| Flux &      |     | stairs at      |    |
| Smallpox    | 10  | St.Thomas      |    |
|             |     | Apostle        | 1  |
|             |     | Kings Evil     | 10 |
|             |     | Lethargy       | 1  |

London - August 15-22, 1665 (M - W)

|                |      |                  |     |
|----------------|------|------------------|-----|
| Murdered at    |      | Starved at Nurse | 1   |
| Stepney        | 1    | Stillbirth       | 8   |
| Palsy          | 2    | Stone            | 2   |
| Plague         | 3880 | Stopping of      |     |
| Pleurisy       | 1    | Stomach          | 16  |
| Quinsy         | 6    | Strangury        | 1   |
| Rickets        | 23   | Suddenly         | 1   |
| Rising of the  |      | Surfeit          | 87  |
| Lights         | 19   | Teeth            | 113 |
| Ruptured       | 2    | Thrush           | 3   |
| Sciatica       | 1    | Tissick          | 6   |
| Scowering      | 13   | Ulcer            | 2   |
| Scurvy         | 1    | Vomiting         | 7   |
| Sore Legge     | 1    | Winde            | 8   |
| Spotted Feaver |      | Wormes           | 18  |
| & Purples      | 190  |                  |     |

During the following century, three workers - Sauvages (1706 - 1777) publishing *Nosologia Methodica*, Linnaeus (1707 - 1778) publishing *Genera Morborum*, and William Cullen (1710 - 1790) of Edinburgh publishing *Synopsis Nosologiae Methodicae*, contributed greatly to the systematic classification of disease, and it was Cullen's work that was most used at the beginning of the nineteenth century.

But it was the work of William Farr (1807 - 1883), the first medical statistician of the General Register Office of England and Wales, who introduced the principle of classifying diseases by anatomical site instead of alphabetically, which had been the practice previously. Farr's work survived as the basis of the International List of Causes of Death and now the International Statistical Classification of Diseases, Injuries and Causes of Death. The International Conference for the Eighth Revision of the International Classification of Diseases convened by the World Health Organisation in Paris in 1965 was attended by 36 member states and one associate member - thus it is a truly international body.

In the introduction to the Manual of the International Statistical Classification of Diseases, Injuries and Causes of Death, it states that "a statistical classification of disease must be confined to a limited number of categories which will encompass the entire range of morbid conditions. The categories should be chosen so that they will facilitate that statistical study of disease phenomena. A specific disease entity should have a separate title in the classification, only when its separation is warranted, because the frequency of its occurrence or its importance as a morbid condition justifies its isolation as a separate category. On the other hand, many titles in the classification will refer to groups of separate but usually related morbid conditions. Every disease or morbid condition, however, must have a definite and appropriate place as an inclusion in one of the categories of the statistical classification. A few items

of the statistical list will be residual titles for other and miscellaneous conditions which cannot be classified under the more specific titles. These miscellaneous categories should be kept to a minimum."

The above principles are adhered to in the International Classification, which is used when deaths are registered.

Identifying the cause of a perinatal death is frequently much more difficult than determining the cause of death in a child or an adult. Pathological examination is not particularly helpful in the majority of cases as the findings are often non-specific.

What is required, therefore, is some relatively simple classification of deaths that deals with broad categories so that all deaths can be classified and general trends can be seen. It was felt the Aberdeen classification met this requirement. A comparison has therefore been made of the existing method of classification, i.e. the registered cause of perinatal death with the Aberdeen classification by which all perinatal deaths occurring in the City of Glasgow during 1970 have been re-classified.

Baird, Walker and Thomson (1954) devised a clinico-pathological classification which has proved to be useful not only in the Aberdeen Maternity Hospital, where excellent record keeping has been the practice for several decades, but also with data from other centres. A modification of this classification was used in the analysis of all the perinatal deaths in the 1958 British Perinatal Mortality Survey.

In the Chapter in "Perinatal Problems" entitled "The Survey Perinatal Deaths reclassified by Special Clinico-Pathological Assessment," Baird and Thomson (1969) state that "an unduly complex classification may create difficulty during statistical analysis and will tend to obscure broad trends. We consider that it is desirable whenever possible to allocate each case to a single cause group, which gives the maximum information with regard to preventability. The rules of classification can scarcely avoid incorporating several arbitrary features but

they should be explicit. If too much is left to the play of individual judgement, comparisons between different series or the description of changes with time will have limited validity. It is not easy for a single observer to maintain consistent standards of judgement over a period of time and the difficulties are multiplied if several observers are involved."

Bearing in mind the above advice of Baird and Thomson, the perinatal deaths were classified using their classification and compared with the registered cause of death.

#### METHOD

The registered cause of death was known for all perinatal deaths occurring in Glasgow during 1970. This information is stored in the Glasgow Corporation Computer, an abstract of particulars at death registration having been made and the cause of death having been coded for the Computer by a member of the medical staff of the Health and Welfare Department using the International Classification of Diseases. The clinical classification has been described in detail in Chapter III.

The clinical cause of death and the registered cause of death were therefore recorded for each perinatal death and the two classifications compared to determine the amount of agreement in the two classifications.

The findings of 404 perinatal deaths occurring in the City of Glasgow during 1970 are discussed, as twin deliveries have been excluded.

#### RESULTS

The clinical cause of death is cross-tabulated with the registered cause in Table CVI.

TABLE CVI.

## Clinical Cause.

| Registered Cause.                                   | M.U. | P.U. | Trauma. | Tox. | APH. | M.D. | F.B. | Other. | No. | %     |
|-----------------------------------------------------|------|------|---------|------|------|------|------|--------|-----|-------|
| 1. Deformity                                        | -    | 1    | -       | -    | 1    | -    | 86   | -      | 88  | 21.8  |
| 2. Conditions of Placenta                           | 13   | 14   | 1       | 3    | 47   | -    | 3    | -      | 81  | 20.0  |
| 3. Immaturity                                       | -    | 35   | 1       | 3    | 23   | 3    | 2    | 4      | 71  | 17.6  |
| 4. Anoxic and Hypoxic Conditions                    | 12   | 6    | 2       | 1    | -    | 2    | 1    | 2      | 26  | 6.4   |
| 5. Other complications of pregnancy and childbirth. | 2    | 2    | 1       | 1    | 11   | -    | 2    | 1      | 20  | 5.0   |
| 6. Haemolytic Disease                               | 1    | 3    | -       | -    | -    | -    | -    | 14     | 18  | 4.4   |
| 7. Toxaemia                                         | -    | -    | -       | 15   | 2    | -    | -    | -      | 17  | 4.2   |
| 8. Conditions of Cord                               | 7    | 4    | 4       | -    | -    | -    | -    | -      | 15  | 3.7   |
| 9. Maternal Disease                                 | 3    | 1    | 1       | 3    | -    | 3    | 1    | -      | 12  | 3.0   |
| 10. Difficult Labour                                | -    | -    | 10      | -    | -    | -    | -    | -      | 10  | 2.5   |
| 11. Birth Injury                                    | 1    | 1    | 2       | -    | 3    | -    | 3    | -      | 10  | 2.5   |
| 12. Fetal Death - Cause Unknown                     | 3    | 2    | 1       | -    | 1    | -    | 3    | -      | 10  | 2.5   |
| 13. Respiratory Distress Syndrome                   | 1    | 2    | -       | 2    | 2    | 2    | -    | -      | 9   | 2.2   |
| 14. Asphyxia of Newborn                             | 1    | 3    | -       | 1    | -    | -    | 1    | 1      | 7   | 1.7   |
| 15. Other Conditions of Fetus                       | 5    | -    | -       | -    | 1    | -    | -    | -      | 6   | 1.5   |
| 16. Haemolytic Disease of Newborn                   | 1    | 2    | -       | -    | -    | -    | 1    | -      | 4   | 1.0   |
| Total                                               | 50   | 76   | 23      | 29   | 91   | 10   | 103  | 22     | 404 | 100.0 |

There is only close agreement in two groups - fetal deformity (86 out of 103 deaths) and Rhesus isoimmunisation (14 out of 17 deaths). This is an expected finding as these two causes of perinatal death are in most cases easily recognised and therefore easily classified. There was not close agreement in the remaining categories. In the mature - cause unknown group, half the deaths were attributed to conditions of the placenta or anoxic or hypoxic conditions. These vague classifications are in keeping with the failure to determine the cause of death, but the remaining 25 deaths in that category were registered as dying from ten other causes which makes the picture very confusing and of little value when the cause of perinatal death and prevention in the future is being assessed. A similar muddled list is obtained for the small babies classified as dying from an unknown cause. Of the 76 deaths classified in this way, 35 were registered as being due to immaturity, 14 due to conditions of the placenta, and the remaining 27 due to a variety of causes. Ten of the trauma deaths were attributed to difficult labour, a further 4 to conditions of the cord, and two due to birth injury. On studying the registered cause of death the remaining seven deaths had no obvious connection with birth trauma. Of the 29 toxæmic deaths, half were registered as dying because of maternal toxæmia. Only 47 of the 91 antepartum hæmorrhage deaths were registered as death being due to conditions of placenta, and a further 23 registered as immaturity. There was not even much agreement in the maternal disease group with only 3 of the 10 deaths being registered as being due to maternal disease.

Agreement between the two methods of classification with regard to type of abnormality was very good (Table CVII).

TABLE CVII.

Abnormalities.

| Registered Classification.               | Clinical Classification. |   |   |    |   |    |
|------------------------------------------|--------------------------|---|---|----|---|----|
|                                          | 1                        | 2 | 3 | 4  | 5 | 6  |
| Anencephaly                              | 36                       | 1 | 1 | -  | - | -  |
| Spina Bifida                             | -                        | 4 | 3 | -  | - | 1  |
| Hydrocephalus                            | -                        | - | 3 | -  | - | -  |
| Congenital Heart Disease                 | -                        | - | - | 10 | - | -  |
| Alimentary                               | -                        | - | - | -  | 4 | -  |
| Other/Multiple                           | 7                        | 1 | - | 3  | 3 | 15 |
| Maternal Disease                         | 1                        | - | - | -  | - | -  |
| Conditions of Placenta                   | 1                        | - | 1 | 1  | - | -  |
| Immaturity                               | -                        | - | 1 | -  | - | 1  |
| Birth Injury                             | -                        | - | - | -  | 1 | -  |
| Other Conditions of Fetus<br>and Newborn | -                        | - | - | -  | 1 | 2  |
| Total                                    | 45                       | 6 | 9 | 14 | 9 | 19 |

1 = Anencephaly.

2 = Spina Bifida.

3 = Hydrocephalus.

4 = Congenital Heart Disease.

5 = Alimentary

6 = Other

Apart from the two groups, fetal deformity and other (which is composed largely - 17 out of 22 - of babies who died of Rhesus incompatibility) there is very little agreement between the clinical classification and the registered classification.

DISCUSSION

In this study it would appear that the analysis of perinatal deaths by registered cause is of limited value. The main reason for this is that there are too many rather non-specific categories. Usually the terminal event leading to death is recorded, but there is no indication of the clinical condition and

the main factor, which was the true cause of death. In the registered cause "Conditions of the Placenta," for example, 47 of the 81 deaths occurred in the antepartum haemorrhage group, 14 in the premature and 13 in the mature - cause unknown groups. Similarly, the registered causes "Immaturity" and "Anoxic and Hypoxic Conditions" occurred in nearly all the clinical classification groups.

This study of perinatal deaths, like that carried out in the British Perinatal Survey, has shown that there is a definite advantage in using a classification in which the clinical history has been considered along with necropsy findings, and not simply pathological details alone. The classification devised by Baird, Walker and Thomson (1954) is short, simple, efficient and could easily be applied by all clinicians working in the perinatal field. This would be of much greater value than studying the registered cause of death, would illustrate specific problems, and thus show where improvement in clinical management was required and thus reduce perinatal mortality in the future.

CHAPTER XIV.

DISCUSSION.

This study of perinatal deaths in Glasgow set out to answer three questions:

- (1) What was the clinical event leading up to perinatal death?
- (2) Which mothers lost their babies, and did these mothers receive adequate antenatal care?
- (3) What changes, if any, might be recommended in obstetric and paediatric care to improve the perinatal outcome?

These questions have been answered.

The general findings from this survey are that two-thirds of the deaths (300 out of a total of 437) were attributed to environmental causes, fetal deformity, premature - cause unknown, and antepartum haemorrhage. The risk of perinatal death has been shown to be raised in mothers aged less than 20 years and over 30 years, in primigravidae, particularly those with illegitimate pregnancies, in women of high parity and in women of poor socio-economic status, belonging to Social Class IV and V (Registrar General Classification). The risk was particularly high in domiciliary confinements and in births where there was a previous history of abortion, stillbirth or neonatal death. The housing conditions of a large number of the women were very poor, and there was a marked area difference in the perinatal mortality rates. Perinatal mortality rates in the East-end of the City of Glasgow were particularly high. In a large number of patients antenatal care was thought to be inadequate and some patients received no care at all.

These findings can easily be summarised by saying the poor results in Glasgow are due to three main causes.

- (1) Poor maternal physique which has resulted from the poor environment in which the women lived and where they spent their formative years.
- (2) Poor obstetric care in some cases.
- (3) Failure of the patients to make use of the medical facilities available to them.

This, however, is an over-simplification of the problem, because if there is a problem of patients not utilising the medical services provided for them, there is also the problem of the medical services not making contact with the people most in need of their help.

The National Health Service was established in 1948 with the object of providing a universal and comprehensive health care system in the United Kingdom. The original form of organisation was tripartite with separate branches responsible for the provision of the family doctor, hospital and public health services. Townsend (1974) writing in the Lancet about "Inequality and the Health Service," asks the question "How far are health care values and practices shaped by the general structure of inequality in society? Or to put this question the other way round, how far has the development of the medical and other professions within the structure of health services positively contributed to the conceptions of status and rewards generally held in society? Does the system of health services help to shape the structure and values of society in general, or is the direction of influence the other way? Can one, indeed, be disentangled from the other? Can equality in medicine, like equality before the law, be practised on a kind of island remote from the cruel inequalities of the rest of social life?"

Townsend goes on to say "Estimates of the effect or value of the Health Service depend, of course, on the kind as well as the availability of information used to measure such an effect or value. There are measures of health, as such, which depend on conceptions of health and there are measures of utilisation and

provision of services, each of which is needed to assist explanations of trends of health and of social differences in mortality and morbidity."

The perinatal period occupies less than 0.5 per cent of the average life span, yet in many countries there are more deaths within this period than during the next 30 years in the life of a population. As infant and childhood mortality is gradually reduced, attention is being increasingly focused on the prevention of death in the perinatal period. Information from health statistics of the perinatal period is of considerable importance in the provision, planning and evaluation of the delivery of health care to mothers and infants. Such information is useful both at the level of the individual patient care and at the level of health care of a community.

One of the major problems in the delivery of health care is the tremendous fragmentation among available services. The position in Glasgow appears to be similar to that of the City of Baltimore where a Maternal and Infant Care Project was set up in 1965. Swallow (1972) describes the situation before the project was started. "Baltimore had, if anything, an embarrassment of riches in health care services. It had two medical schools. It had a number of excellent teaching hospitals. It had a plethora of obstetric beds. It had one of the oldest health departments in the country, one which had been long concerned with maternal and child care. It had 200 to 250 public health nurses and they were making some visits to mothers and infants. The nurse was considered a family nurse and was supposed to look after all the problems found in the family. Baltimore's Education Department and its Welfare Department were long established and were in communication with the Health Department. There were a large range of voluntary services concerned with matters infringing on health.

"Yet, in spite of all the resources, the maternal mortality rate for Baltimore City in 1962 was 5.2 per 10,000 live births compared to 3.8 for the rest of Maryland. The perinatal mortality rate for the City was 42.1 compared to 30.5 for the rest of the State, and the infant mortality rate was 32.0 compared

to 22.6. Of all births in Baltimore City 12.0 per cent were classified as premature compared to 7.8 per cent for the rest of Maryland."

One of the reasons for the high rate, according to Swallow (1972) was that Baltimore's health services were not working together in the optimal fashion and failure of co-ordination hit hardest in the low-income areas. She felt that the chief problem was the lack of systematic follow-up of mother and child through the maternal and neonatal period. The Project aimed at improving patient care and follow-up by making more appropriate use of all available resources and at improving the system of communication. This project attempted to end the traditional separation of services between obstetrics and paediatrics. The "645 Days" concept was developed whereby they aimed at giving continuity of care through the 40 weeks of gestation, delivery and the following months until the infant reached his first birthday - 645 days. Responsibility for the primary care was placed on the one large city-wide service available which was the Public Health Nursing Services. The activities of the public health nurse were supported by obstetrical and other nursing staff, social workers, nutritionists, dentists and home-helpers. The combined activities of all these workers connected the patient in the community to the medical structures such as hospital, social services or educational institutions.

There are similar problems in the City of Glasgow. In 1970 there were nine maternity hospitals where a patient could receive antenatal care and be delivered. The patient could receive antenatal care from various sources. She could be looked after during pregnancy entirely by her general practitioner, entirely by the maternity hospital staff or entirely by her local authority clinic doctor. Care could also be shared between any or all of these three doctors. The patient could elect to be delivered at home, being looked after throughout her pregnancy by her general practitioner and domiciliary midwife, or to be delivered in hospital, or she may have denied her pregnancy and received no care at all.

In a city the size of Glasgow, therefore, with various types of care and

different places where antenatal care can be received, it is difficult to organise a consistent standard of care. Members of the medical profession do not take kindly in being dictated to about when in pregnancy they ought to see their patients; how often they should be seen; who is a high risk patient; which patient is a suitable candidate for combined antenatal care; and what in fact does combined care mean?

The cities of Aberdeen and Dundee have low perinatal mortality rates - 20 per 1000 and 19 per 1000 respectively for 1970. It is therefore interesting to compare the maternity services in these cities with those of Glasgow. The main difference is that in Aberdeen and Dundee there is only one obstetric division in each city. University and National Health Service staff work together in organising principles for antenatal care. After discussion between hospital staff and general practitioner these principles are brought into practice. In Aberdeen, for instance, each patient is seen as early as possible in her pregnancy at the antenatal clinic at the Maternity Hospital. Details of her previous deliveries are available, and at this booking visit a decision is taken about her subsequent care, depending upon her age, parity and previous medical and obstetric history. If the decision is taken that the patient will receive shared antenatal care between the maternity hospital staff and her general practitioner, she is always seen at the Maternity Hospital at certain predetermined periods of gestation or more frequently if any problems arise. Policy decisions are also made about induction of labour, management of pre-eclampsia, management of diabetes in pregnancy, management of the Rhesus patient, and so on. All the medical staff, therefore, have guide lines to follow when they are looking after their patients. Newsletters are sent out regularly to general practitioners working in the city and in the country areas, keeping them informed about management of certain cases. Since the 1950s the obstetric services in these two cities, Aberdeen and Dundee, have been administered in this way with continuing improvement in the perinatal mortality rates.

How then can the perinatal mortality rate in the City of Glasgow be improved?

This must be looked at from the point of view of

- (a) immediate action,
- (b) long term planning.

#### Immediate Action

From this study of perinatal deaths in Glasgow it would appear that a number of steps could be taken immediately which would improve the antenatal care and hopefully the perinatal outcome. From the organisational side the importance of antenatal care and early referral of the patient to the maternity hospital for assessment could be stressed to the general practitioner by means of a newsletter. Each maternity hospital could look at the time taken from referral of the patient to hospital by the general practitioner to actually being seen at the antenatal clinic. The delay should be no longer than two to three weeks, and if it is longer steps must be taken to reduce this waiting time.

The medical staff in each maternity hospital could apply the clinical classification of deaths to their perinatal deaths to determine what the main problem with their area is, and whether a change in management might improve the outcome.

By providing a family planning service in the postnatal period and offering sterilisation to those patients who wish this operation, the number of deliveries in the highly parous group would be reduced, and consequently the number of deaths in this high risk group would also be reduced.

The existing maternity services in the geographical areas of high perinatal loss in the city should be studied. If the patients do not have a clinic in their area, have to travel long distances to receive antenatal care and do not have adequate help from their health visitor because she is so overworked, reorganisation of available resources and the establishment of a medical centre in the area would

help to increase the amount of care the patient was receiving.

The number of deaths in each one of the eight groups of perinatal deaths could be reduced by closer attention to detail in the antenatal period.

Mature - Cause Unknown: The women most at risk from this cause of death are the highly parous patients. These are the patients who usually receive shared antenatal care because with their family commitments it is easier for them to attend their general practitioner than the hospital antenatal clinic. It might be wise for these patients to be seen only at the hospital clinic from 32 weeks of pregnancy onwards. Particular attention should be paid to the weight gain pattern of these high risk patients and assessment of the feto-placental unit could be made by routine oestriol estimations.

Premature - Cause Unknown: The main problem in this group is that the patient did not receive antenatal care and it is going to be a gradual process by education before this can be improved. In the meantime, a policy decision could be taken whereby all patients in premature labour were only delivered in hospitals with intensive neonatal care facilities. If an unbooked patient arrived at a hospital without such facilities and on examination she was found to have a small baby which by her dates was very premature, she could be transferred immediately to one of the main centres.

Trauma: The reduction of trauma deaths depends on improved obstetric care, by more careful management of breech deliveries, cord complications and other complications of labour.

Toxaemia: A number of babies in this group were mature enough to survive. It would appear that by closer supervision of these patients and by balancing up the state of the fetus in utero against its chance of survival if delivered immediately against the mother's condition, might reduce the number of deaths from this cause.

Antenartum Haemorrhage: The largest group of haemorrhage deaths was due to

placental abruption. A number of the patients in this group did not receive any antenatal care. It was felt, however, that an earlier recourse to Caesarean Section might have improved the perinatal outcome.

Maternal Disease: Three of the twelve deaths in this group were due to the pre-diabetic condition of the mother. To manage such cases successfully throughout the pregnancy, a special diabetic/antenatal clinic should be set up for a city the size of Glasgow. One or two such clinics would be sufficient to look after all these patients.

Fetal Deformity: With the discovery of the association of raised alpha-feto-protein liquor levels in patients with a fetus with a central nervous system abnormality, all patients with a previous history of such an abnormality should be screened, and if such an abnormality was present the patient should be offered a termination of pregnancy. All patients who deliver an abnormal baby would be given genetic counselling.

Rhesus Incompatibility: All patients must be made aware of their blood group and if they are Rhesus negative the problem of Rhesus incompatibility explained to them. In this study a number of patients did not attend for care until late in pregnancy. All patients should be seen at a special antenatal clinic, perhaps two would be sufficient for the City of Glasgow. It is important to reduce the number of centres dealing with this problem so that the doctors looking after these patients see enough patients and so maintain their skill at carrying out intra-uterine transfusion. It is also very important that the babies are delivered in a hospital with intensive neonatal care and haematology specialists.

This study of perinatal deaths occurring in Glasgow during 1970 has shown where the problems lie. There is considerable scope for reducing rapidly the current high mortality; this lies in identifying the women at risk early in pregnancy, monitoring them carefully during the antenatal period and delivery, and in encouraging greater use of the now widespread family planning services.

### Long Term Planning

Improvement in perinatal mortality in the long term can be looked at from three different areas:

- (a) Improvement of the general health of future mothers.
- (b) Care during pregnancy (including labour, delivery and the puerperium).
- (c) Follow-up of the families after discharge from hospital.

#### (a) Improvement of the General Health of Future Mothers:

By health education and the development of a good nutritional status the general level of family health ought to improve. Children at school whatever their ability should be prepared for family life and parenthood. It is important that all children should know about the physiology of human reproduction and the various forms of contraception. In an article entitled "Pregnancy Outcome and Fertility Control in Aberdeen," Thompson and Aitken-Swan (1973) state that "Experience in Aberdeen shows that there is still misunderstanding and ignorance about conception among girls in every social class and there is much need for accurate information on sex and sexual relationships for both boys and girls. However desirable it is that parents should instruct their children in such an emotionally sensitive area, we have to accept that many are ill-equipped to do so precisely because their own parents all too often failed in this respect. If we are to break this vicious circle, the main burden of sex instruction for the time being must fall on professional educators and the factual content of their instruction must go beyond the physiology of sex and reproduction and include detailed information on contraception in the context of responsible sexual behaviour." This last sentence is of particular importance because in 1968 as a result of a recommendation of the Grampian Television Schools Advisory Committee, Aberdeen became the first area in Britain in which a television series on sex education for use in schools was produced. The series "Living and Growing" was prepared by

an Aberdeen obstetrician, K.J. Dennis, for children aged 10 to 13 years and won an international award. The series dealt with the human life cycle in the context of the family unit and made only passing reference to the mechanics of sexual intercourse and no reference at all to contraception, family planning and sterilisation (Thompson and Aitken-Swan, 1973). It is generally accepted that human biology should be taught at school level. Pupils at the secondary stage ought to be given information about contraception in the context of responsible sexual behaviour and at this stage the importance of early antenatal care could be explained to them.

(b) Care during Pregnancy:

The report on the Integration of Maternity Work (1973) suggests that a programme of obstetric care will include specialist obstetric units, general practitioner maternity beds and units, arrangements for domiciliary confinements, antenatal and postnatal care, family planning, and, where necessary, genetic counselling, health education, care and supervision of infants and liaison with the social services.

In order that this plan can work efficiently there must be close co-operation between all the people involved with the pregnant patients.

Within the reorganisation of medical services, the City of Glasgow has been divided into ~~four~~ divisions, north, south, <sup>and east</sup> east and west, for administrative purposes under the Greater Glasgow Area Health Board. It is important that all the people concerned with obstetric care in each division meet and make some decisions about obstetric management in their area. This will require liaison with general practitioners, hospital medical and nursing staff, community physicians, health visitors, social workers and records officers. At the present time there is much overlap from one area to another, patients attending clinics in one district and being delivered in another. It would make organisation less complex if all patients living in one district received all their care in that district. It is estimated that in 1974 there will be approximately thirteen thousand deliveries in

the City of Glasgow, but this figure will be greatly increased by including all the births from Greater Glasgow. It will therefore be likely that there will be between four and five thousand deliveries in each division. This is the optimum number of deliveries for an area to include all types of care.

The General Practitioner. The family doctor, whether he is particularly interested in obstetric care or not, is going to be the first person in determining the type of care the patient is going to receive. It is to be hoped that the patient will realise the importance of antenatal care and that she will present herself to her doctor as soon as she suspects she is pregnant. If the general practitioner, after taking a history, thinks that pregnancy is likely he should refer that patient immediately to the local maternity hospital. If the general practitioner is interested in obstetrics and is on the Obstetric List, he should state this in the referral letter and indicate his willingness to carry out shared antenatal care. If this is to be done a shared care record card must be used containing all the information about each antenatal visit whether in hospital or at the general practitioner's surgery, so that details of the patient's weight gain, blood pressure, urinalysis and so on are recorded at each visit and known to both the hospital and the family doctor. At the end of pregnancy this information should be retained in the patient's hospital records.

Hospital Staff. Hospitals for a large number of patients are very rigid, frightening places. The patients often have to wait for a long time before they are seen and the individual often is not given much consideration. The fact that a patient had to wait for a long time at the clinic and was late for her children arriving home from school, may not be very important when there are a hundred patients to be seen, but it is important enough to make that patient wish to receive her care from some other source. The obstetrician assesses the patient at her first antenatal visit and taking into account her age, parity, height, social circumstances and previous medical and obstetric histories, makes the decision about what type

of care the patient should receive. He decides where she should be delivered. If she falls into the high risk category, she will be booked for delivery at the main hospital, but if there are no problems she may be booked for delivery in a peripheral hospital or in a general practitioner unit. This booking could be altered if any complication occurs during pregnancy.

The hospital service will also help the patient with regard to mothercraft, advice about diet during pregnancy, physiotherapy and preparation classes for delivery. Family planning advice ought to be given in the puerperium.

Community of Physicians. The report on "Community Medicine in Scotland" (1973) states that "wide range of activities with which a maternity services division will be concerned affords considerable scope for a specialist in community medicine to apply his skills in the measurement and interpretation of trends in maternal and neonatal health, and in the evaluation of all aspects of maternity care. There will be need to institute investigation of perinatal mortality in various groups of the population and to see that appropriate provision is made for high risk groups. An important aspect of his contribution will be to ensure communication with management, with local authority social work departments, and with health education services. The planning and organisation of a comprehensive family planning programme within the maternity services will include general practitioners, specialist obstetric units, health visitors and social workers."

Health Visitors and Social Workers. These people in many cases are visiting the patient in her home before she becomes pregnant and have built up a good relationship with her. They will be able to advise the patient about the importance of antenatal care, reassure her about any problem occurring during pregnancy and be able to inform the doctor looking after her about any important occurrence that might alter the patient's obstetric management.

Records. One of the problems in a city the size of Glasgow is that a patient may have previously been delivered in any one of the nine maternity hospitals, or at

home, and it is sometimes difficult to obtain an accurate description of what happened. At the present time, if the patient's previous history appears to be important, details of this delivery are requested from the hospital where she had her previous confinement. In the case of a highly parous woman, several hospitals may have to be contacted. A letter is then returned with a summary of the delivery. In some cases this process may take several valuable weeks. It would be hoped that a linked record system could be set up for all obstetric patients at an area or perhaps national level. A planned system of recall which might be computer controlled would make all the details of previous pregnancies readily available.

(c) Follow-up of the Families after Discharge from Hospital.

In the report "Towards an Integrated Child Health Service" (1973) priority for the vulnerable is discussed in a chapter of that title. Unreliable groups are defined as groups of people, who because of something in themselves or their environment, are actually or potentially exposed to special hazard. "Unreliable families are most often found in social classes IV and V (Registrar General Classification). They frequently live in poor surroundings. Parents in such families are sometimes of limited intelligence and have difficulty in giving proper care to their children; one effect of this is that they may make poor use of health and welfare services. The identification of vulnerable families will depend upon close co-operation between doctors, health visitors and social workers. This will be a great deal easier in health centre practice where the health team are working from a common base." The development of these children must be watched very carefully. Pre-school education must be encouraged in these areas with a high concentration of vulnerable families and a domiciliary family planning service is essential.

By improvement of the general health of future mothers, improved care during pregnancy and by careful follow-up of the vulnerable families and provision of a domiciliary family planning service the perinatal mortality rate in the City of Glasgow should be greatly reduced.

Llewelyn Jones (1974) in his book "Human Reproduction and Society," sums up the reasons for collecting perinatal information as follows:

"At a national or regional level the data are valuable as -

- (1) They permit health planners to identify avoidable factors and to suggest corrective measures which may be taken.
- (2) They permit the annual assessment of the extent of perinatal mortality and morbidity, and the changes which occur in response to the development of new services.
- (3) The information obtained provides those concerned with administering health services, including teaching and research, some means to measure the effectiveness of the services in meeting stated objectives.
- (4) It enables health planners to direct investigation to explain why certain areas of the nation have a rate different from the mean.
- (5) With accurate data the information can be used as a longitudinal research tool for multiple cause correlations and analysis."

In the long term, however, the study of perinatal mortality rates will not be a sensitive enough index for evaluating the effectiveness of the obstetric service (Towards an Integrated Child Health Service, 1973). Some measurement of morbidity will be required. A feed-back to the obstetrician about the development of the child will be essential. The value of this long term follow-up of the children born during the survey week of the 1958 British Perinatal Mortality Survey has already been demonstrated. In "From Birth to Seven" (Davie, Butler and Goldstein (1972) reported on the first seven years of the children's lives. In "Born to Fail?" Wedge and Prosser (1973) gave an account of the social circumstances of children in Britain aged 14 years of age. The sixteen year assessment will be made this year.

While perinatal mortality rates are still used as an index of the effectiveness of the maternity service, there is need to standardise the definition of

perinatal death. As Butler (1967) says "A more comprehensive and international standardized system of registration and vital statistics reporting would greatly improve the comparability of international perinatal data, while information of ethnic and geographical variations might provide important clues concerning etiology."

Future Work.

Since this survey was carried out in 1970 there has been a drop in the birth rate in the city of Glasgow from 17.9 per thousand of population to 15.1 in 1972. The last report of the Medical Officer of Health for the City of Glasgow was published giving information about 1972 data, and shows that Glasgow has the highest perinatal and infant mortality rates when compared with the other large cities in Scotland and England (Table CVIII).

TABLE CVIII.  
Mortality Rates.

|                   | Birth Rate/1000<br>Population | Perinatal Mortality<br>Rate/1000 live and<br>stillbirths | Infant Mortality<br>Rate/1000<br>live births |
|-------------------|-------------------------------|----------------------------------------------------------|----------------------------------------------|
| Scotland          | 15.1                          | 23.7                                                     | 18.8                                         |
| Glasgow           | 15.1                          | 28.0                                                     | 25.0                                         |
| Edinburgh         | 12.9                          | 19.0                                                     | 15.0                                         |
| Aberdeen          | 13.0                          | 20.0                                                     | 13.0                                         |
| Dundee            | 13.7                          | 21.0                                                     | 15.0                                         |
| England and Wales | 14.7                          | 21.7                                                     | 17.3                                         |
| Birmingham        | 15.5                          | 25.1                                                     | 21.5                                         |
| Manchester        | 14.6                          | 27.1                                                     | 22.3                                         |
| Liverpool         | 14.5                          | 24.9                                                     | 14.8                                         |
| Leeds             | 14.2                          | 24.5                                                     | 19.4                                         |

(Table taken from the Report of the M.O.H. Glasgow, 1972).

(1) It is proposed to analyse the perinatal deaths again for the year 1974, four years after this present study, to determine whether there has been any change in the pattern of perinatal deaths.

(2) As this study has shown that the second commonest cause of perinatal death following fetal deformity was the premature - cause unknown group, accounting for 22 per cent of all perinatal deaths, an epidemiological study of all babies born three weeks before term at the Glasgow Royal Maternity Hospital is being undertaken. The aim of this study is to identify the patients and determine whether they received adequate antenatal care. A prospective study of high risk patients will then be undertaken.

(3) From this study it has been shown that many patients attended late for antenatal care. A prospective study is therefore being undertaken of all new patients attending the professorial antenatal clinic at the Glasgow Royal Maternity Hospital to determine which factors influence a patient in coming for antenatal care, and if she is late in booking where is the delay? Does the patient delay in attending her general practitioner, does the doctor wait for two months to see if the pregnancy is progressing before referring the patient to hospital, or is the delay in the appointments office of the antenatal clinic? It is hoped that an answer will be obtained for these questions.

(4) One area in the east end of the city had a high perinatal mortality rate for all causes of perinatal death except for central nervous system abnormality. A special antenatal clinic is therefore being set up in a tenement building in a housing scheme of low density housing in this area where there are immense social problems and a considerable degree of unemployment. The aim of the clinic is to determine whether providing a clinic in the heart of a deprived community which will be more flexible than a hospital clinic will encourage the patients to make use of the facilities provided for them at the clinic, and to determine whether by this means the perinatal outcome can be improved.

CHAPTER XV.

SUMMARY.

Chapter I. Introduction.

(1) A historical review of the growth of the City of Glasgow was undertaken to show how the adverse social conditions in Glasgow developed.

(2) The history of the development of the maternity and child welfare was traced, as was the Corporation ideas on town planning. It was seen that although public health measures were improving, conditions remained dreadful for a large number of Glasgow families and that the perinatal mortality rate in the City of Glasgow has been consistently higher than that for Scotland.

(3) At this time of reorganisation of the health services in general and the medical services in particular, it was felt important to assess the problems in a bad area. This study was therefore undertaken to analyse the causes of the perinatal deaths in the City of Glasgow and attempt to determine the reasons for the high perinatal mortality rate. Such a study has not been undertaken previously in Glasgow.

Chapter II. The Survey.

(1) A review of previous reports on perinatal mortality in Glasgow was undertaken which showed that the selection of patients for hospital delivery was not very efficient, and that an appreciable number of high risk births continued to occur at home.

(2) Other significant factors were shown to be a high birth rate, a large population of high parity births, adverse socio-economic conditions (both past and present) and poor maternal physique.

(3) This study had three aims -

- (a) to classify all perinatal deaths in 1970 by clinical cause,
- (b) to identify the women who were losing their babies and to determine whether they came for and received adequate antenatal care,
- (c) to determine what changes, if any, might be recommended in obstetric and paediatric care to improve the perinatal outcome.

(4) Perinatal deaths relating to Glasgow births in 1970 were identified from data held in the computer-based system of linked child health records. Completeness of ascertainment was confirmed by the examination of hospital records. The clinical cause of each death was then determined and coded, using the classification designed by Baird, Walker and Thomson (1954).

#### Chapter III. Classification of Perinatal Deaths.

The Aberdeen classification was used and the following eight main groups were differentiated:

- (1) Mature - cause unknown, birth weight over 2500 g.
- (2) Premature - cause unknown, birth weight 2500 g. or less.
- (3) Trauma.
- (4) Toxaemia.
- (5) Antepartum haemorrhage.
- (6) Maternal Disease.
- (7) Fetal deformity.
- (8) Other (Rhesus factor, infection, etc.)

#### Chapter IV. Maternal Characteristics.

(1) During 1970 there were 16,748 births to women resident in Glasgow. There were 462 perinatal deaths and the records of 437 cases were studied in detail. Two-thirds of the deaths belonged to the environmental group of deaths (unexplained prematurity, fetal defects and antepartum haemorrhage) and are less amenable to obstetrical intervention, being related to the effects of unfavourable environmental influences on the mother's reproductive function.

(2) The risk of a perinatal death was shown to be raised in mothers aged less than 20 and above 30 in primiparae, at high parities and in Social Classes IV and V. The risk was also high in illegitimate primiparous births, domiciliary confinements and births where there was a previous history of abortion, stillbirth or neonatal death.

Chapter V. Perinatal Deaths due to -

(a) Mature - Cause Unknown.

(1) The women at risk in this group were found to be not the elderly primigravidae as expected, but the highly parous patients.

(2) Of the 43 patients delivered in hospital, intra-uterine death occurred in 26 cases. To improve the perinatal outcome, therefore, a change in obstetric management must take place before labour begins and methods whereby the fetus can be monitored during the antenatal period are discussed.

(b) Premature - Cause Unknown.

(1) Low birth weight babies were divided into two main groups, (a) pre-term infants, and (b) term infants who were light-for-dates.

(2) The mothers of the 53 pre-term infants in single pregnancies attended late for antenatal care; 17 women attended for their first visit after the 19th week of pregnancy, and 18 women received no care at all.

(3) Methods whereby an attempt to prevent pre-term labour were discussed, but it appeared that the problem is to identify the patient at risk and unfortunately this study makes very little contribution to the solution.

(c) Trauma.

(1) Perinatal deaths due to birth trauma can be divided into three main groups, -

- (a) breech delivery,
- (b) cord complications,
- (c) other causes.

Three patients delivered at home and a fourth patient delivered in hospital but had

received no previous care. It was felt that in all the breech deliveries (5), in one of the cases with cord complications, and in all the other traumatic deliveries (6), closer supervision of the patients during labour was required, and in the case of the breech presentations the obstetrician must be prepared to carry out a Caesarean Section as soon as any sign of fetal distress or obstruction occurs.

(d) Toxaemia.

(1) There were 29 deaths in this group. Although the aetiology of the condition remains unknown and treatment can only be empirical, 17 of the babies were born after 33 weeks gestation; more intensive antenatal care might therefore have improved the outcome.

(e) Antepartum Haemorrhage.

(1) There were 94 deaths due to antepartum haemorrhage.

(2) There was a high perinatal loss in those over 40 years of age, of high parity and of Social Class V.

(3) There were 8 deaths due to placenta praevia,  
62 deaths due to abruptio placentae,  
20 deaths due to other bleeding.

(4) In general, the care of these patients who lost their babies because of antepartum haemorrhage was unsatisfactory, and it was felt that a number of deaths might have been prevented by more active obstetric management.

(f) Maternal Disease.

There were 12 perinatal deaths attributed to maternal disease. Three of these were due to maternal pre-diabetic condition.

It was suggested that, as the number of diabetic and pre-diabetic patients is relatively few in each hospital, care might be concentrated in one centre for the Glasgow region.

(g) Fetal Deformity.

(1) There were 105 deaths due to abnormality, 63 occurring in the central

nervous system, 46 of which were due to anencephaly.

(2) There was a larger percentage of Social Class IV and V (46 per cent) compared with all deliveries in Glasgow (32 per cent). There was also an area difference of central nervous system abnormalities.

(3) Until the aetiology of the various abnormalities is known, it is unlikely that the perinatal mortality rates will be greatly reduced. Considerable maternal suffering could be lessened by earlier diagnosis of the condition, and termination of the pregnancy at that point offered to the patient.

#### (h) Rhesus Incompatibility and other Causes of Perinatal Death.

(1) Seventeen of the 23 deaths in this group were due to Rhesus incompatibility. Ten of these patients did not attend until the 20th week of pregnancy or later for antenatal care, and two received no care despite the fact that they had both lost babies because of this condition.

(2) Details of the patients are given and modern methods of treatment discussed.

(3) To improve the perinatal outcome the patients must be seen early in pregnancy so that amniocentesis can be carried out. The importance of this must be stressed to all Rhesus negative patients.

(4) To ensure that the patient receives expert care, she ought to be seen at a special clinic for such patients. For a city the size of Glasgow, only three or four such centres would be required.

#### Chapter VI. Weight Gain.

(1) The maternal weight/height ratio - weight gain between 20 and 30 weeks gestation and percentiles for baby weight were calculated where possible.

(2) Where details were known, it was found that the women who lost their babies were heavier than they ought to be for their height; 27 per cent of them failed to gain adequate weight and 60 per cent of the babies were growth retarded.

(3) As the normal weight gain pattern of Glasgow patients is not known,

this is an area of antenatal care where much more research is required.

(4) It is suggested that a greater awareness of the importance of weight gain or failure to gain weight might lead to more active management and assessment of the intra-uterine state of the fetus.

#### Chapter VII. Neonatal Findings.

(1) There was not enough information in the hospital records kept during the neonatal period for detailed retrospective analysis.

(2) It was found that 78 babies were known to have an Apgar score of less than 5 at the first count, 100 babies suffered from some form of respiratory distress, and 43 babies had an intracranial haemorrhage.

(3) It is suggested that in a city the size of Glasgow there should be three specialist maternity units with intensive neonatal care facilities where the most difficult obstetric and "at risk" deliveries take place.

(4) A high standard of antenatal care is required so that high risk patients are detected early, and arrangements can be made for delivery at a specialist maternity centre.

#### Chapter VIII. Multiple Births.

(1) Thirty-three perinatal deaths occurred in multiple (see twin) pregnancies. In 10 pregnancies both babies died, and in 13 further pregnancies one baby died. Twenty-five deaths were due to the cause, premature - cause unknown.

(2) Diagnosis of twin pregnancy can now be made very early in pregnancy with the aid of ultrasound. Early diagnosis of twin pregnancy, close supervision during pregnancy, and an attempt to prevent premature labour ought to improve the perinatal outcome in twin pregnancies.

#### Chapter IX. Domiciliary Deliveries.

(1) The 34 cases of domiciliary delivery are discussed in detail. Nine deaths occurred to patients booked for home confinement, 13 patients were booked for hospital delivery and 12 patients had no arrangements made for delivery.

(2) Only 3 of the patients booked for delivery at home were considered suitable for this by age, parity, height and previous history criteria.

(3) Proper selection of patients for domiciliary delivery and quick referral to hospital as soon as complications develop would help to reduce the number of perinatal deaths in this group of women.

#### Chapter X. Perinatal Mortality by Place of Confinement.

(1) In the City of Glasgow during 1970 there were 9 Maternity Hospitals, 3 General Practitioner Units and 2 Nursing Homes where deliveries occurred. The perinatal mortality rate varied from centre to centre and the causes of death were different.

(2) The gestation length at referral was not known for 42 per cent of patients, and 12 per cent of patients were not referred until after the 19th week of pregnancy.

(3) The gestation length at the first antenatal visit was not known for 27 per cent of patients, and 36 per cent of patients were not seen until after the 19th week of pregnancy.

(4) The time interval from referral to the first antenatal visit varied from hospital to hospital, but 57 patients who had a perinatal death had to wait more than six weeks for their first appointment.

#### Chapter XI. Housing Conditions.

(1) A description of the poor housing conditions in Glasgow is given.

(2) Fifty point four per cent of all Glasgow families having a baby in 1970 lived in overcrowded conditions (39.3 per cent of all families with perinatal deaths).

(3) Perinatal death by ethnic group was analysed but there was no unusual finding, although the India/Pakistan group had a slightly larger proportion of deaths than would be expected from their number resident in the City of Glasgow.

Chapter XII. Geographical Distribution of Perinatal Deaths.

(1) The perinatal mortality rates by ward of the City of Glasgow were studied.

(2) The general findings were that in all categories of death apart from central nervous system abnormality the east side of the city, particularly Provan, where there was a large number of births, had a very high perinatal mortality rate.

(3) Ways in which antenatal care might be improved for the patients in this area are discussed.

Chapter XIII. Comparison of Clinical and Pathological Classification.

(1) The clinical cause of death was cross-tabulated with the registered cause of death. There was only close agreement in two groups-fetal deformity (86 out of 103 deaths) and Rhesus isoimmunisation (14 out of 17 deaths).

(2) From this study it would appear that the analysis of perinatal deaths by registered cause is of limited value. The main reason for this is that there are too many rather non-specific categories.

(3) It is suggested that the classification devised by Baird, Walker and Thomson (1954) is short, simple, efficient and could be easily applied by all clinicians working in the perinatal field. This would be of much greater value than studying the registered cause of death, would illustrate specific problems, and would show where improvement in clinical management was required, thus reducing perinatal mortality in the future.

Chapter XIV. Discussion.

(1) The problems in attempting to organise antenatal care in a city the size of Glasgow are discussed. The organisation of antenatal care in Aberdeen is discussed.

(2) Methods whereby the perinatal mortality rate in the City of Glasgow can be improved are looked at from the point of view of -

- (a) immediate action,
- (b) long term planning.

(3) Future Work. As a result of this study three further studies have been started and one is projected -

- (1) Survey of all perinatal deaths in 1974.
- (2) Epidemiological study of pre-term deliveries.
- (3) Study of antenatal clinic attendance.
- (4) An antenatal clinic in a deprived area, to be started in 1975.

APPENDIX.

PROFORMA FOR PERINATAL MORTALITY STUDY.



**PREVIOUS HISTORY:** Abortions  
 Livebirths  
 Stillbirths (specify cause)  
 Total births  
 Total pregnancies  
 N.N.D. (specify cause)  
 Complications: Code 0 No 1 Yes  
 Antepartum haemorrhage  
 Toxaemia  
 Rhesus incompatibility  
 Low birth weight (2500 g. and under)  
 Cervical incompetence  
 Caesarean Section  
 Congenital abnormality (specify)  
 Other (specify)

**PHENNANCY:** L.M.P. / / Cycle ..... 1 Regular 2 Irregular  
 E.D.D. / / 1 Sure 2 Not sure

**Antenatal Care:**

Gestation at time of G.P.'s referral/letter  
 Gestation at 1st visit to hospital, clinic, midwife.  
 Booking - Place 0 Nil 1 Hospital 2 Home 4 Nursing Home  
 Care from: 1 G.P. 2 Hospital 4 LMA 8 Other  
 Visits to hospital ANC (number)  
 Other antenatal visits (number)  
 Hospital admission

**Antenatal Complications:** Hyperemesis (admitted)  
 Previous renal disease  
 Urinary tract infection  
 Cardiac disease  
 Diabetes  
 Anaemia  
 Fibroids or ovarian tumour  
 Hypertension - transient, B.P.:  
 - sustained, B.P.:  
 Proteinuria  
 Eclampsia

**Code** 0 No  
 1 Yes  
 unless  
 otherwise  
 stated

Rh negative with antibodies  
 Multiple pregnancy  
 Bleeding before 24 weeks  
 Bleeding after 24 weeks  
     1 Placenta praevia  
     2 Abruptio placentae  
     4 Other or not known  
 Hydramnios  
 Breech presentation  
 Unstable lie  
 Premature rupture of membranes  
 Intra-uterine infection  
 Other (specify)

Antenatal Procedures: ECV: 0 No 1 With GA 2 Without GA  
 Amniocentesis  
 Intra-uterine transfusion  
 Other (specify)

Weight Increase: Weight (lb) at first visit  
                                     20 weeks  
                                     30 weeks

Code: Increase, 20-30 weeks  
 1 Light Weight/height ratio (20 weeks)  
 2 Normal Weight gain ratio (20-30 week increase)  
 3 High

LABOUR: Place (code hospital, etc.)  
 Date of Delivery  
 Gestation (completed weeks)  
 Induction 0 None 2 IV Syntocinon  
           1 ARM 4 Buccal pitocin  
 Interval, rupture-delivery: 1 -12 2 12- 3 24-  
                                   4 36- 5 48+  
 Duration: Total 1 -12 2 12- 3 24-  
                                   4 36- 5 48+  
 2nd Stage 1 -1 2 1- 3 1½+  
 Method: 0 Spontaneous 1 Forceps 2 Breech  
           3 Vacuum 4 Section (LS)  
           5 Section (Classical) 6 NK

Operative Delivery: Inco-ordinate uterine action  
 Failed or trial forceps  
 Disproportion

Code: Contracted pelvis  
 0 No or NA Foetal Distress  
 1 Yes Variable lie (more than 36 wks)  
 Prolapsed Cord  
 Shoulder presentation  
 Face/Brow/Compound  
 O.P.  
 Transverse Lie  
 Medical reasons (specify)  
 Other reason (specify)  
 General Anaesthetic

Foetal Distress: 0 None 1 FH 2 Meconium 3 Both  
 Foetal Heart: On admission - 0 Not heard 1 Heard  
 Absent before delivery -  
 0 Not applicable 3 12 hrs.-  
 1 Less than 1 hr. 4 24 hrs +  
 2 1 hour -

INFANT: Age at death 0 stillborn 1 - 7 for day  
 Sex 1 M 2 F  
 Weight (grams)  
 Maturity (percentile) 1 -5 4 25- 7 90-  
 2 5- 5 50- 8 95+  
 3 10- 6 75-

Placental weight (grams)

Survey No.

IF STILLBORN, LEAVE THIS PAGE EMPTY (except survey number)

Apgar score (first) Code 1,2,3, ..... 8,9,0      Score  
Minutes

Complications and treatment

(except where stated, code 0 No 1 Yes)

Resuscitation    0 None                    4 Hyperbaric  
                  1 IPPR intubation    8 Other  
                  2 IPPR face mask

Suction

Respiratory distress    All causes  
                                  RDS  
                                  Infection  
                                  Aspiration  
                                  Pneumothorax  
                                  Other (specify)  
                                  NK

Birth injury            IC haemorrhage (all types)  
                                  IC membrane tear    Falx  
                                                                  Tentorium  
                                                                  Other  
                                  IC haemorrhage - subdural  
                                                                  subarachnoid  
                                                                  intraventricular  
                                                                  Other  
                                  Other birth injury (specify)

Pneumonia    1 prenatal congenital.    2 aspiration.    4 postnatal

Other infection (specify)

Jaundice    1 Haemolytic disease (specify)  
                  2 Other causes (specify)

Electrolyte imbalance (specify)

Hypoglycaemia (less than 20)

Hypocalcaemia (less than 8)

Anaemia    1 Presumptive blood loss (specify and whether clinical  
                                                                  or pathological evidence)  
                  2 Other (specify)

Other complications (specify)

Transfusion    0 No    1 Exchange    2 Booster

IV Fluids      0 No    1 Short (-1 hr)    2 Long term

Antibiotics

Mechanical Ventilation

Other (specify)

---

TRANSFERS:      Mother   0 No  
                                 1 Antenatal (hospital to hospital)  
                                 2 In labour (hospital to hospital)  
                                 3 Emergency (home to hospital)

                                 Infant   0 No  
                                                 1 Home to hospital  
                                                 2 Hospital to hospital (specify from where)  
                                                 4 Not applicable

---

AUTOPSY REPORT:      0 No    1 Yes

Details -

---

REGISTERED CAUSE:

---

CLINICAL CAUSE OF DEATH:      \*Code   0 No    1 Yes

- 1 Mature - cause unknown
- 2 Premature - cause unknown
- \*3 Trauma
- \*4 Toxaemia
- \*5 APH
- \*6 Maternal Disease (specify)
  - 7 Foetal defect    0 None
  - 1 Anencephaly
  - 2 Spina bifida
  - 3 Hydrocephalus
  - 4 CHD (specify)
  - 5 Alimentary
  - 6 Other (specify)
- \*8 Other (specify)

Leading cause (code 1 - 8)

---

REFERENCES.

- ALBERMAN, H. (1974) Factors influencing perinatal wastage. Clinics in Obstet. Gynec. 1:1
- ARGAR, V. (1953) A proposal for a new method of evaluation of the newborn. Anaesth. and Analg. 32: 260
- ARNOTT, N. (1840) Official Report to Poor Law Commissioners.
- BAIRD, D. (1964) The Epidemiology of Prematurity. J. Pediat. 65: 6: 909
- BAIRD, D. (1969) Perinatal Mortality. Lancet, 1: 511
- BAIRD, D. (1970) Perinatal Mortality. Develop. Med. Child Neurol., 12: 368
- BAIRD, D. (1973) Social influences on reproductive performance. J. Reprod. Fert., Suppl. 19, 585
- BAIRD, D. (1974) in Environmental Medicine. Ed. Howe, G.M. Lorraine J.A. Ch. 13. Environmental Factors in Relation to Obstetrics. Wm. Heinemann Medical Books Ltd.
- BAIRD, D. and THOMSON, A.M. (1969) in Perinatal Problems. p. 2, 217: 277 Ed. Butler, N.R. and Alberman, E.D. Edinburgh and London, Livingstone.
- BAIRD, D., THOMSON, A.M. and BILLIOWICZ, W.Z. (1957) Birth weights and placental weights in pre-eclampsia. J. Obstet. Gynaec. Brit. Emp., 64: 370
- BAIRD, D., THOMSON, A.M. and DUNCAN, E.M.L. (1953) Causes and prevention of stillbirths and first week deaths; evidence from Aberdeen clinical records. J. Obstet. Gynaec. Brit. Emp., 60: 17
- BAIRD, D., WALKER, J. and THOMSON, A.M. (1954) The causes and prevention of stillbirths and first week deaths. J. Obstet. Gynaec. Brit. Emp., 61: 433
- BELLY, J.S. and KURLAND, I.I. Relationship of maternal weight gain and weight of newborn infant. (1945) Amer. J. Obstet. Gynec., 50: 202

- BINGHAM, A.W. (1932) The prevention of obstetric complications by diet and exercise. Amer. J. Obstet. Gynec., 23: 38
- BLAIR, R.G. (1973) Abruptio of the placenta. A review of 189 cases occurring between 1965 and 1969. J. Obstet. Gynaec. Brit. Cwlth., 80: 242
- BROCK, D.J., and SUTCLIFFE, R.G. (1972) Alpha - fetoprotein in the antenatal diagnosis of anencephaly and spina bifida. Lancet, ii, 197
- BULMER, M.G. (1970) The biology of twinning in man. p. 68 Clarendon Press, Oxford.
- BUTLER, N.R. (1967) Causes and prevention of perinatal mortality. W.H.O. Chronicle, 21: 43: 67
- BUTLER, N.R. (1974) Nutrition and Fetal Development. Ed. Myron Winick, Ch. 9. Late postnatal consequences of fetal malnutrition. John Wiley and Sons, New York, London, Sydney, Toronto.
- BUTLER, N.R. and ALBERMAN, E. (1969) Ed. Perinatal Problems. Livingstone, Edinburgh and London.
- BUTLER, N.R. and BONHAM, D.G. (1963) Ed. Perinatal Mortality. Livingstone, Edinburgh and London.
- CAMERON, M. (1889) The Caesarian Section: With notes of a successful case. B.M.J. 1: 180
- CAMERON, M. (1890) Remarks on Caesarian Section with notes of a second successful case. B.M.J. 1: 1524
- CHALMERS, A.K. (1930) The Health of Glasgow, 1818-1925. An outline. Bell and Bain, Glasgow.
- CHURCHILL, J.A., NEFF, J.W. and CAULDWELL, D.F. (1966) Birth weight and intelligence. Obstet. Gynec., 28: 425
- CLARKE, D.A. (1968) Prevention of Rhesus iso-immunisation. Lancet, ii: 1
- COURT, D. and JACKSON, A. (1972) Paediatrics in the Seventies. Developing the Child Health Services. p. 45 Published for the Nuffield Provincial Hospitals Trust by the Oxford University Press.
- COWAN, R. (1837) Statistics of fever and smallpox in Glasgow. Read to Statistical Society of Glasgow, 1837.

- COWAN, R. (1840) in *The Dawn of Scottish Social Welfare. A Survey from mediaeval times to 1863.*  
Author, Ferguson, T.  
Thomas Nelson and Sons, Ltd., London, Edinburgh,  
Paris, Melbourne, Toronto and New York.
- CROSSIE, V.M. (1971) *The pre-term baby and other babies with low birth weight.*  
Churchill Livingstone, Edinburgh and London.
- CUMMING, H.A. (1934) *An interpretation of weight changes during pregnancy.*  
*Amer. J. Obstet. Gynec.*, 27: 808
- DAVIE, R., BUTLER, N.R. and GOLDSTEIN, H. (1972) *From Birth to Seven. A report of the National Child Development Study.*  
Longmans in association with the National Children's Bureau.
- DAVIS, C.H. (1923) *Weight in pregnancy; its value as a routine test.*  
*Amer. J. Obstet. Gynec.*, 6: 575
- DAW, B. (1974) *Twin Pregnancy - The effect of recent socio-ecological factors.*  
*Health Bulletin (Edinb.)* 32: 91
- DEPARTMENT OF HEALTH AND SOCIAL SECURITY (1971) *Report of the Expert Group on Special Care for Babies.* p. 6.  
London. Her Majesty's Stationery Office.
- DONALD, I. (1969) *Practical Obstetric Problems, 4th Edition.* p. 425  
Lloyd-Luke, London.
- DUFFUS, G.M., MacGILLIVRAY, I. and DENNIS, K.J. (1971) *The relationship between baby weight and changes in maternal weight, total body water, plasma volume, electrolytes and proteins and urinary oestriol excretion.*  
*J. Obstet. Gynaec. Brit. Com.,* 78: 97
- DUFFUS, G.M., FUNSTALL, M.B. and MacGILLIVRAY, I. (1968) *Intravenous Chlormethiazole in pre-eclamptic toxemia in labour.*  
*Lancet*, i, 335
- DUNCAN, E.H.L., BAIRD, D. and THOMSON, A.M. (1952) *The causes and prevention of stillbirths and first week deaths, Part I. The evidence of vital statistics.*  
*J. Obstet. Gynaec. Brit. Emp.*, 59: 183
- EASTMAN, N.J. and JACKSON, H. (1968) *Weight relationships in pregnancy.*  
I. *The bearing of maternal weight gain and pre-pregnancy weight on birth weight in full term pregnancies.*  
*Obstet. Gynec. Survey*, 23: 11: 1

- EDWARDS, J.H. (1958) Congenital malformations of the central nervous system in Scotland. Brit. j. prev. Soc. Med., 12: 115
- ERIKSSON, M., CATZ, C.S. and JAFFE, S.J. (1973) Drugs and Pregnancy. Clin. Obstet. Gynaec. 16: 199
- FARR, W. (1839) Registrar General of England and Wales First Annual Report. p. 99
- FEDRICK, J. and WILSON, T.S. (1971) Malformations of the central nervous system in Glasgow. Brit. j. prev. Soc. Med., 25: 210
- FIELD, B. and KERR, C. (1973) Potato blight and neural tube defects. Lancet, 11, 507
- FINN, R. (1960) Liverpool Medical Institution: Symposium on the role of inheritance in common diseases. Erythroblastosis. Lancet, 1: 526
- FISH, J.S., BARTHOLOMEW, R.A., COLVIN, E.D. and GRIMES, W.H. (1951) The role of marginal sinus rupture in antenatal haemorrhage. Amer. J. Obstet. Gynec., 61: 20
- FUCHS, F., FUCHS, A.R., POBLENTE, V.F. and RISK, A. (1967) Effect of alcohol on threatened premature labor. Amer. J. Obstet. Gynec., 29: 627
- GRAY, M.J. (1968) Use and abuse of thiazides in pregnancy. Clin. Obstet. Gynec., 11: 568
- GUARDIAN, THE (1974) Open file. 26: 9: 74
- GUTTMACHER, A.F. and KOHL, S.G. (1958) The fetus of multiple gestations. Obstet. Gynec., N.Y. 12: 528
- HANLEY, B.J. (1934) Gain of weight in pregnancy in relation to weight of newborn. West. J. Surg. 42: 251
- HILLMAN, L.M. (1970) Personal Communication.
- HILLMAN, L.M. and PRITCHARD, J.A. in Williams Obstetrics, 14th Edition. p. 634. (1971) Butterworth, London.
- HIBBARD, B.M. and JEFFCOATE, T.N.A. (1966) Abruptio placentae. Obstet. Gynec., 27: 155
- HOLMAN, R., LABITTE, F., SPENCER, K. and WILSON, H. (1970) Socially deprived families in Britain. p. 211 The Bedford Square Press of the National Council of Social Service.

- HOWE, G.M. (1972) Ed. Atlas of Glasgow and the West Region of Scotland. Holmes McDougall, Edinburgh.
- HYTTEN, F.E., and LEITCH, I. (1971) The Physiology of Human Pregnancy, p. 284. Blackwell Scientific Publications, Oxford.
- HYTTEN, F.E., THOMSON, A.M. and TAGGART, N. (1966) Total body water in normal pregnancy. J. Obstet. Gynaec. Brit. Cwlth., 73: 553
- ILLSLEY, R. (1967) in Childbearing - Its Social and Psychological Aspects. Eds. Richardson, G.A., Guttmacher, A.P. p. 75. The Williams and Williams Company.
- KIMSLEY, W.F.F., BILLEWICZ, W.Z. and THOMSON, A.M. (1962) A new weight-for-height standard based on British anthropometric data. Brit. j. prev. Soc. Med. 16: 189
- KLOPPER, A. (1969) in Foetus and Placenta, p. 511. Ed: Klopper, A., Diezfalusy, E. Blackwell Scientific Publications, Oxford and Edinburgh.
- LAW, R.J. (1967) Standards of obstetric care; the report of the North West Metropolitan Regional Obstetric Survey. Livingstone, Edinburgh.
- LEADER (1974) The Initiation of Labour. Lancet, i: 124
- LECHWORTH, A.T. and CHARD, T. (1972) Placental lactogen levels as a screening test for fetal distress and neonatal asphyxia. Lancet, i, 704
- LILEY, A.W. (1965) Amniocentesis and Amniography in hemolytic disease. Year Books of Obstetrics and Gynecology, 1964-65, p. 256. Year Book Medical Publishers, Chicago.
- LIEWELYN-JONES, D. (1974) Human Reproduction and Society, p. 457. Faber and Faber, London.
- LOBBER, J., STEWART, C.R. and MILFORD WARD, A. (1973) Alpha fetoprotein in antenatal diagnosis of anencephaly and spina bifida. Lancet, i, 1187
- LOVE, E.J. and KINCH, R.A.M. (1965) Factors influencing the birth weight in normal pregnancy. Amer. J. Obstet. Gynec., 91: 342

- LOWE, C.R., ROBERTS, C.J. and LLOYD, S.  
(1971) Malformations of central nervous system and softness of local water supplies.  
Br. Med. J., 2: 357
- LUNAN, C.B. (1973) The management of abruptio placentae.  
J. Obstet. Gynaec. Brit. Cwlth., 80: 120
- MacGILLIVRAY, I. (1961) Hypertension in pregnancy and its consequences.  
J. Obstet. Gynaec. Brit. Cwlth., 68: 557
- MacGILLIVRAY, I. (1967) The significance of blood pressure and body water changes in pregnancy.  
Scot. Med. J., 12: 237
- MacGILLIVRAY, I. and DAVEY, D.A. (1963) Hypertension in late pregnancy.  
Biochemical Clinics, 2: 299
- MacGREGOR, A. (1967) Public Health in Glasgow, 1905 - 1946, p. 113.  
E.S. Livingstone, Ltd., Edinburgh and London.
- McILROY, A.L. and RODWAY, H. (1937) Weight changes during and after pregnancy with special reference to the early diagnosis of toxæmia.  
J. Obstet. Gynaec. Brit. Emp., 44: 221
- MACNAUGHTON, M.C. (1974) Antenatal Care.  
The Practitioner, 212: 633
- MANSLEY, R.D. (1973) Housing and social deprivation.  
Department of Planning, Corporation of the City of Glasgow.
- MANUAL OF THE INTERNATIONAL STATISTICAL CLASSIFICATION OF DISEASES, INJURIES AND CAUSES OF DEATH (1967) World Health Organisation, Geneva, 1: IX
- MILIC, A.B. and ADAMSON, K. (1969) The relationship between anencephaly and prolonged pregnancy.  
J. Obstet. Gynaec. Brit. Cwlth., 76: 102
- MONTGOMERY REPORT - DEPARTMENT OF HEALTH FOR SCOTLAND (1959) Maternity Services in Scotland.  
Her Majesty's Stationery Office, Edinburgh.
- MURDOCH, D. and FOULKES, J.F. (1952) Antepartum Haemorrhage.  
J. Obstet. Gynaec. Brit. Emp., 59: 786
- NELSON, T.R. (1955) A clinical study of pre-eclampsia.  
J. Obstet. Gynaec. Brit. Emp., 62: 48
- NEVIN, N.C., NESBITT, S. and THOMPSON, W. (1973) Myelocoele and alpha-fetoprotein in amniotic fluid.  
Lancet, 1: 1383
- PAINTIN, D.B. (1962) The epidemiology of antepartum haemorrhage. A study of all births in a community.  
J. Obstet. Gynaec. Brit. Cwlth., 69: 614

- FREEL, J. (1969) Introduction to Perinatal Problems.  
Eds. Butler, N.R. Alberman, E.  
Livingstone, Edinburgh and London.
- RAE, J.H. (1974) Family Circumstance and Location of the  
Socially Deprived.  
Department of Planning, Corporation of the  
City of Glasgow.
- RAJA, R.L. TAMBY, ANDERSON, A.B.M., and TURNBULL, A.C.  
(1974) Endocrine changes in premature labour.  
Brit. Med. J. 4: 67
- REGISTRAR GENERAL SCOTLAND Annual Report.  
(1969) Her Majesty's Stationery Office, Edinburgh.
- REGISTRAR GENERAL SCOTLAND CensuS (10 per cent sample).  
(1966) County Report for Glasgow City.  
Her Majesty's Stationery Office, Edinburgh.
- REGISTRAR GENERAL SCOTLAND CensuS Report. The County Report for  
(1971) Glasgow City.  
Her Majesty's Stationery Office, Edinburgh.
- RENEWICK, J.H. (1972) Hypothesis. Anencephaly and spina bifida are  
usually preventable by avoidance of a specific  
unidentified substance present in certain  
potato tubers.  
Brit. J. prev. Soc. Med., 26: 67
- REPORT (1970) Medical Officer of Health, City of Glasgow,  
produced by the Corporation of the City of  
Glasgow.
- REPORT (1972) Medical Officer of Health, City of Glasgow,  
produced by the Corporation of the City of  
Glasgow.
- RICHARDS, I.D.G., DONALD, E.M. and HAMILTON, F.M.W.  
(1970) The use of maternity care in Glasgow.  
In McLachlan, G. Shegog, R.F. Eds.  
In the beginning: Studies of maternity services,  
p. 105. Oxford University Press, London.
- RICHARDS, I.D.G., HAMILTON, F.M.W. and NICHOLSON, M.F.  
(1969) Perinatal Mortality in Glasgow.  
Health Bulletin (Edinb.), 27: 43
- RICHARDS, I.D.G., McINTOSH, H.T. and SWENNIE, S.  
(1972) A genetic study of anencephaly and spine bifida  
in Glasgow.  
Dev. med. Child. Neurol., 14: 626

- RICHARDS, I.D.G., and NICHOLSON, M.F.  
(1970) The Glasgow linked system of child health records.  
Dev. med. Child Neurol., 12: 357
- RICHARDS, I.D., ROBERTS, C.I. and LLOYD, S.  
(1972) Area differences in prevalence of neural tube malformations in South Wales.  
A study of possible demographic determinants.  
Br. J. Prev. Soc. Med., 26: 89
- ROGERS, S.C. (1969) Epidemiology of stillbirths from congenital abnormalities in England and Wales 1961-66.  
Dev. Med. Child Neurol., 11: 617
- RUSSELL, J.B. (1905) Public health administration in Glasgow. p. 314.  
Ed. Chalmers, A.K.  
James Maclehose and Sons, Glasgow.
- SCOTTISH HOME AND HEALTH DEPARTMENT  
(1973a) Community Medicine in Scotland. p. 18  
Her Majesty's Stationery Office, Edinburgh.
- SCOTTISH HOME AND HEALTH DEPARTMENT  
(1973b) The integration of maternity work.  
Her Majesty's Stationery Office, Edinburgh.
- SCOTTISH HOME AND HEALTH DEPARTMENT  
(1973c) Towards an Integrated Child Health Service.  
p. 38, 54.  
Her Majesty's Stationery Office, Edinburgh.
- SINGER, J.E., WESTPHAL, M. and NISWANDER, K.  
(1968) Relationship of weight gain during pregnancy to birth weight and infant growth and development in the first year of life.  
A report from the collaborative study of cerebral palsy.  
Obstet. Gynec., 31: 417
- SLEMONS, J.M. and PAGAN, R.H.  
(1927) A study of the infants birth weight and the mothers weight gain during pregnancy.  
Amer. J. Obstet. Gynec., 14: 159
- SMITH, A. and MACDONALD, I.S.  
(1965) Social circumstances related to childbearing in Glasgow, 1963.  
Health Bulletin (Edinb.), 23: 3
- SMITH, A., RICHARDS, I.D.G., NICHOLSON, M.F. and GRANICK, E.  
(1970) The Glasgow linked system of child health records. in Nuffield Provincial Hospitals Trust.  
Problems and Progress in Medical Care. p. 145.  
(Essays on Current Research, 4th series. Ed. G. McLachlan).  
Oxford University Press., London.

- SWALLOW, K.A. (1972) The Baltimore Maternal and Infant Care Project. Annals of the New York Academy of Sciences, 196: 84
- SWENNIE, S. (1974) Personal Communication.
- SYMONDS, J.G. (1838) Reports from Assistant Handlooms Weavers Commissioners. Parliamentary Paper issued 27.3.1839.
- TALBERT, L.M. (1972) The Rh Problem in Obstetrics. Self instructional materials project. School of Medicine, University of North Carolina, Chapel Hill.
- THOMSON, A.M. and BILLEWICZ, W.Z. (1963) Nutritional Status, Maternal Physique and Reproductive Efficiency. Proc. Nutr. Soc., 22: 55
- THOMSON, A.M., BILLEWICZ, W.Z. and HYTHEN, F.E. (1968) The assessment of fetal growth. J. Obstet. Gynaec. Brit. Cwlth., 75: 903
- THOMSON, A.M., HYTHEN, F.E. and BILLEWICZ, W.Z. (1967) The epidemiology of oedema during pregnancy. J. Obstet. Gynaec. Brit. Cwlth., 74: 1
- THOMPSON, B. and AITKEN-SWAN, J. (1973) Pregnancy outcome and fertility control in Aberdeen. Brit. J. Prev. Soc. Med., 27: 137
- TOOMBS, P.W. (1931) The relationship between mother's gain during pregnancy and infant's birth weight Amer. J. Obstet. Gynec., 22: 851
- TOWNSEND, P. (1971) Introduction to Family Poverty. Programme for the Seventies. Ed. Bull.D. Gerald Duckworth and Co. Ltd., Published in association with the Child Poverty Action Group.
- TOWNSEND, P. (1974) Inequality and the Health Service. Lancet, i: 1179
- TURNBULL, A.C., FLINT, A.P.F., JEREMY, J.Y., PAPPEN, P.T., KEIRSE, M.J.N.C. and ANDERSON, A.B.M. (1974) Significant fall in progesterone and rise in oestradiol levels in human reproductive plasma before onset of labour. Lancet, i: 101
- TURNBULL, A.C., GREGORY, P.J. and LAWRENCE, K.M. (1973) Antenatal diagnosis of fetal abnormality with special reference to amniocentesis. Proc. Roy. Soc. Med., 66: 1115
- USHER, R.H. (1970) The role of the neonatologist. Pediat. Clin. N. Amer., 17: 199
- WATERHOUSE, J.A. (1950) Twinning in Twin Pedigrees. Brit. J. Soc. Med., 4: 197

- WALKER, J. (1954) Foetal Anoxia.  
J. Obstet. Gynaec. Brit. Emp., 61: 162
- WALKER, W. (1968) The management of Rhesus iso-immunisation.  
J. Obstet. Gynaec. Brit. Gwllth., 75: 1207
- WEDGE, P. and PROSSER, M. (1973) Born to Fail?  
Arrow Books, in association with the National  
Children's Bureau.
- WESSELIUS-de-CASPARIS, A., THIERY, M., YO-LE- SIAN, A., BAUMGARTEN, K.,  
BROSTENS, J., GAMISANS, O., STOLK, J.A. and VIVIER, W.  
(1971) Results of double-blind, multicentre study  
into Ritodrine in premature labour.  
Br. Med. J., 111: 144
- WHITFIELD, C.R., CHAN, W.H., SPROULE, W.B. and STEWART, A.D.  
(1972) Amniotic fluid lecithin - sphingomyelin ratio  
and fetal lung development.  
Br. Med. J., 11: 85
- W.H.O. EXPERT COMMITTEE ON MATERNAL AND CHILD HEALTH  
(1961) Public health aspects of low birth weight.  
Technical Report Series, No. 217.

ADDENDUM.

TABLE 1.

Gestation Length at Delivery by Clinical Cause of Death (Single births only)

|       | 30 wks. | 30      | 31     | 32     | 33     | 34     | 35     | 36     | 37     | 38     | 39     | 40      | 41     | 42     | 42+    | N/S     |
|-------|---------|---------|--------|--------|--------|--------|--------|--------|--------|--------|--------|---------|--------|--------|--------|---------|
| M.U.  | 50      | -       | -      | -      | -      | -      | 1      | 5      | 2      | 4      | 7      | 12      | 6      | 6      | 3      | 4       |
| P.U.  | 76      | 4       | 4      | 3      | 3      | 6      | 4      | 1      | 4      | 3      | 1      | 4       | 1      | 0      | 2      | 7       |
| Tr.   | 23      | -       | -      | 1      | -      | 1      | 1      | 1      | -      | 2      | 7      | 5       | 1      | -      | -      | 4       |
| Tox.  | 29      | 1       | 2      | 1      | -      | -      | 2      | 1      | 5      | 3      | -      | 3       | -      | 1      | -      | 4       |
| APH   | 91      | 7       | 5      | 6      | 5      | 7      | 2      | 7      | 5      | 5      | 2      | 4       | 3      | -      | -      | 16      |
| M.D.  | 10      | 1       | 1      | -      | -      | -      | -      | 2      | -      | -      | -      | 3       | -      | 1      | -      | 2       |
| F.D.  | 103     | 3       | 7      | 8      | 8      | 6      | 6      | 6      | 12     | 10     | 5      | 8       | 6      | -      | 1      | 12      |
| Other | 22      | 1       | 1      | 1      | 1      | 4      | 5      | 1      | -      | 3      | -      | 2       | -      | -      | -      | 1       |
| Total | 404     | 60      | 16     | 20     | 20     | 17     | 24     | 24     | 28     | 30     | 22     | 41      | 17     | 8      | 6      | 50      |
|       |         | (14.9%) | (4.0%) | (5.0%) | (5.0%) | (4.2%) | (5.9%) | (5.9%) | (6.9%) | (7.4%) | (5.4%) | (10.1%) | (4.2%) | (2.0%) | (1.5%) | (12.4%) |

TABLE 2.

Method of Delivery by Clinical Cause of Death.

|              | Total      | 0                            | 1                           | 2                           | 3                         | 4                          | 5                         | 6                         |
|--------------|------------|------------------------------|-----------------------------|-----------------------------|---------------------------|----------------------------|---------------------------|---------------------------|
| M.U.         | 50         | 38                           | 7                           | 1                           | 1                         | 2                          | 0                         | 1                         |
| P.U.         | 101        | 61                           | 6                           | 28                          | 0                         | 5                          | 0                         | 1                         |
| Tr.          | 23         | 2                            | 6                           | 10                          | 1                         | 2                          | 1                         | 1                         |
| Tox.         | 29         | 18                           | 4                           | 2                           | 0                         | 5                          | 0                         | 0                         |
| APH          | 94         | 55                           | 6                           | 23                          | 0                         | 8                          | 1                         | 1                         |
| M.D.         | 12         | 7                            | 1                           | 2                           | 0                         | 2                          | 0                         | 0                         |
| F.D.         | 105        | 58                           | 12                          | 22                          | 0                         | 10                         | 0                         | 3                         |
| Other        | 23         | 13                           | 3                           | 2                           | 0                         | 5                          | 0                         | 0                         |
| <b>Total</b> | <b>437</b> | <b>252</b><br><b>(57.7%)</b> | <b>45</b><br><b>(10.3%)</b> | <b>90</b><br><b>(20.6%)</b> | <b>2</b><br><b>(0.5%)</b> | <b>39</b><br><b>(8.9%)</b> | <b>2</b><br><b>(0.5%)</b> | <b>7</b><br><b>(1.6%)</b> |

- 0 = Spontaneous
- 1 = Forceps
- 2 = Breech
- 3 = Vacuum Extraction
- 4 = Caesarean Section (Lower uterine segment)
- 5 = Caesarean Section (Classical)
- 6 = Unknown.

TABLE 3.

Birth Weight (Kg.) by Clinical Cause  
of Death.

|       | Total. | 0+            | 1+             | 2+             | 3+            | 4+           | N/S          |
|-------|--------|---------------|----------------|----------------|---------------|--------------|--------------|
| M.U.  | 50     | 0             | 0              | 20             | 24            | 5            | 1            |
| P.U.  | 101    | 35            | 40             | 17             | 0             | 0            | 9            |
| Tr.   | 23     | 0             | 1              | 12             | 6             | 3            | 1            |
| Tox.  | 29     | 5             | 14             | 5              | 4             | 1            | 0            |
| APH   | 94     | 15            | 39             | 26             | 11            | 1            | 2            |
| M.D.  | 12     | 1             | 5              | 2              | 2             | 0            | 2            |
| F.D.  | 105    | 8             | 35             | 32             | 13            | 1            | 16           |
| Other | 23     | 2             | 6              | 8              | 4             | 1            | 2            |
| Total | 437    | 66<br>(15.1%) | 140<br>(32.0%) | 122<br>(27.9%) | 64<br>(14.6%) | 12<br>(2.8%) | 33<br>(7.6%) |

TABLE 4.

Sex of Infant by Clinical Cause  
of Death.

|       | Total. | Male. | Female. | N/S. |
|-------|--------|-------|---------|------|
| M.U.  | 50     | 28    | 21      | 1    |
| P.U.  | 101    | 57    | 42      | 2    |
| Tr.   | 23     | 12    | 11      | -    |
| Tox.  | 29     | 17    | 12      | -    |
| APH   | 94     | 68    | 26      | -    |
| M.D.  | 12     | 7     | 5       | -    |
| F.D.  | 105    | 51    | 53      | 1    |
| Other | 23     | 14    | 9       | -    |
| Total | 437    | 294   | 179     | 4    |

Male/Female Ratio = 100.0 : 70.5

TABLE 5.

Number of Perinatal Deaths by Ward of Residence and Clinical Cause.

| Ward.        | No. of Births. | No. of Deaths. | M.U.      | P.U.      | Tz.       | Tox.      | AHI.      | M.D.      | F.D.       | Other     |
|--------------|----------------|----------------|-----------|-----------|-----------|-----------|-----------|-----------|------------|-----------|
| 1.           | 676            | 10             | 2         | 4         | -         | -         | 3         | -         | 1          | -         |
| 2.           | 241            | 9              | -         | -         | 2         | 2         | 2         | -         | 3          | -         |
| 3.           | 615            | 17             | 2         | 3         | -         | 1         | 8         | -         | 2          | 1         |
| 4.           | 304            | 8              | 1         | 3         | -         | -         | 2         | -         | 1          | 1         |
| 5.           | 603            | 8              | 1         | 4         | 1         | -         | 1         | -         | -          | 1         |
| 6.           | 444            | 15             | 2         | 4         | 2         | -         | 1         | -         | 2          | 4         |
| 7.           | 1228           | 47             | 9         | 11        | 1         | 2         | 15        | 1         | 7          | 1         |
| 8.           | 628            | 12             | 1         | 3         | -         | -         | 4         | -         | 4          | -         |
| 9.           | 481            | 17             | 4         | 1         | -         | -         | 5         | 1         | 6          | -         |
| 10.          | 283            | 10             | -         | -         | -         | 2         | 5         | -         | 3          | -         |
| 11.          | 77             | 6              | 2         | -         | -         | -         | 2         | -         | 2          | -         |
| 12.          | 220            | 5              | 1         | -         | -         | -         | 1         | -         | 3          | -         |
| 13.          | 276            | 4              | -         | 2         | -         | -         | -         | -         | 2          | -         |
| 14.          | 233            | 6              | -         | 2         | -         | -         | 2         | -         | 2          | -         |
| 15.          | 317            | 6              | -         | 1         | 1         | -         | -         | 2         | 2          | -         |
| 16.          | 593            | 25             | 6         | 4         | 2         | 1         | 5         | -         | 4          | 3         |
| 17.          | 597            | 17             | 1         | 4         | 2         | -         | 2         | 1         | 5          | 2         |
| 18.          | 491            | 15             | 3         | 1         | -         | 2         | 1         | 1         | 5          | 2         |
| 19.          | 295            | 6              | -         | 1         | -         | 2         | -         | -         | 3          | -         |
| 20.          | 310            | 10             | -         | 2         | -         | 1         | 3         | -         | 2          | 2         |
| 21.          | 317            | 11             | -         | 2         | 2         | 1         | 3         | -         | 3          | -         |
| 22.          | 269            | 3              | -         | -         | -         | 1         | 1         | -         | -          | 1         |
| 23.          | 336            | 9              | -         | 5         | 1         | -         | -         | -         | 3          | -         |
| 24.          | 703            | 17             | 2         | 6         | -         | 1         | 5         | -         | 3          | -         |
| 25.          | 281            | 9              | -         | 4         | 1         | 1         | 1         | -         | 2          | -         |
| 26.          | 168            | 4              | 1         | -         | -         | -         | 1         | 1         | 1          | -         |
| 27.          | 235            | 5              | -         | 2         | -         | 1         | 1         | -         | 1          | -         |
| 28.          | 362            | 12             | 2         | 2         | 1         | 2         | 1         | -         | 3          | 1         |
| 29.          | 504            | 7              | 1         | 1         | 1         | 1         | -         | -         | 3          | -         |
| 30.          | 382            | 9              | 1         | 1         | 1         | -         | 2         | -         | 4          | -         |
| 31.          | 347            | 7              | -         | 2         | -         | 2         | 2         | -         | 1          | -         |
| 32.          | 491            | 9              | 1         | 1         | -         | -         | 3         | 1         | 2          | 1         |
| 33.          | 323            | 7              | -         | 5         | -         | -         | 1         | -         | 1          | -         |
| 34.          | 715            | 26             | 5         | 6         | 1         | 1         | 5         | 2         | 6          | -         |
| 35.          | 600            | 11             | 1         | 3         | -         | 1         | -         | 1         | 4          | 1         |
| 36.          | 385            | 5              | -         | 1         | 1         | 3         | -         | -         | -          | -         |
| 37.          | 895            | 24             | 1         | 3         | 3         | 1         | 5         | -         | 9          | 2         |
| <b>Total</b> | <b>16225</b>   | <b>428</b>     | <b>50</b> | <b>94</b> | <b>23</b> | <b>29</b> | <b>93</b> | <b>11</b> | <b>105</b> | <b>23</b> |

Area of Residence unknown for 5236 births.

Total taken from Report of Medical Officer of Health, Glasgow, 1970.

Twin deaths taken as single births for area of residence.

