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J. J. MURPHY, M.Ch., F.R.C.S.I.

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DANIEL G. KELLY, M.Ch., F.R.C.S.I.

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Ophthalmologist:

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MISSM. MORAN

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MRS. P. McCOMBE

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MISS S. KAVANAGH, M.C.S.P.
Glossary of Terms

BOOKED PATIENT: A patient who is seen once at the antenatal clinic, other than the occasion on which she is admitted. This includes patients seen by the Consultant staff in their Consulting rooms.

ABORTION: Expulsion of products of conception before the end of the 28th week of pregnancy.

MATERNAL MORTALITY: Death of a patient, booked or unbooked, for whom the hospital has accepted responsibility, during pregnancy or within six weeks of delivery whether in the hospital or not.

Maternal mortality is calculated against the total number of mothers in the hospital including abortions, ectopic pregnancies and hydatidiform moles.

STILLBIRTH (S.B.): A baby born after the end of the 28th week of pregnancy who shows no sign of life.

FIRST WEEK NEONATAL DEATH (N.N.D.): Death of a baby born alive after the end of the 28th week of pregnancy, within 7 days, either in the hospital or after transfer to another hospital.

PERINATAL MORTALITY: The sum of stillbirths and first week neonatal deaths as defined.

CALCULATION OF PERINATAL MORTALITY RATE: The perinatal mortality rate refers to the number of perinatal deaths (S.B. and 1st week N.N.D.) per 1,000 total births at or over 28 weeks.

Abbreviations used throughout report:

A.N.C. Antenatal course
A.P.H. Antepartum haemorrhage
A.R.M. Artificial rupture of membranes
B./N.B. Booked/non-booked
B.P.D. Bi-parietal diameter
C.A.N.C. Combined antenatal care
C.P.A.P. Continuous positive airway pressure
C.T.G. Cardiotogography
C.V.P. Central venous pressure
D.I.C. Disseminated intravascular coagulation
F.H.H./N.H. Fetal heart heard/not heard
F.M.N.F. Fetal movement not felt
G.T.T. Glucose tolerance test
I.P.P.V. Intermittent positive pressure ventilation
I.U.D. Intrauterine death
L.S. Lecithin/sphingomyelin
L.S.C.S. Lower segment Caesarean Section
M.S.U. Mid-stream urinalysis
P.E.T. Pre-eclamptic toxaemia
P.G.E. Prostaglandin
P.P.H Post-partum haemorrhage
P.R.O.M. Premature rupture of membranes
R.D.S. Respiratory distress syndrome
R.I.U.G. Retarded intrauterine growth
S.C.B.U. Special Care Baby Unit
S.E. Socio-economic group
S.R.O.M. Spontaneous rupture of membranes
S.V.D. Spontaneous vertex delivery
U.T.I. Urinary tract infection
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### Guinness Lectures

<table>
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<th>Year</th>
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<td>University of Liverpool</td>
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<tr>
<td>1970</td>
<td>Professor N. Butler</td>
<td>&quot;British Perinatal Survey.&quot;</td>
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<td>1971</td>
<td>Sir Dugald Baird</td>
<td>&quot;How Many Children.&quot;</td>
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<td>University of Aberdeen.</td>
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<td>1972</td>
<td>Professor C. A. Janeway</td>
<td>&quot;The Immunological Relationship between Mother and Fetus.&quot;</td>
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<td></td>
<td>Boston.</td>
<td>&quot;Not Two but One.&quot;</td>
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<td>1973</td>
<td>Professor F. Geldenhuys</td>
<td>&quot;The Obstetrician-Gynaecologist and Diseases of the Breast.&quot;</td>
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<td>University of Pretoria.</td>
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<td>1978</td>
<td>Professor Keith P. Russell</td>
<td>&quot;Preterm Birth and the Developing Brain.&quot;</td>
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<td>University of Southern California School of Medicine.</td>
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<td>1979</td>
<td>Dr. J. S. Wigglesworth</td>
<td>&quot;The Obstetrician—a Biologist or a Sociologist.&quot;</td>
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<tr>
<td></td>
<td>Institute of Child Health</td>
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<td></td>
<td>University of London.</td>
<td></td>
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<tr>
<td>1980</td>
<td>Professor James Scott</td>
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<tr>
<td></td>
<td>University of Leeds.</td>
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</table>
**Introduction**

Seven thousand seven hundred and twelve babies of over 28 weeks gestation were born during 1980; this represented an increase of 0.9 per cent. There were 75 stillbirths and 33 neonatal deaths so that the perinatal mortality rate for the year was 14.0 per thousand. Congenital malformations accounted for 43 per cent of the total perinatal deaths and when such cases were excluded the stillbirth rate was 6.9 per thousand and the neonatal death rate was 1.2 per thousand giving a corrected perinatal mortality rate of 8.1 per thousand. The major improvement noted during 1980 was the large decrease in the number of normal neonatal deaths, so that during the year there were only 9 early neonatal deaths among the 7,613 normal liveborn infants of greater than 28 weeks maturity.

The comparative table summarising perinatal mortality rate for the past ten years clearly shows the tremendous improvement that has taken place in neonatal care with the neonatal death rate for normal infants dropping from 6.2 in 1971 to 1.2 in 1980. It is also noteworthy that since 1975 there has been no reduction in the normal stillbirth rate, and indeed the last few years has seen a slight increase. Clearly, the improvement in perinatal mortality rates in recent years has been entirely due to improved neonatal care. Indeed, it is disappointing to note that the obstetricians have been unable to reduce the normal stillbirth rate in the past six years. The number of intra-partum deaths each year is now extremely low and strenuous efforts must be made to identify the at risk fetus in utero. As mentioned in the chapter relating to stillbirths, avoidable factors of one form or another were noted in 18 of 48 normal antepartum deaths and in the 5 intra-partum deaths that occurred during 1980. It is essential that such factors be reduced if perinatal mortality is to improve.

Grants totalling £5,220,787 were received from the Department of Health. The cost per occupied bed was £61.00 per day. This can be compared with the figure of £43.00 per day for 1979, and represents an increase of 41.9 per cent.

The research programme was intensified during 1980. Studies being performed by Doctors Bernard Stuart and John Murphy, previously referred to in other reports, were concluded during the year. Arrangements were made subsequently to expand the study of blood flow into an assessment of cerebral blood flow in the neonate, together with an assessment of the incidence and severity of intraventricular haemorrhage both in normal and ill neonates. These latter studies were due to commence during 1981. A prospective study into the progress and outcome of breech delivery was also concluded during 1980, as was the two year follow-up study of these babies being conducted by Dr. Keane and Dr. O'Connell.

During the year, Dr. J. Clinch was conferred with a Fellowship of the Royal College of Obstetricians and Gynaecologists. Dr. D. O'Brien was appointed as Irish Representative to the Hospital Recognition Committee of the Royal College of Obstetricians and Gynaecologists, and Dr. Duignan continued as Irish member on the Council of that college.

The Guinness Lecture in 1980 was given by Professor James Scott. Professor Scott has been a long standing supporter of Irish obstetrics. His high moral standards have been a credit for all to see. We were thus
particularly pleased to have him give the Guinness Lecture and his excellent presentation attracted a large appreciative audience.

Two former Masters died during 1980. Dr. R. Corbet, who had been Master between 1936 and 1942 and Dr. J. J. Stuart who was Master from 1957 to 1963. Both of these men had devoted many many years of work to the Coombe Hospital. They are very sadly missed both by the hospital and their families. Two Assistant Masters resigned during the year to take up other appointments. Dr. John Murphy was appointed as Consultant Obstetrician/Gynaecologist to the National Maternity Hospital and we wish him well in his new post; we were very sorry to lose him. Dr. R. Gossa left Ireland to work in England. These Assistant Masters were replaced by Doctor P. Bowman and Doctor F. Lynch.

Several changes have taken place in the presentation of this year's report. A new ten-year table summarising perinatal mortality has been included, which clearly indicates that the drop in perinatal mortality has been entirely due to a decrease in the number of normal live-born infants dying in the neonatal period. Each perinatal death has been given a specific number and avoidable factors are discussed in the chapter on stillbirths and neonatal deaths. In subsequent chapters a full summary is given of each normal neonatal death, while only the case numbers of babies dying from congenital malformations have been listed. A new chapter entitled "Management of labour" has been included which summarises procedures performed in the labour ward together with the outcome of patients with malpresentation in labour. In addition, the gynaecological section of the report has been altered slightly so that this chapter now includes both the routine gynaecological work together with a summary of cases seen both in the Colposcopy and Infertility Clinics. It is thus possible to get an easier breakdown of the gynaecological work load.

I am grateful for the help given to me by the Assistant Masters in compiling this report. In addition I would like to thank the following who helped to prepare special sections: J. Clinch (Induction of Labour), J. Drumm (Ultrasound and Haemolytic Disease), M.I. Drury (Diabetic Clinic), A. T. Greene (Infertility and Diabetic Clinic), E. Griffin (Paediatric Department and Haemolytic Disease), M. Hooper (Varicose Vein Clinic), F. Martin (Pathology Department), R. Mulcahy (Cardiac Department), P. O'Connell (Paediatric Assessment Unit), Miss Gregg (Social Services Department), Miss O'Loughlin (Physiotherapy Department) and Mrs. Rhatigan (Radiology Department).

I would like to take the opportunity also of thanking Dr. J. O'Riordan and staff of the Blood Transfusion Service Board for the continued first class service they gave us during 1980.

I would like to conclude by expressing my personal thanks to all members of the hospital staff, both medical and non-medical, who have continued to maintain a high standard of care; I would also like to thank the Chairman and Board members who devoted so much of their spare time to the running of the hospital. Finally I would like to thank my secretary Miss Corrigan and all the staff in the medical records department who were so meticulous in collecting the data for this report. Hopefully it will soon be possible to get a computer which will facilitate the easier collection of more detailed data.

NIALL DUIGNAN.
## Comparative Table for 10 Years

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</thead>
<tbody>
<tr>
<td>Babies born</td>
<td>6,726</td>
<td>7,856</td>
<td>7,631</td>
<td>7,721</td>
<td>7,301</td>
<td>7,111</td>
<td>7,099</td>
<td>7,516</td>
<td>7,642</td>
<td>7,712</td>
</tr>
<tr>
<td>Perinatal deaths</td>
<td>174</td>
<td>181</td>
<td>161</td>
<td>179</td>
<td>132</td>
<td>117</td>
<td>113</td>
<td>105</td>
<td>117</td>
<td>108</td>
</tr>
<tr>
<td>Mortality rate</td>
<td>25.8</td>
<td>23.0</td>
<td>21.4</td>
<td>23.2</td>
<td>18.1</td>
<td>16.5</td>
<td>15.9</td>
<td>13.9</td>
<td>15.3</td>
<td>14.0</td>
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<tr>
<td>Mothers delivered</td>
<td>7,423</td>
<td>8,607</td>
<td>8,428</td>
<td>8,494</td>
<td>7,970</td>
<td>7,809</td>
<td>7,781</td>
<td>8,267</td>
<td>8,385</td>
<td>8,336</td>
</tr>
<tr>
<td>Maternal deaths</td>
<td>1</td>
<td>11</td>
<td>1</td>
<td>Nil</td>
<td>Nil</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Caesarean section %</td>
<td>5.5</td>
<td>4.6</td>
<td>6.3</td>
<td>6.7</td>
<td>6.5</td>
<td>7.9</td>
<td>7.9</td>
<td>7.3</td>
<td>7.6</td>
<td>7.8</td>
</tr>
<tr>
<td>Forceps %</td>
<td>13.3</td>
<td>12.8</td>
<td>13.4</td>
<td>14.1</td>
<td>12.6</td>
<td>9.9</td>
<td>8.7</td>
<td>7.8</td>
<td>7.3</td>
<td>7.3</td>
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<tr>
<td>Induction %</td>
<td>16.2</td>
<td>30.0</td>
<td>40.6</td>
<td>44.0</td>
<td>36.0</td>
<td>28.6</td>
<td>29.8</td>
<td>24.1</td>
<td>23.4</td>
<td>21</td>
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</table>
Statistical Summary

Mothers delivered after 28 weeks gestation ... 7,596
Mothers delivered before 28 weeks gestation ... 731
Hydatidiform Moles . . . . . . . . . . . . . . . . . . . . . . . . . . 2
Ectopic Pregnancies . . . . . . . . . . . . . . . . . . . . . . . . . . 7

Mothers delivered after 28 weeks gestation
  Primigravidae . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 2,215
  Multiparae . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 5,381
  Booked cases . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 7,504
  Non-booked cases . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 92

Maternal Deaths

Infants delivered over 28 weeks gestation 7,712
  Sets of twins . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 115
  Set of triplets . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . . 1

Perinatal Deaths

  Booked cases 98
  Non-booked cases 10
Statistical Analysis of Hospital Population

Age-Group

<table>
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<tr>
<th>Age-Group</th>
<th>Total Births</th>
<th>% Hospital Population</th>
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<tbody>
<tr>
<td>&lt; 20 years</td>
<td>399</td>
<td>5.2</td>
</tr>
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<td>20-24 years</td>
<td>1,760</td>
<td>23.1</td>
</tr>
<tr>
<td>25-29 years</td>
<td>2,541</td>
<td>33.5</td>
</tr>
<tr>
<td>30-34 years</td>
<td>1,950</td>
<td>25.7</td>
</tr>
<tr>
<td>35-39 years</td>
<td>781</td>
<td>10.3</td>
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<tr>
<td>40+ years</td>
<td>165</td>
<td>2.2</td>
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Parity

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<th>Parity</th>
<th>Total Births</th>
<th>% Hospital Population</th>
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<tbody>
<tr>
<td>0</td>
<td>2,215</td>
<td>29.2</td>
</tr>
<tr>
<td>1,2,3</td>
<td>4,538</td>
<td>59.7</td>
</tr>
<tr>
<td>4+</td>
<td>843</td>
<td>11.1</td>
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Birth Weight

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<tr>
<th>Birth Weight</th>
<th>Total Births</th>
<th>% Hospital Population</th>
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<tbody>
<tr>
<td>&lt; 1,000 g.</td>
<td>12</td>
<td>0.2</td>
</tr>
<tr>
<td>1,001-1,500 g.</td>
<td>43</td>
<td>0.6</td>
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<td>1,501-2,000 g.</td>
<td>90</td>
<td>1.1</td>
</tr>
<tr>
<td>2,001-2,500 g.</td>
<td>275</td>
<td>3.6</td>
</tr>
<tr>
<td>2,501-3,000 g.</td>
<td>1,069</td>
<td>13.9</td>
</tr>
<tr>
<td>3,001-3,500 g.</td>
<td>2,740</td>
<td>35.5</td>
</tr>
<tr>
<td>3,501-4,000 g.</td>
<td>2,466</td>
<td>32.0</td>
</tr>
<tr>
<td>4,001-4,500 g.</td>
<td>874</td>
<td>11.3</td>
</tr>
<tr>
<td>4,501+ g.</td>
<td>142</td>
<td>1.8</td>
</tr>
<tr>
<td>Not weighed</td>
<td>1</td>
<td>0.1</td>
</tr>
</tbody>
</table>

Gestation

<table>
<thead>
<tr>
<th>Gestation</th>
<th>Total Births</th>
<th>% Hospital Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>28-31 weeks</td>
<td>38</td>
<td>0.5</td>
</tr>
<tr>
<td>32-36 weeks</td>
<td>281</td>
<td>3.6</td>
</tr>
<tr>
<td>37-41 weeks</td>
<td>6,959</td>
<td>90.3</td>
</tr>
<tr>
<td>42+ weeks</td>
<td>434</td>
<td>5.6</td>
</tr>
</tbody>
</table>

Comment: These tables refer only to mothers whose pregnancies progressed beyond 28 weeks gestation. The mothers' age group was similar to that recorded in 1979, though the number of women having their fifth or more child dropped from 15 to 11 per cent. There was a continued slight increase in the incidence of pre-term birth in relation to 1978 and 1979; during the year 420 infants (5.6 per cent) weighed less than 2.5 kilograms at birth. Three hundred and nineteen babies (4.1 per cent) were less than 37 weeks gestation. The number of patients whose pregnancies progressed beyond 42 weeks gestation increased from 4.8 to 5.6 per cent.
**Perinatal Mortality**

<table>
<thead>
<tr>
<th>Category</th>
<th>Number</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of infants born after 28 weeks</td>
<td>7,712</td>
<td></td>
</tr>
<tr>
<td>Perinatal Deaths</td>
<td>108</td>
<td>14.0</td>
</tr>
<tr>
<td>Booked Patients</td>
<td>7,504</td>
<td>13.0</td>
</tr>
<tr>
<td>Non-booked Patients</td>
<td>92</td>
<td>108</td>
</tr>
<tr>
<td>Stillbirths</td>
<td>75</td>
<td>9.7</td>
</tr>
<tr>
<td>Neonatal Deaths</td>
<td>33</td>
<td>4.3</td>
</tr>
</tbody>
</table>

Perinatal mortality calculated for infants 1,000 grams or more: 98, 12.7%

Perinatal mortality calculated for infants 500 grams or more: 124, 16.0%

**Analysis of Perinatal Mortality**

<table>
<thead>
<tr>
<th>Category</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antepartum Deaths</td>
<td>48</td>
</tr>
<tr>
<td>Intrapartum Deaths</td>
<td>5</td>
</tr>
<tr>
<td>Neonatal Deaths</td>
<td>9</td>
</tr>
<tr>
<td>Congenital Malformations</td>
<td>46</td>
</tr>
<tr>
<td></td>
<td>108</td>
</tr>
</tbody>
</table>
Statistical Analysis of Perinatal Deaths

### Age-Group

<table>
<thead>
<tr>
<th>Total Births</th>
<th>Perinatal Deaths</th>
<th>Perinatal Mortality Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 20 years</td>
<td>399</td>
<td>5</td>
</tr>
<tr>
<td>20-24 years</td>
<td>1,760</td>
<td>18</td>
</tr>
<tr>
<td>25-29 years</td>
<td>2,541</td>
<td>25</td>
</tr>
<tr>
<td>30-34 years</td>
<td>1,949</td>
<td>38</td>
</tr>
<tr>
<td>35-39 years</td>
<td>781</td>
<td>14</td>
</tr>
<tr>
<td>40+ years</td>
<td>165</td>
<td>8</td>
</tr>
</tbody>
</table>

### Parity

<table>
<thead>
<tr>
<th>Total Births</th>
<th>Perinatal Deaths</th>
<th>Perinatal Mortality Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>2,215</td>
<td>30</td>
</tr>
<tr>
<td>1,2,3,</td>
<td>4,537</td>
<td>52</td>
</tr>
<tr>
<td>4+</td>
<td>843</td>
<td>26</td>
</tr>
</tbody>
</table>

### Birth Weight

<table>
<thead>
<tr>
<th>Birth Weight</th>
<th>Total Births</th>
<th>Perinatal Deaths</th>
<th>Perinatal Mortality Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1,000 g</td>
<td>12</td>
<td>12</td>
<td>1,000</td>
</tr>
<tr>
<td>1.001-1,500 g</td>
<td>43</td>
<td>19</td>
<td>441</td>
</tr>
<tr>
<td>1.501-2,000 g</td>
<td>90</td>
<td>13</td>
<td>144</td>
</tr>
<tr>
<td>2.001-2.500 g</td>
<td>275</td>
<td>15</td>
<td>54</td>
</tr>
<tr>
<td>2.501-3,000 g</td>
<td>1,069</td>
<td>19</td>
<td>17</td>
</tr>
<tr>
<td>3.001-3,500 g</td>
<td>2,740</td>
<td>24</td>
<td>8.7</td>
</tr>
<tr>
<td>3.501-4,000 g</td>
<td>2,466</td>
<td>3</td>
<td>1.2</td>
</tr>
<tr>
<td>4.001-4.5(H) g</td>
<td>874</td>
<td>1</td>
<td>1.1</td>
</tr>
<tr>
<td>4,501+ g.</td>
<td>142</td>
<td>1</td>
<td>7.0</td>
</tr>
<tr>
<td>Not weighed</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

### Gestation

<table>
<thead>
<tr>
<th>Gestation</th>
<th>Total Births</th>
<th>Perinatal Deaths</th>
<th>Perinatal Mortality Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>28-31 weeks</td>
<td>38</td>
<td>19</td>
<td>500</td>
</tr>
<tr>
<td>32-36 weeks</td>
<td>281</td>
<td>30</td>
<td>106</td>
</tr>
<tr>
<td>37-41 weeks</td>
<td>8,959</td>
<td>42</td>
<td>6.0</td>
</tr>
<tr>
<td>42+ weeks</td>
<td>434</td>
<td>17</td>
<td>39</td>
</tr>
</tbody>
</table>

**Comment:** The above table which applies only to patients of greater than 28 weeks gestation, clearly showed affect which age, parity, birth weight and gestation exert on perinatal mortality rates. It is of interest that all 12 babies weighing less than 1,000 grams, born after 28 weeks gestation died, while 4 infants weighing less than 1,000 grams and under 28 weeks gestation survived.
### Analysis of Perinatal Mortality for 10 Years

<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Perinatal Deaths</td>
<td>174</td>
<td>181</td>
<td>161</td>
<td>179</td>
<td>132</td>
<td>117</td>
<td>113</td>
<td>105</td>
<td>117</td>
<td>108</td>
</tr>
<tr>
<td>Mortality Rate</td>
<td>25.8</td>
<td>23.0</td>
<td>21.4</td>
<td>23.2</td>
<td>18.1</td>
<td>16.5</td>
<td>15.9</td>
<td>13.9</td>
<td>15.3</td>
<td>14.0</td>
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<tr>
<td>Antepartum deaths</td>
<td>69</td>
<td>77</td>
<td>41</td>
<td>65</td>
<td>42</td>
<td>44</td>
<td>31</td>
<td>43</td>
<td>48</td>
<td>48</td>
</tr>
<tr>
<td>Percentage of Total</td>
<td>40</td>
<td>43</td>
<td>26</td>
<td>36</td>
<td>31</td>
<td>37</td>
<td>27</td>
<td>41</td>
<td>41</td>
<td>44</td>
</tr>
<tr>
<td>Intrapartum deaths</td>
<td>18</td>
<td>14</td>
<td>12</td>
<td>10</td>
<td>5</td>
<td>8</td>
<td>3</td>
<td>6</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Percentage of Total</td>
<td>10</td>
<td>8</td>
<td>7</td>
<td>6</td>
<td>5</td>
<td>7</td>
<td>3</td>
<td>6</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Neonatal deaths</td>
<td>41</td>
<td>37</td>
<td>44</td>
<td>43</td>
<td>23</td>
<td>29</td>
<td>18</td>
<td>19</td>
<td>19</td>
<td>9</td>
</tr>
<tr>
<td>Percentage of Total</td>
<td>24</td>
<td>20</td>
<td>27</td>
<td>24</td>
<td>17</td>
<td>25</td>
<td>16</td>
<td>18</td>
<td>16</td>
<td>8</td>
</tr>
<tr>
<td>Fetal Abnormalities</td>
<td>46</td>
<td>52</td>
<td>64</td>
<td>61</td>
<td>62</td>
<td>36</td>
<td>61</td>
<td>37</td>
<td>48</td>
<td>46</td>
</tr>
<tr>
<td>Percentage of total</td>
<td>26</td>
<td>29</td>
<td>40</td>
<td>34</td>
<td>47</td>
<td>31</td>
<td>54</td>
<td>35</td>
<td>41</td>
<td>43</td>
</tr>
</tbody>
</table>

**PERINATAL MORTALITY EXCLUDING CONGENITAL MALFORMATIONS**

<p>| | | | | | | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Normal Babies</td>
<td>6,680</td>
<td>7,003</td>
<td>7,567</td>
<td>7,660</td>
<td>7,239</td>
<td>7,075</td>
<td>7,038</td>
<td>7,479</td>
<td>7,594</td>
<td>7,666</td>
</tr>
<tr>
<td>Stillbirth Rate</td>
<td>13.0</td>
<td>11.6</td>
<td>7.0</td>
<td>9.8</td>
<td>6.5</td>
<td>7.3</td>
<td>4.8</td>
<td>6.5</td>
<td>6.5</td>
<td>6.9</td>
</tr>
<tr>
<td>Neonatal Death Rate</td>
<td>6.2</td>
<td>4.8</td>
<td>5.9</td>
<td>5.7</td>
<td>3.2</td>
<td>4.1</td>
<td>2.6</td>
<td>2.5</td>
<td>2.5</td>
<td>12.0</td>
</tr>
<tr>
<td>Perinatal Mortality Rate</td>
<td>19.2</td>
<td>16.4</td>
<td>12.9</td>
<td>15.5</td>
<td>9.7</td>
<td>11.4</td>
<td>7.4</td>
<td>9.0</td>
<td>9.0</td>
<td>8.1</td>
</tr>
</tbody>
</table>
Stillbirths

The changes in perinatal mortality over the past 10 years are summarised in the table. When congenital malformations are excluded it becomes apparent that the recent improvements in perinatal mortality are entirely due to improved neonatal care; indeed, the stillbirth rate for normal infants has increased since the low figure of 4.8 per thousand recorded in 1977.

Seventy-five of the 7,712 babies born after 28 weeks gestation in 1980 were stillborn, giving a stillbirth rate of 9.7 per thousand. Twenty-two stillbirths (29 per cent) were due to congenital malformation. Thus, there were 53 stillbirths among the 7,666 normal infants born after 28 weeks gestation; the stillbirth rate therefore for normal infants was 6.9 per thousand. Four hundred and nineteen of the 7,712 babies (5.4 per cent) weighed less than 2.5 kilograms at birth; 39 of these were stillborn and accounted for 52 per cent of the stillbirths.

The time at which intra-uterine death occurred is summarised in the following table:

<table>
<thead>
<tr>
<th>Cause</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dead on referral</td>
<td>3</td>
</tr>
<tr>
<td>Dead on admission to hospital</td>
<td>35</td>
</tr>
<tr>
<td>Died in antenatal ward</td>
<td>10</td>
</tr>
<tr>
<td>Died in labour</td>
<td>5</td>
</tr>
<tr>
<td>Congenital malformation</td>
<td>22</td>
</tr>
</tbody>
</table>

**ANTEPARTUM DEaths**

Forty-eight intra-uterine deaths occurred during the antenatal period and they accounted for 64 per cent of the stillbirths. Avoidable factors of one form or another were noted in 18 of the 48 normal antepartum deaths; these factors will be discussed in the individual cases. The main clinical factors giving rise to intra-uterine death are summarised in the following table:

<table>
<thead>
<tr>
<th>Cause</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cause unknown</td>
<td>16</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>13</td>
</tr>
<tr>
<td>Placental insufficiency</td>
<td>6</td>
</tr>
<tr>
<td>Hypertension/Pre-eclampsia</td>
<td>5</td>
</tr>
<tr>
<td>Multiple pregnancy</td>
<td>2</td>
</tr>
<tr>
<td>Diabetes</td>
<td>2</td>
</tr>
<tr>
<td>Cord accidents</td>
<td>2</td>
</tr>
<tr>
<td>Jaundice</td>
<td>2</td>
</tr>
<tr>
<td>Status asthmaticus</td>
<td>1</td>
</tr>
</tbody>
</table>
Cause Unknown (16)

No apparent cause for intra-uterine death could be found in 16 instances. All these babies were normal weight for gestation at the time of intra-uterine death. Four of the 16 deaths occurred before 37 weeks gestation and 12 afterwards. While no definite cause for the intra-uterine death could be found in any of the 16 cases, avoidable factors were present in 3 instances. Case 12 presented at 39 weeks gestation with a history of diminished fetal movement. A CTG showed deceleration and induction was arranged for the following morning. When the patient was admitted the fetal heart could not be heard. This patient should obviously have been admitted direct from the antenatal clinic. Case 14 gave a family history of diabetes but a glucose tolerance test was not performed. Case 16 was an unbooked 41 year old who was admitted after 42 weeks gestation with an intra-uterine death. Earlier delivery of this 41 year old patient would obviously have overcome this stillbirth.

Less than 37 weeks (4)

   Post-mortem: Maceration.

   Post-mortem: Maceration.

   Post-mortem: Early maceration.

   Post-mortem: Maceration.

Over 37 weeks (12)

   Post-mortem: Maceration.

   Post-mortem: Maceration.

   Post-mortem: Maceration.

   Post-mortem: Not performed.

*Post-mortem:* Intra-uterine anoxia.


*Post-mortem:* Maceration.


*Post-mortem:* Intra-uterine anoxia.


*Post-mortem:* Maceration.


*Post-mortem:* Intra-uterine anoxia.


*Post-mortem:* Maceration.

(15) B.49146. Para 0\textsuperscript{‘}. Age 29. S.E.I. Gestation 41 weeks. Irregular cycle. F.M. ceased at 41 weeks. Spontaneous labour and forceps delivery of macerated male infant weighing 3.06 kilograms.

*Post-mortem:* Maceration. Congenital heart.


*Post-mortem:* Maceration.

**Antepartum Haemorrhage (13)**

Thirteen stillbirths were directly due to accidental haemorrhage and this number represents a considerable increase on recent years. Avoidable factors were thought to be present in 5 of the 13 deaths. Case 17 was an in-patient for 4 weeks with persistent vaginal bleeding; although intra-uterine death occurred at 30 weeks gestation it could be argued that earlier delivery would have saved this infant. Case 18 was admitted at 31 weeks gestation with an antepartum haemorrhage; this baby was obviously grossly growth retarded in utero and earlier admission might have provided a more favourable outcome. Case 20 died as a result of abruptio placentae in association with severe P.E.T.; this patient should have been delivered earlier. Case 22 died at a direct result of an accidental haemorrhage following a difficult external cephalic version which was performed without general anaesthesia. Case 24 was admitted to a country hospital with an antepartum haemorrhage at 36 weeks gestation and transferred to the Coombe Hospital. However, intra-uterine death occurred during transfer
from the country hospital. It would have been wiser to have delivered this patient locally rather than to have transferred her to the Coombe.


Post-mortem: Not performed.


Post-mortem: Maceration.


Post-mortem: Maceration.


Post-mortem: Not performed.


Post-mortem: Refused.


Post-mortem: Maceration.


Post-mortem: Intra-uterine anoxia.


Post-mortem: Intra-uterine anoxia.


Post-mortem: Intra-uterine anoxia.


Post-mortem: Refused.

US

Post-mortem: Intra-uterine anoxia secondary to placental abruption.


Post-mortem: Intra-uterine anoxia.


Post-mortem: Intra-uterine anoxia.

**Placental Insufficiency (6)**

The term placental insufficiency is used to indicate any abnormality of placental function, either from clinical or post-mortem evidence. Six stillbirths were judged to have been due directly to placental insufficiency. Avoidable factors were present in 5 of these stillbirths: Cases 30 and 31 were noted to be small for dates at the antenatal clinic but were not admitted to hospital; Case 32 was an unbooked patient who had not attended anybody for antenatal care; Cases 34 and 35 should have been induced earlier and in both instances retarded intra-uterine growth was not picked up antenatally.


Post-mortem: Maceration.

(31) B. 44762. Para 1. Age 20. S.E.5. Gestation 34 weeks. Booked at 15 weeks gestation. Previous pre-term labour at 35 weeks, baby weighed 2.51 kilograms. Last seen at antenatal clinic at 33 weeks. Thought to be small. Scan arranged but admitted ten days later without any indication for antenatal care; Cases 34 and 35 should have been induced earlier and in both instances retarded intra-uterine growth was not picked up antenatally.

Post-mortem: Intra-uterine anoxia.


Post-mortem: Maceration.


Post-mortem: Refused.


Post-mortem: Maceration.


Post-mortem: Intra-uterine anoxia with autolysis of some organs.
Hypertension/Pre-Eclampsia (5)

Five stillbirths occurred as a direct result of pre-eclampsia or chronic hypertension; three of the deaths were associated with hypertension (37, 38, 40) while 2 deaths were associated with pre-eclampsia (36, 39). Avoidable factors were deemed to be present in 4 of these 5 cases: Case 37 attended the renal clinic with chronic hypertension but had no tests of placental wellbeing performed prior to intra-uterine death at 34 weeks; Case 38, who had a bad obstetric history, presented at approximately 24 weeks with uncertain dates and the fetal heart could not be heard at her next visit 4 weeks later; Case 39 presented under the Combined Antenatal Care Scheme but despite an elevated blood pressure between 32 and 36 weeks, she was not referred back to the hospital; Case 40 should also have been admitted to hospital earlier.


Post-mortem: Refused.


Post-mortem: Maceration.


Post-mortem: Refused.


Post-mortem: Maceration.


Post-mortem: Intra-uterine anoxia.

Multiple Pregnancy (2)

Two stillbirths occurred in association with multiple pregnancy. Both of these were macerated second twins in whom intra-uterine death had occurred many weeks prior to delivery, and both of these cases were deemed to be unavoidable.

weighing 3.45 kilograms born with Apgar scores of 9 and 10. Twin II macerated stillborn male infant weighing 1.15 kilograms.

Post-mortem: Maceration.


Post-mortem: Maceration.

**Diabetes (2)**

Two deaths occurred in association with clinical diabetes. One of these (43) was avoidable, while the other was deemed to be unavoidable. Case 43, a primigravida with no family history of diabetes was booked at 17 weeks gestation. Despite the presence of repeated glycosuria, no postprandial blood sugars were taken and the patient was not referred to the diabetic clinic prior to her admission in diabetic coma at 27 weeks. Intra-uterine death occurred at 28 weeks gestation as a direct result of a diabetic coma. Case 44 was a severe diabetic who was in hospital for many weeks prior to intra-uterine death. The baby was clinically grossly dysmature but was deemed to be too small to deliver prior to intra-uterine death at 30 weeks.

(43) B.55759. Para 0[^*]. Age 25. S.E.4. Gestation 28 weeks. First visit at 17 weeks gestation, —glycosuria + 3. Further visit 6 weeks later, —glycosuria + 2. Subsequent visits at 23 and 25 weeks showed no glycosuria but there was progressive weight loss. U.T.I. treated with Nitrofuradantin. Admitted at 27 weeks with vomiting. Within 24 hours developed ketoacidosis and coma. Insulin commenced and improvement was rapid. However the fetal heart disappeared on the morning of the 28th week of pregnancy. Spontaneous labour and delivery of stillborn female infant weighing 1.21 kilograms.

Clinical diagnosis: I.U.D. due to diabetes.

Post-mortem: Intra-uterine anoxia.

(44) B.1969. Para 3[^+2]. Age 30. S.E.3. Diabetic patient for 21 years. Gestation 30 weeks. Booked at 6 weeks gestation, which was confirmed by ultrasound scan. Insulin levels were controlled and regular attender. However, noted to have retarded intrauterine growth from 24 weeks gestation onwards. Grossly dysmature and I.U.D. at 30 weeks. At this time the uterine fundus was only equal to that of a 24 week pregnancy. Spontaneous labour and delivery one week later of female macerated infant weighing 850 grams.

Post-mortem: Refused.

**Cord accidents (2)**

Two antepartum deaths appeared to be directly due to cord accidents prior to the onset of labour and an avoidable factor was present in one instance. Case 45 had been attending an antenatal class in the hospital for 2 hours prior to informing the staff that she had not felt much fetal movement in the preceding two days. On immediate examination the fetal heart rate was heard at only 50/min. and it stopped five minutes later. Obviously this unfortunate case might have been avoided if the patient had informed the staff 2 hours earlier.

(45) B.47117. Para 0[^10]. Age 27. S.E.1. Gestation 36 weeks. Previous infertility and investigations found to be normal. Spontaneous pregnancy. Booked at 6 weeks gestation. Normal A.N.C. until admitted to hospital with history of no fetal movements for 2 days. F.H.H. only 50/min. on admission and stopped a few minutes later. PGE induction and delivery of stillborn female infant weighing 3.4 kilograms showing early maceration. Noted to have a true knot in cord and also cord tightly around neck.

Post-mortem: Maceration.

Post-mortem: Maceration.

Jaundice (1)

One stillbirth occurred in association with severe jaundice. This patient was transferred from a country hospital at 36 weeks gestation with deep jaundice and a coagulation defect. Intra-uterine death occurred 2 days following admission to hospital and the patient improved remarkably following delivery. On the basis of a subsequent liver biopsy, the jaundice was thought to have been due to an acute fatty degeneration.

(47) N.B.53432. Para 0. Age 20. S.E.5. Gestation 37 weeks. Transferred from country hospital at 37 weeks gestation with jaundice of one week's duration. F.H.H. on admission. Patient was jaundiced with high bilirubin levels, very low platelet levels and moderately raised transaminases. A provisional diagnosis of either leptospirosis, obstructive jaundice or viral hepatitis was made. Two days following admission, patient went into spontaneous labour. F.H.N.H. Prophylactic forceps delivery of a stillborn female infant weighing 2.65 kilograms. Primary post-partum haemorrhage treated with platelets and blood. Patient improved following delivery. Three days later patient transferred to general hospital. There liver biopsy performed and diagnosis of acute fatty liver made.

Post-mortem: Maceration.

Status Asthmaticus (1)

One intra-uterine death occurred as a direct complication of status asthmaticus. The mother's condition was too serious to affect delivery prior to intra-uterine death. This case has been deemed to be unavoidable.


Post-mortem: Maceration.

Congenital Malformations

Twenty-two of the 75 stillbirths (29 per cent) were due to congenital malformations. Twenty-one of these abnormalities arose in the central nervous system, while one baby died of multiple abnormalities.

Central Nervous System (21)


Post-mortem: (i) Anencephalus with Spina Bifida, (ii) Exomphalus. (iii) Maceration.
Post-mortem: Not performed.

Post-mortem: Refused.

Post-mortem: Anencephalus.

Post-mortem: Anencephaly.

Post-mortem: Refused.

Post-mortem: Refused.

Post-mortem: Anencephalus.

Post-mortem: Anencephalic—macerated.

Post-mortem: Refused.

Post-mortem: Microcephalus and Spina Bifida.

Post-mortem: (i) Anencephalus and Spina Bifida, (ii) Maceration.

Post-mortem: Anencephalus.

Post-mortem: (i) Anencephalus (ii) Potter’s Syndrome (iii) Maceration.


Post-mortem: (i) Anencephalus. (ii) Maceration.


Post-mortem: Not performed.


Post-mortem: (i) Anencephalus. (ii) Maceration.


Post-mortem: (i) Hydrocephalus and Spina Bifida, (ii) Horse-shoe kidney.


Post-mortem: Hydrocephaly.


Post-mortem: Not performed.


Post-mortem: (i) Hydrocephalus, (ii) Congenital Cardiac lesion—V.S.D.

Multiple (1)


Post-mortem: (i) Exomphalus. (ii) Congenital Cardiac lesion—V.S.D.

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**INTRAPARTUM DEATHS**

*Comparative Table for 10 Years*

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24
Five intra-partum deaths occurred during 1980 and avoidable factors were present in every case. Interestingly, 4 of the 5 deaths appeared to be due directly to cord accidents. Case 71 was admitted to hospital at 39 weeks gestation with repeated shows; she was thought not to have been in labour and was transferred to an antenatal ward for some hours prior to re-admission to the delivery suite with contractions and vaginal bleeding; fetal bradycardia was evident and an immediate Caesarean section performed, with delivery of a fresh stillborn infant; the I.U.D. appeared to have been due to strangulation of the neck by a nuchal cord. Case 72 was being monitored in labour and a bad recording may have been responsible for the absence of signs of fetal distress. Case 73 might have been avoided had A.R.M. been performed earlier. In Case 74 the fetal heart rate was being monitored continuously and a Type II deceleration was not noted. Case 75 was directly attributed to a cord prolapse, the cervix was 8 cms. dilated. This patient was labouring in the antenatal ward and earlier transfer to the delivery suite may have prevented this accident. In addition to the 5 stillbirths, 3 neonatal deaths (76-78) were directly due to problems which arose during labour.


Post-mortem:
(i) Strangulation of the neck by a nuchal cord.
(ii) Visceral congestion and oedema with pulmonary haemorrhage.
(iii) Normal placenta.


Post-mortem: Refused.


Post-mortem: Intra-uterine anoxia.


Post-mortem: Intra-partum asphyxia.

(75) B.6131. Para 5\textsuperscript{+0} Age 33. S.E.5. Gestation 41 weeks. Booked at 15 weeks. Admitted at 37 weeks with P.E.T. Head too high for A.R.M. Spontaneous onset of labour at 41 weeks—prolapce of cord at 8 cms. Spontaneous delivery of fresh stillborn male infant weighing 2.70 kilograms.

Post-mortem: Intra-partum asphyxia (cord prolapse).
NEONATAL DEATHS

Thirty-three of the 7,637 live born infants died in the first seven days of life, so that the neonatal death rate for 1980 was 4.3 per thousand. Twenty-four of the 33 deaths (73 per cent) were due to lethal congenital malformation. Thus, there were only 9 early neonatal deaths among the 7,613 normal live born infants of greater than 28 weeks maturity so that the early neonatal death rate among normal live born infants during 1980 was only 1.18 per thousand. This represents a significant reduction on neonatal death rates previously recorded and it reflects the improved standard of neonatal care currently available in the hospital. Six of the 9 normal neonatal deaths weighed less than 2.5 kilograms and only 3 mature normal infants died. The main causes of death were as follows:

Cerebral Anoxia ........................................ 3
Intraventricular Cerebral Haemorrhage .......... 2
Respiratory Dysfunction ............................. 2
Haemolytic Disease ................................ 1
Necrotising Enterocolitis .......................... 1
Congenital Malformations ......................... 24

Cerebral Anoxia (3)

Three neonatal deaths were directly attributable to intra-uterine anoxia which arose during labour and these cases should be considered in conjunction with the 5 intra-partum deaths previously referred to (71-75). Avoidable factors were present in 2 cases (76 and 78) and it could be argued that Case 77 was also avoidable. Case 76 was noted to have retarded intra-uterine growth at 37 weeks, which was confirmed by ultrasound scan. However, the patient was not admitted to hospital and subsequently presented herself in labour one week later with acute fetal distress. Admission to hospital at 37 weeks gestation would probably have avoided this loss. Case 78 should have been delivered by Elective Caesarean section. In Case 77 labour was induced at 41 weeks gestation. The intra-uterine anoxia appeared to be due to a ruptured vasa praevia and it could be argued that if labour had not been induced this accident might not have occurred.

(76) B.43120. Para 21. S.E.5. Gestation 38 weeks. Booked at 23 weeks. R.I.U.G. noted at 37 weeks and confirmed by scan. Booked for further scan but was admitted in labour at 38 weeks with slow irregular fetal heart and meconium stained liquor. L.S.C.S. performed. Female infant weighing 2.72 kilograms, flat at delivery. At delivery the baby had no apex and cardiac massage was given followed by intubation. Intermittent positive pressure was used and an apex beat was obtained. On admission to the S.C.B.U. the baby had occasional spontaneous respirations and required reintubation. Initial pH was 6.86 at 30 minutes of age. Anti cerebral oedema therapy was initiated and the baby remained critically ill. Further resuscitation was not undertaken. The baby died on the 2nd day.

Post-mortem: Non specific, and could be attributed to intra-uterine asphyxia.

(77) B.45670. Para 1. Age 23. S.E.3. Gestation 41 weeks. Normal antenatal record. Labour induced at 41 weeks. Some 2 hours later during strong contractions brisk vaginal haemorrhage with marked fetal bradycardia. Emergency L.S.C.S. Baby very flat, appeared exsanguinated, cord flacid. Presumptive diagnosis of ruptured vasa praevia. Female alive weighing 3.45 kilograms born with Apgar scores 1 and 0. While blood was being drawn from an O Neg. donor for immediate transfusion following delivery, the infant had a cardiac arrest. Within 4 hours of delivery the infant was transfused with 200ml. of fresh blood. Following this and during the latter part of the transfusion the baby was noted to be oozing from puncture sites. This tendency was counteracted with protamine sulphate pending coagulation screen result. By 14 hours of age marked cerebral signs were noted and the baby remained anuric. Subsequently continued oozing and drop in haemoglobin led to exchange transfusion which was well tolerated with little improvement in oozing from puncture sites. The baby's cerebral status deteriorated with seizures and at the age of 48 hours the coagulopathy was almost
corrected, and urinary output also improved. The baby continued to require full ventilation and at the age of 35 days had a cardiac arrest and was not resuscitated further.

*Post-mortem:* (i) Severe anoxic injury with cerebral haemorrhage.
(ii) Pulmonary haemorrhage and oedema.

(78) B.41239. Para 1st Age 30. S.E.2. Gestation 42 weeks. Booked at 8 weeks gestation. Uneventful A.N.C. except for anaemia. Admitted at 38 weeks gestation because of oblique breech. By 42 weeks cephalic presentation stable but high. Clinically big baby. PGE induction. Head did not descend in pelvis in second stage of labour and L.S.C.S. performed. Male infant weighing 4.71 kilograms, born with Apgar scores of 5 and 4. Baby was admitted to S.C.B.U. and following resuscitation the endotracheal tube was removed at 25 mins. Initial pH showed severe mixed metabolic respiratory acidosis, with a pH of 6.8. Following extubation the baby had poor respiratory effort with extreme hypotonia and staring associated with a fine tremor of face and feet. Treatment with Mannitol and Dexamethasone was commenced and the baby was slow to pass urine. At 24 hours of age the baby was noted to be having increasing respiratory distress associated with pneumonia. The baby was further supported with ventilation with some improvement. Neurological examination continued to be very abnormal. At 46 hours of age the baby was markedly hypoxic and despite every effort to improve ventilation the baby died.

*Post-mortem:* (i) Severe anoxic changes in the brain.
(ii) Bilateral bronchial pneumonia.
(hi) Pneumococcus was grown from the endotracheal tube and eye swab.

**Intraventricular Cerebral Haemorrhage (2)**

Two deaths in premature infants were due to massive intraventricular cerebral haemorrhage. A third pre-term infant (83) was also found to have an intraventricular haemorrhage, but the main factor associated with fetal demise in this patient was haemolytic disease and the case will be described later. Case 79 was a patient with a multiple pregnancy who developed acute hydramnios at 28 weeks gestation; transabdominal amniotomy was followed by premature labour and the first twin died of a large intraventricular cerebral haemorrhage. While it might be argued that administration of Dexamethasone to the mother prior to the performance of amniotomy might have improved the outcome for the second twin (82) which died of hyaline membrane disease, it is doubtful if it would have been of any benefit to the first twin. Case 80 presented in premature labour with a transverse lie and ruptured membranes at 29 weeks gestation. Although the baby was born in good condition with Apgar scores of 8 and 9, the baby died at 31 days of age as a result of widespread haemorrhages and this case has been deemed unavoidable.

(79) B.19241. Para 3rd Age 34. S.E.2. Gestation 28 weeks. Booked at 13 weeks gestation. Multiple pregnancy diagnosed on scan. Acute hydramnios at 28 weeks and admitted. Transabdominal amniotomy but labour occurred. Forceps delivery of live male infants. Twin 1, birth weight 1.25 kilograms, Apgar scores 0 and 3. The infant was intubated on delivery and given I.P.P.V. with slow improvement. Blood gases revealed severe mixed respiratory metabolic acidosis with adequate oxygenation. The baby's fontanelle was rather full and sutures splayed and C.S.F. was uniformly bloodstained. The baby died at 10 hours of age.

*Post-mortem:* Large intraventricular haemorrhage extending through the mid brain, cerebellum and into sub-arachnoid space. Total atelectasis of both lungs.

(80) B.25055. Para 2nd Age 34. S.E.3. Gestation 29 weeks. Booked at 10 weeks gestation. Regular attender at the antenatal clinic. Admitted at 29 weeks in labour with transverse lie. L.S.C.S. Male infant weighing 1.55 kilograms born with Apgar scores of 8 and 9. Following admission to S.C.B.U. the baby was intubated and given C.P.A.P. Clinical kyphoscoliosis was confirmed with X-ray. Full septic work-up was carried out in view of P.R.O.M. Chest X-ray was consistent with mild R.D.S. Antibiotics were commenced. Over the following 24 hours the baby became oedematous and at the age of 24 hours the infant collapsed. This was in association with severe metabolic acidosis with subsequent attacks of severe apnoea. The oedema increased and a pericardial effusion was aspirated. The baby had subsequent cardiac arrests and died at 3i days of age. Lumbar puncture performed at this time was bloodstained.
All cultures were sterile apart from an eye swab which grew actinitobacter species which was sensitive to the antibiotic used.

Post-mortem: (i) Massive intraventricular haemorrhage.
(ii) Haemopericardium.
(iii) Pulmonary and renal haemorrhages.

Respiratory Dysfunction (2)

The number of neonatal deaths as a result of respiratory dysfunction fell yet again during 1980. The type of respiratory dysfunction was classified as follows:

- Hyaline membrane disease
- Hyaline membrane disease and pulmonary atelectasis
- Hyaline membrane disease and cerebral haemorrhage
- Pulmonary atelectasis alone

The two babies that died as a result of respiratory dysfunction were born at 28 and 29 weeks respectively. Case 81 was a patient with multiple pregnancy who developed acute hydramnios at 28 weeks gestation. Premature labour followed transabdominal amniotomy and the second twin developed early respiratory dysfunction. This death might have been prevented had the mother been given Dexamethasone prior to the performance of transabdominal amniotomy. Case 82 was admitted in premature labour at 29 weeks gestation and delivered 1½ hours following admission to hospital. There was no obvious cause for the premature labour. Severe birth asphyxia with subsequent gross metabolic acidosis must have contributed to the failure to adequately resuscitate this pre-term infant.

(81) B.19241. Para 3. Age 34. S.E.2. Gestation 28 weeks. Booked at 13 weeks gestation. Multiple pregnancy diagnosed on scan. Acute hydramnios at 28 weeks and admitted. Transabdominal amniotomy but labour occurred. Forceps delivery of live male infants. Twin II, birth weight 961 grams. Clinically was severely undernourished with weight lying on the 10th centile. The baby responded well to resuscitation having been flat on delivery and was ventilated. At 4 hours of age the baby's colour disimproved and the ductus was noted to have opened. The baby shortly thereafter developed bilateral pneumothoraces which were drained with little improvement in the baby's condition which was associated with marked bradycardia. N.N.D. 24 hours.

Post-mortem: Bilateral pulmonary atelectasis with early changes of H.M.D. The gastrointestinal tract was almost devoid of meconium.

(82) N.B.55896. Para 2. Age 33. S.E.3. Gestation 29 weeks. No significant past history. Transferred from a country hospital in premature labour at 29 weeks. S.R.O.M. 2 hours earlier, clear liquor. After a labour lasting 1½ hours spontaneous delivery of live female infant weighing 1.19 kilograms, born with Apgar scores of 1 and 7. Infant transferred to S.C.B.U. and commenced on nasopharyngeal C.P.A.P. and shortly thereafter a nasotracheal tube was passed and full ventilation commenced. With this there was some improvement but the peripheries remained grey in colour. An arterial gas revealed a severe metabolic acidosis with pH = 7. Base Excess—20 while gases were normal. Further support was given with bicarbonate and fresh frozen plasma to no avail and the infant died 51 hours following delivery.

Post-mortem: (i) Hyaline membrane disease.
(ii) Subarachnoid haemorrhage.

Haemolytic Disease (1)

One baby died as a result of severe haemolytic disease. This patient's antenatal course was uneventful until she developed hydrops syndrome at 29 weeks gestation, in association with anti-Kell antibodies. Her previous pregnancies had been uneventful and neither of her previous infants were jaundiced. An Emergency Caesarean section was performed following an antepartum haemorrhage. Despite intensive treatment the baby died at 7
days of age. Although the anti-Kell antibodies were not discovered until 29 weeks gestation, this case is considered to be unavoidable because, even with the presence of anti-Kell antibodies, amniocentesis would not have been performed until about 28 weeks gestation in view of the negative past history.

(83) B. 18689. Para 2

Age 28. S.E.3. Gestation 30 weeks. Booked at 10 weeks gestation. Blood group A Rh. positive. Previous pregnancies (i) full term delivery followed by primary P.P.H. and blood transfusion, (ii) full term normal delivery, no complications. Present pregnancy uneventful until 29 weeks gestation when patient complained of oedema of legs, feeling tired and being uncomfortable. B.P. 140/100, no proteinuria, hydramnios. Scan showed huge placenta. Grade I-II praevia. Infant appeared hydropic with ascites. Mother found to have anti-Kell antibodies. A.P.H. and emergency L.S.C.S. with delivery of female infant weighing 2.2 kilograms, born with Apgar scores of 1 and 1. The baby was markedly hydropic and required intubation. Hydrops fetalis secondary to anti-Kell antibodies. Baby given 2 exchange transfusions and 3 top-up transfusions. Developed hypoglycaemia and hypercalcaemia which were treated with peritoneal dialysis. Baby failed to respond and died at 7 days of age. The mother was given 6 units of blood during L.S.C.S. and developed puerperal pyrexia due to bacteroides but made a good recovery.

Post-mortem:
(i) Massive intra cerebral haemorrhage.
(ii) Massive extra medullary haematopoeisis related to haemolytic disease.
(iii) Traumatic emphysema and subpleural bulla.

Necrotising Enterocolitis (I)

One baby born at 41 weeks gestation died as a result of necrotising enterocolitis. This pregnancy was a multiple pregnancy in whom two fetuses had been noted at ultrasound scan. In the event the mother had a triplet pregnancy, the third baby consisting of an acardiac monster. The pregnancy was allowed to proceed to 41 weeks gestation and the first triplet, a breech delivery weighing 2.31 kilograms, was dysmature. This neonatal death should be regarded as avoidable, as multiple pregnancies should be delivered at term or sooner.

(84) B. 10470. Para 3

Age 30. S.E.4. Gestation 41 weeks. Booked at 17 weeks gestation. Scan at 24 weeks because of gross hydramnios revealed multiple pregnancy. Two fetuses noted. Spontaneous onset of labour at 41 weeks with delivery of 3 infants. The first and second were normal females while the third was an acardic monster. Triplet I: assisted breech delivery female infant weighing 2.31 kilograms, born with Apgar scores of 3 and 9 Meconium aspirated from trachea at delivery and bronchial lavage was carried out. Small for dates. Mild cerebral irritability with depressed anterior fontanelle. P.C.V. 75%. Initial fluid restriction because of cerebral irritability which did not progress. On second day feeds were not tolerated and I.V. fluids were given. General condition less good. Full septic work-up performed and antibiotics commenced. Abdominal X-ray suggestive of necrotising enterocolitis with subsequent perforation. Following surgical consultation conservative management continued but later further deterioration and ventilatory support given. Urinary output dropped, with evidence of abdominal condition being less well localised. When baby stabilised she was transferred to another hospital for surgical intervention. However, operation was withheld and the abdomen drained. Subsequently, renal status deteriorated further, associated with cardiac arrhythmia secondary to hypercalcaemia which was corrected with peritoneal dialysis, using the drainage tube with restoration of urinary output. However, baby had sudden unexplained cardiac arrest on the 7th day.

Post-mortem:
(i) Peritonitis mainly localised to site of perforation.
(ii) Left renal papillary necrosis.
(iii) Necrotising enterocolitis.
(iv) Small left cerebral ventricular haemorrhage.

Congenital Malformations

Congenital malformations accounted for no less than 24 of the 33 early neonatal deaths. The site of the abnormality was as follows:
Central Nervous System ........................................ 6
Pulmonary—Renal Tract ........................................ 7
Multiple .......................................................... 4
Cardio-Vascular System ......................................... 3
Respiratory System .............................................. 2
Chromosome Abnormality ....................................... 2

Central Nervous System


\textit{Post-mortem:} Hydro-microcephalus.

(86) B.8871. Para 7\textsuperscript{+1} Age 42. S.E.2. Gestation 34 weeks. Booked at 12 weeks gestation when B.P. 160/100. Chronic hypertension. Admitted at 34 weeks in labour. One hour after admission delivered of an anencephalic male infant weighing 1.41 kilograms. N.N.D. 45 minutes.

\textit{Post-mortem:} Anencephalus.

(87) B.12206. Para 7\textsuperscript{+0} Age 38. S.E.3. Gestation 35 weeks. Booked at 16 weeks. Noted to have polyhydramnios at 33 weeks. Scan—anencephalic. Delivered by Caesarean section because of three previous L.S.C.S. Male infant weighing 1.47 kilograms. N.N.D. 5 minutes.

\textit{Post-mortem:} Not performed.

(88) N.B.55811. Para 2\textsuperscript{+0} Age 32. S.E.3. Gestation 38 weeks. Referred from country hospital at 38 weeks with a diagnosis of hydrocephaly and Spina Bifida. Assisted breech delivery following spontaneous labour 3 days later of female infant weighing 3.12 kilograms. N.N.D. minutes.

\textit{Post-mortem:} (i) Hydrocephalus.

(ii) Spina Bifida.


\textit{Post-mortem:} None performed.


\textit{Post-mortem:} Hydrocephalus and Spina Bifida.

Pulmonary—Renal Tract


\textit{Post-mortem:} (i) Pulmonary hypoplasia and immaturity.

(ii) Renal immaturity.

(92) B.54039. Para 0\textsuperscript{+1} Age 23. S.E.3. Gestation 34 weeks. First seen at 11 weeks gestation. Uneventful A.N.C. Admitted in premature labour at 34 weeks gestation. Assisted breech delivery of live female infant weighing 2.12 kilograms, born with Apgar scores of 1 and 2. N.N.D. minutes.

\textit{Post-mortem:} Potter's Syndrome.

Post-mortem: 
(i) Polycystic kidneys. 
(ii) Cystic dysplasia of liver. 
(iii) Pulmonary hypoplasia.


Post-mortem: Potter's Syndrome.

(95) B.20618. Para 4½ Age 33. S.E.5. Gestation 40 weeks. Booked at 18 weeks gestation. Combined Antenatal Care Scheme. Spontaneous delivery at term of male infant weighing 3.31 kilograms, born with Apgar scores of 7 and 9. N.N.D. hours.

Post-mortem: 
(i) Polycystic hydronephrotic kidneys. 
(ii) Pulmonary immaturity and hypoplasia.


Post-mortem: 
(i) Polycystic kidneys. 
(ii) Liver—cystic dysplasia of bile ducts. 
(iii) Pulmonary hypoplasia.

(97) B. 10615. Para5½ Age 33. S.E.3. Gestation 42 weeks. Previous hydrocephalus. Booked at 17 weeks under Combined Antenatal Care Scheme. Seen again at 28 weeks, noted to be small for dates. Ultrasound scan showed B.P.D. consistent with only 21 to 22 weeks maturity and reduced liquor. Subsequent scans showed no growth. No fetal urine in bladder. Spontaneous onset of labour at 42 weeks and breech delivery of live male infant weighing 2.27 kilograms, born with Apgar scores of 2 and 1. N.N.D. 4 hours.

Post-mortem: Refused.

Comment: The diagnosis of Potter's syndrome is presumptive and is based on the ultrasound reports.

Multiple


Post-mortem: Mermaid Fetus.


Post-mortem: 
(i) Exomphalos. 
(ii) Spina Bifida. 
(iii) Polycystic kidneys.

(100) B.53224. Para 0½ Age 19. Gestation 37 weeks. First seen at 33 weeks gestation when B.P. 140/95. Size 29 weeks. Admitted. Scan confirmed baby smallish, 31-32 weeks in size one week later. B.P. settled and discharged home after 5 days. Remainder of A.N.C. uneventful, although diastolic B.P. raised to 100 on occasions as outpatient. Admitted at 37 weeks with spontaneous onset of labour. A.R.M. to accelerate at 5 cms. Vertex engaged. One hour later
found to be fully dilated with loop of cord in vagina. Spontaneous delivery of live male infant weighing 2.5 kilograms. Baby had multiple large A.V. and capillary malformations consistent with Klippel-Trenary Weber Syndrome. N.N.D. 2 days.

Post-mortem:  
(i) Cardiac failure secondary to massive haemangiomata.  
(ii) Congenital anomalies.

(101) B. 10470. Para 3\textsuperscript{+1} Age 30. S.E.4. Gestation 41 weeks. Booked at 17 weeks gestation. Scan at 24 weeks because of gross hydramnios which revealed multiple pregnancy. Two fetuses noted. Spontaneous onset of labour at 41 weeks with delivery of 3 infants. The first and second were normal females, while the third was an acardiac monster. Triplet I: see Case 84, Triplet III: acardiac monster, N.N.D. 30 minutes.

Post-mortem: Acardiac Monster.

Cardio-Vascular System


Post-mortem:  
(i) Congenital cardiac anomaly—tricuspid atresia.  
(ii) Anasarca.


Post-mortem:  
(i) Congenital cardiac anomaly.  
(ii) Pulmonary hypoplasia and atelectasis.

(104) N.B. 42121. Para 12\textsuperscript{+2} Age 33. S.E.5. Gestation 38 weeks. No antenatal care. Arrived in spontaneous labour and delivered 2h hours later of alive male infant weighing 3.5 kilograms, born with Apgar scores of 9 and 10. Placenta healthy. N.N.D. 5 days.

Post-mortem:  
(i) Congenital cardiac anomaly. Single ventricle.  
(ii) Early necrotising enterocolitis.

Respiratory System

(105) B.46199. Para 1\textsuperscript{+0} Age 17. S.E.5. Gestation uncertain. Booked at 26 weeks. Scan showed ascites in fetal abdomen. Spontaneous onset of labour. Spontaneous delivery of a live female infant weighing 2.16 kilograms. Poor response to resuscitation and died within minutes.

Post-mortem:  
(i) Diaphragmatic hernia, left side.  
(ii) Congenital cystic right lung.  
(iii) Hypoplastic left heart with anomalous venous drainage.

(106) B.14841. Para 3\textsuperscript{+1} Age 33. S.E.4. Gestation 38 weeks. Gross hydramnios clinically at 33 weeks. Spontaneous onset of labour at 38 weeks. Controlled release of liquor at full dilatation. Small abruption. Female infant weighing 1.84 kilograms. Noted to be very mucosy with inability to pass a naso-gastric tube below the level of the cords. Intubated with difficulty. Physical examination revealed a very dysmature infant, who was cyanosed, mucosy and was felt to have a tracheo-oesophageal fistula clinically. Transferred to another hospital for further management. N.N.D. one week.

Post-mortem:  
(i) Tracheo-oesophageal Fistula.  
(ii) Oesophageal Atresia.

Chromosome Abnormality


Post-mortem: Edward's Syndrome.

\textit{Post-mortem:} (i) Multiple congenital abnormalities—pulmonary hypoplasia.  
(ii) Diaphragmatic hernia, oesophageal atresia.
Paediatric Department

During the year 1,094 of the 7,651 live-born infants (14.2 per cent) were admitted to the Special Care Unit for observation and treatment. In addition, a further 322 babies were admitted from outside the hospital. It is gratifying to record that the number of hospital born babies, who required admission to the Special Care Unit, dropped by a further 1 per cent during 1980.

The birthweight, gestation and mode of delivery for the babies requiring admission to the Special Care Unit are summarised in the following tables:

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<td>Vacuum</td>
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</tr>
<tr>
<td>Caesarean Section —Elective</td>
<td>80</td>
</tr>
<tr>
<td>—Emergency</td>
<td>152</td>
</tr>
</tbody>
</table>

Apart from babies who were admitted to the Special Care Unit, other infants required special management in the post-natal wards or careful follow-up in the out-patient clinic and the following table enumerates some of the more common conditions which were treated in the Special Care Unit, in postnatal wards, or in the out-patient department of the hospital. The numbers in the table do not necessarily correspond to the total number of infants treated, as some infants had more than one of the tabulated conditions.

<table>
<thead>
<tr>
<th>CONDITION</th>
<th>TOTAL</th>
<th>N.N.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prolonged Rupture of Membranes (greater than 24 hours)</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Birth Asphyxia</td>
<td>221</td>
<td></td>
</tr>
<tr>
<td>Cerebral Irritability (jittery)</td>
<td>167</td>
<td></td>
</tr>
<tr>
<td>Hypertonia</td>
<td>67</td>
<td></td>
</tr>
<tr>
<td>Hypotonia</td>
<td>38</td>
<td></td>
</tr>
<tr>
<td>Convulsions</td>
<td>24</td>
<td></td>
</tr>
<tr>
<td>Hyponatraemia</td>
<td>9</td>
<td></td>
</tr>
</tbody>
</table>
Outcome of Infants weighing 1.5 Kilograms or less

As the viability of pre-term infants continues to improve, the traditional requirement of the achievement of 28 weeks gestation in order to be considered viable needs to be reviewed. In view of this, included in this section of the report will be those infants who have survived who were less than 28 weeks gestation in addition to those who achieved 28 weeks. However, to make a comparison of data with previous years, two sets of figures will be presented: All infants of 1.5 kilograms or less, regardless of gestational age (a), and those infants of 28 weeks gestation and greater weighing 1.5 kilograms or less (b).

In 1980, seven of 14 infants born alive less than 28 weeks gestation survived and a brief outline of their course is given in a special section. It needs to be emphasised, however, that even greater care is required for this very premature group from the moment their mother enters the labour room. Paediatric care should be sought before delivery to ensure a state of readiness for the reception of this very high-risk infant who needs to be given every chance available, commencing with early and adequate resuscitation.

Seventy-three infants were delivered weighing 1,500 grammes or less (a) 55 were 28 weeks gestation or greater (b). 26 of the infants were stillborn (a), 22 of whom had reached 28 weeks gestation (b). 13 had severe congenital anomalies-case numbers 49 to 56, 60-64. There were 13 normal

<table>
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<tr>
<th>CONDITION</th>
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<th>N.N.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrolyte Disturbance-Transient</td>
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<td></td>
</tr>
<tr>
<td>Hypoglycaemia</td>
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<td>2</td>
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<tr>
<td>Hypocalcaemia</td>
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<td></td>
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<tr>
<td>Prematurity</td>
<td>252</td>
<td>11</td>
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<tr>
<td>Small for Gestational Age</td>
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<tr>
<td>Hyaline Membrane Disease (R.D.S.)</td>
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<td>Transient Tachypnoea of Newborn</td>
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<td>Pulmonary Atelectasis</td>
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<td>Pneumothorax</td>
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<td>Meconium Aspiration</td>
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<td>(seen beyond vocal cords)</td>
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<td>Aspiration of Liquor</td>
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<tr>
<td>Pneumonia</td>
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<td>Pulmonary Haemorrhage</td>
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<td>Hypothermia (Less than 35°C)</td>
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<td>Necrotizing Enterocolitis</td>
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<td>Jaundice-Physiological</td>
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<td>(greater than 200 mmol)</td>
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<td>Jaundice-Of Prematurity</td>
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<td>Jaundice-Breast Milk</td>
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<td>Jaundice-Treated</td>
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<td>Septic Spots</td>
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<td>Staphylococcal Infection</td>
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<td>Breast Abscess</td>
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<td>Abscess</td>
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<td>Conjunctivitis</td>
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<td>Bronchiolitis</td>
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<td>Urinary Tract Infection</td>
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<td>Meningitis</td>
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<td>U.R.T.I.</td>
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<td>Monilia (Perineal)</td>
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<td>Monilia (Oral)</td>
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<td>Inguinal Hernia</td>
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<tr>
<td>Pyloric Stenosis</td>
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<td></td>
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<td>Dermatitis</td>
<td>359</td>
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<tr>
<td>Feeding Problem</td>
<td>236</td>
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</table>

35
stillborn infants (a), nine of whom were 28 weeks gestation or greater—case numbers 1, 17-19, 36, 37 and 42-44 (b).

Of the 47 liveborn infants 16 (34%) died (a). There were 33 liveborn infants of 28 weeks gestation or greater (b) of whom nine (27%) died, six of severe congenital anomalies incompatible with survival (cases 86, 87, 91, 101,107 and 108). There were three early neonatal deaths in normal babies (cases 79, 81 and 82). These three babies represented one third of the early neonatal deaths in infants of 28 weeks gestation or greater who did not have major congenital anomalies.

Thus in 1980, of 41 normal infants delivered weighing less than or equal to 1,500 grammes, there were 10 neonatal deaths with 76% surviving (a); of those greater than or equal to 28 weeks gestation without major anomaly, there were three neonatal deaths with a survival rate of 89% (b).

The mean gestational age of the 31 infants weighing 1.5 kilograms or less who survived was 30.5 ± 3, (range 26-35 weeks) fa), and for the 24 who were 28 weeks and more 31.8 ± 2.2 weeks, (range 28-35 weeks) (b). The mean birth weight for the 31 infants was 1.28 ± 0.18 kilograms (range 0.735-1.5 kilograms) (a), and for those of 28 weeks and more 1.35 ± 0.11 kilograms (range 1.1-1.5 kilograms) (b). 17 mothers were documented as smokers of five or more cigarettes per day. There were seven non-smokers and there was no record for the remaining six mothers. There were 16 male and 15 female infants and one set of twins in the group.

Small for Gestational Age Infants

As in previous years, the centiles of Lubchenco et al have been used to determine whether a baby falls into this category.

There were seven babies in this group whose mean gestational age was 34.4 ± 0.97 weeks (range 33.6-36 weeks) and mean birth weight 1.37 ± 0.12 kilograms (range 1.13-1.47 kilograms). It is to be noted that none of these infants were less than 28 weeks gestation. The mean maternal age was 21.1 ± 5.5 years, (range 17-31 years). The mean parity was 1.14±03 (SD 0.9+08). Three mothers smoked five or more cigarettes per day and one did not smoke. Five of the seven were recognised antenatally to have retarded intrauterine growth. Four were delivered by elective caesarean section, one by breech extraction, one spontaneous vertex and one with forceps. One mother had an E.coli urinary tract infection. The mean Apgar scores at one and five minuters were 7 ± 2 and 9 ± 2.

Three mothers received dexamethasone prior to delivery. Six of the seven infants developed jaundice, four requiring phototherapy and two albumen. Respiratory distress was present in three; one of these had oesophageal atresia with severe respiratory distress syndrome complicated by interstitial emphysema and bi-lateral pneumothoraces; the other two had mild respiratory distress syndrome and did not require ventilatory support.

Two infants developed hypocalcaemia and one hypoglycaemia. One required total parenteral nutrition (oesophageal atresia) prior to transfer for gastrostomy at 18 days and one infant appeared to have had a reaction when Intralipid was commenced but tolerated vamin and dextrose until transpyloric feeds were established. Two infants required top-up transfusions.

Two received antibiotics, one for pneumonia and one for suspected sepsis. Only one infant had evidence of patent ductus arteriosus which closed spontaneously. None of the seven infants had evidence of congenital infection. In-patient stay was relatively uneventful apart from the infant who had oesophageal atresia and on whom definitive surgery was carried out at the age of 13 weeks.
Preterm Infants

Twenty-four of the 31 surviving infants were appropriate for gestational age. They include seven infants who were less than 28 weeks gestation. The mean gestational age for the group was 29.5 ± 2.5 (range 26-34 weeks) and the mean birth weight was 1.25 ± 0.19 kilograms (range 0.735-1.5 kilograms). The mean maternal age was 27.4 ± 6.2 years, (range 16-40 years). The mean parity was 2.04^9 (SD 2.1^11). There were 15 mothers documented as smokers of five or more cigarettes per day. In this group of infants, there was one set of twins and one second twin. The ante-natal course was complicated by antepartum haemorrhage in nine, two because of placenta praevia and one associated with vasa praevia. Two mothers had had Shirodkar sutures inserted and one mother had pre-eclampsia. Delivery was spontaneous in 11, by caesarean section in four, in one of whom it was performed as an emergency because of severe APH and in another for the delivery of a second twin who lay transversely in a mother who had had a previous caesarean section. Six deliveries were by breech, one of whom had a prolapsed cord, and two were with forceps. Seven mothers had either a partial or completed course of dexamethasone.

The mean Apgar scores for the infants at one and five minutes were 5 ± 3 and 8 ± 2 respectively.

Twenty infants had respiratory distress syndrome, 17 of whom required assisted ventilation and CPAP. Complications included pneumothorax in five, atelectasis in seven and in another infant who did not have hyaline membrane disease, pneumonia in five with aspiration occurring in two. Six infants developed broncho-pulmonary dysplasia, minor in three while being more severe in the other three. Three infants had pulmonary haemorrhage, one secondary to coagulopathy following exchange transfusion with heparinized blood which responded readily to protamine and in the other two this was associated with their respiratory condition. Theophylline was used in nine infants to wean from ventilation or CPAP and no infant became dependant on it. Those infants with broncho-pulmonary dysplasia, once weaned from CPAP, came off oxygen uneventfully. 11 infants were noted to have significant patent ductus arteriosus, two of whom were treated with indomethacin. None of these infants required surgical ligation of their ductus. 17 of the 31 infants required one or more top-up transfusions.

Fifteen infants had evidence of sepsis at some time during their course, three of whom had necrotizing enterocolitis. 18 infants received antibiotics either for sepsis or for chest complications at least once during their hospital stay. 23 infants of the group had phototherapy for jaundice while 13 of these also received albumen. Two infants required exchange transfusion, one of whom had ABO incompatability. Seizures occurred in five infants and in one case, they were associated with hepato-encephalopathy-this same infant on the second day of life had seizures and subsequently developed necrotizing enterocolitis followed by further seizures associated with hepato-encephalopathy. This infant has had a poor outcome developmentally associated with microcephaly. Evidence of intra-cranial haemorrhage was present in two infants, one of whom required a shunt. Despite early use of additional calcium supplements, hypocalcaemia developed in 16 infants. Hypoglycaemia was documented transiently in three, 17 infants required parenteral nutrition with Vamin and a significant number of these also received Intralipid.

Once again this group of low birth weight, pre-term infants have taken up a considerable proportion of the work in the unit, particularly those who developed complications such as recurrent atelectasis and broncho-pulmonary dysplasia. However, none of the infants who developed broncho-pulmonary dysplasia appeared to have significant sequelae related to their chest following discharge home.
Late Neonatal Deaths

In addition to the 33 early neonatal deaths, there were 12 late neonatal deaths. Eight of these were associated with severe congenital anomalies, of whom only one reached surgery. Details of these 12 infants are given below.

(109) Para 2\(^{+0}\). Age 27. Gestation 41 weeks. Birth weight 4.81 kilograms. Apgar scores were 3 and 6 at one and five minutes respectively. Following delivery by Caesarean Section for fetal distress, the baby was found to have Spina Bifida and Hydrocephalus. Baby died on 19th day.

(110) Para 4\(^{-1}\). Age 32. Gestation 36 weeks. Mother has small antepartum haemorrhage at 32 weeks and pre-eclampsia at 35 weeks. Following spontaneous rupture of membranes at 36 weeks, infant was the first of undiagnosed twins. Pale on delivery. Subsequent course uneventful until 66 hours of age, when found apnoeic with cardiac arrest. Resuscitated but required intermittent positive pressure ventilation, anti-cerebral oedema therapy and anti-convulsants. Baby died at 8 days. Autopsy revealed Large intraventricular haemorrhage. Right adrenal haemorrhage. Broncho pneumonia.

(111) Para 2\(^{+0}\). Age 29. Birth weight 2.30 kilograms. Apgar scores 9 and 10 at one and five minutes respectively. Baby was found to have Encephalocoele.

(112) Para 0\(^{-10}\). Age 24. Birth weight 2530 grams. Apgar scores 9 and 9 at one and five minutes respectively. Baby was admitted to the special care unit following delivery-small for dates and also a poor feeder. Baby died without resuscitation on the 11th day. Autopsy revealed Hydromicrocephalus with cerebellar hypoplasia.

(113) Para 3\(^{-10}\). Age 35. Birth weight 3.26 kilograms. Apgar scores were 8 and 9 at one and five minutes respectively. Baby was found to have multiple anomalies. Karyotype analysis confirmed Patau’s Syndrome. Autopsy revealed Hypoplastic left ventricle.

(114) Para 2\(^{-1}\). Age 28. Birth weight 2.835 kilograms. Apgar scores were 2 and 8 at one and five minutes respectively. Baby was delivered by caesarean section because of breech presentation. On delivery a lumbosacral myelomeningocele was found with no clinical hydrocephaly. Prior to transfer for surgery the infant had a seizure. Following surgery, the baby did not regain consciousness and required ventilatory support. Baby died on 13th day.

(115) Para 2\(^{-10}\). Age 34. Birth weight 3.74 kilograms. Apgar scores were 4, 7 and 9 at one, five and ten minutes respectively. Following delivery, multiple anomalies were noted with congestive cardiac failure on the 2nd day. Pending karyotyping report, baby was transferred for cardiological assessment. Numerous defects were found at cardiac catheterisation following which the baby went into clinical septic shock dying on the 9th day with extensive necrotising enterocolitis in addition to congenital cardiac defect. Karyotyping confirmed the clinical impression of chromosomal anomaly. Patau’s Syndrome.

(116) Para 5\(^{+1}\). Age 40. Gestation 40 weeks. Birth weight 3.38 kilograms. Apgar scores were 8 and 10 at one and five minutes respectively. Early neonatal course was uneventful. Infant admitted at the age of 6 days to another hospital with Staphylococcal septicaemia, which was associated with inappropriate dilution of feeds. However, there was no significant hypernatraemia at the time of admission. The baby died at the age of 8 days from Renal infarction and haemorrhage having made little response to therapy.

(117) Para 1\(^{-10}\). Age 23. Gestation 37 weeks. Birth weight 1.7 kilograms. Baby was a second twin and a breech presentation. The baby initially had mild respiratory distress syndrome and developed pneumothoraces. The baby also had minor congenital anomalies including an imperforate anus secondary to a membrane which was perforated on the first day of life, a hemivertebra deformity which were associated with two umbilical vessels. The baby developed evidence of a significant patent ductus arteriosus on the 4th day and went into congestive failure. Also around this time, the baby had symptoms suggestive of intraventricular haemorrhage which was supported by ultrasound examination. Because of this indomethacin was contra-indicated and the baby was treated with Lasix, Digoxin to no avail, and, on the 8th day, the baby was transferred to another hospital for ductus ligation. This was an uneventful procedure for the baby, and resulted in an improvement in the cardio ventricular status. On the following day, the baby developed evidence of a further intraventricular haemorrhage and remained ventilator dependant until death on the 7th post-operative day. This was confirmed at autopsy. Though this infant had several anomalies, none of them were significant enough to have contributed to this infants demise. The gestational age of the baby should have protected her against developing intraventricular haemorrhage, but from a pulmonary point of view she behaved in an extremely premature way.
Para 1. Age 24. Gestation 31 weeks. Birth weight 1.7 kilograms. Male infant, assisted breech delivery. Apgar scores 4, 6 and 7 at one, five and seven minutes respectively. On admission to the special care unit, the baby was noted to be pale and floppy and meconium had been aspirated from the trachea at delivery. I.P.P.V. was required for seven minutes and baby was then commenced on the ventilator. The diagnosis was meconium aspiration syndrome. The baby was difficult to ventilate and required paralysis. On the 11th day, the baby deteriorated suddenly and clinical findings were consistent with intraventricular haemorrhage. Ventilatory management of the baby continued to be difficult and the baby finally died on the 12th day of life. At Autopsy there was no haemorrhage within the ventricular system but there were marked hypoxic changes and the Pulmonary pathology was that of dysplasia following meconium aspiration and there was an associated renal infarction.

Para 3. Age 33. Gestation 41 weeks. Birth weight 2.21 kilos. Apgar scores five at one and eight at five minutes. No abnormality was observed antenatally until T+3 when the mother noted decreased foetal movement. C.T.G. was reactive at that time. Following delivery, it was apparent that the infant had a number of abnormalities, and clinically a diagnosis of Edwards Syndrome was made. This was confirmed on Karyotype analysis and the infant died at the age of 21 days. Autopsy refused.

Para 0. Age 26. Birth weight 2.77. Apgar scores nine and ten at one and five minutes respectively. Infant was discharged home on the sixth day following an uneventful early neonatal course. At the age of 19 days, the infant was brought back to the hospital because of loose stools, feeding poorly at the breast and a whining cry. The baby was admitted and extreme weight loss was noted, the weight having dropped to 2.04 kilos. There was gross disturbance of the central nervous system and in addition there was evidence of hypertonic dehydration associated with a metabolic acidosis. Blood and urine were collected for metabolic screen, which was positive for Maple Syrup Urine Disease. The infant regrettably deteriorated and required ventilatory support. However, within 12 hours of admission, she became markedly hypotonic and was unresponsive to all visible stimuli. E.E.G. was consistent with brain death and she died shortly following this examination. This infant had screening for inborn errors of metabolism on the 5th day of life and the initial report on this was normal. However, on further examination of that 5th day dried blood card and using a more specific biochemical method, there was evidence of the presence of the condition in the specimen. At autopsy, the brain was soft and necrotic with complete loss of architecture.

Congenital Malformations

<table>
<thead>
<tr>
<th>Number of Cases</th>
<th>Incidence per cent</th>
<th>Survived</th>
<th>Stillbirth</th>
<th>Early N.D.</th>
<th>Late N.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>741.0 Spina Bifida and Hydrocephalus</td>
<td>535</td>
<td>6.9</td>
<td>11</td>
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<tr>
<td>741 Spina Bifida</td>
<td>22</td>
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<td>6</td>
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<td>742.3 Congenital Hydrocephalus</td>
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<td>46</td>
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</table>

Comment: As in previous years, congenital anomalies accounted for a very significant number of neonatal deaths. Of the early neonatal deaths they accounted for 24 out of 33 in infants of 28 weeks and more gestational age. There has been an even greater number of congenital anomalies in those infants who died in the late neonatal period, only one of whom was considered amenable to surgery.
<table>
<thead>
<tr>
<th>Condition 1</th>
<th>Condition 2</th>
<th>Total</th>
<th>Survived</th>
<th>Stillbirth</th>
<th>Early N.N.D.</th>
<th>Late N.N.D</th>
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<tbody>
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<td>Anencephalus, Spina Bifida and Exomphalos</td>
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<td>Anencephalus and Exomphalos</td>
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<td>Diaphragmatic Hernia, Cystic Lung and Congenital Heart Disease</td>
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<tr>
<td>Diaphragmatic Hernia, Pulmonary Hypoplasia and Oesophageal Atresia</td>
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<td>Undescended Testes</td>
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<tr>
<td>Congenital Abnormalities of Urinary Tract</td>
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<td>Umbilical Hernia</td>
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<tr>
<td>Congenital Adrenal Hyperplasia</td>
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<td>Congenital Eye Anomalies</td>
<td></td>
<td>10</td>
<td>10</td>
<td>0</td>
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<td>Anomalies of Ear, Face &amp; Neck</td>
<td></td>
<td>10</td>
<td>10</td>
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<tr>
<td>Cleft palate and/or Lip</td>
<td></td>
<td>11</td>
<td>10</td>
<td>0</td>
<td>1</td>
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<tr>
<td>Pyloric Stenosis</td>
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<td>13</td>
<td>13</td>
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<td>Other anomalies of g.i. tract</td>
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<td>10</td>
<td>8</td>
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<td>Sternomastoid Tumour</td>
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<td>Congenital dislocation of hip</td>
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<tr>
<td>Unstable hip</td>
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<tr>
<td>Varus deformity of feet</td>
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<td>57</td>
<td>57</td>
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<td>0</td>
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<tr>
<td>Valgus deformity of feet</td>
<td></td>
<td>11</td>
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<tr>
<td>Other anomalies of feet</td>
<td></td>
<td>12</td>
<td>12</td>
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<td>Anomalies of limbs (excluding hips and feet)</td>
<td></td>
<td>7</td>
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<tr>
<td>Musculoskeletal anomalies</td>
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<td>11</td>
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<tr>
<td>Anomalies of face and bones</td>
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<td>3</td>
<td>3</td>
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<td>0</td>
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<tr>
<td>Anomalies of spine</td>
<td></td>
<td>3</td>
<td>2</td>
<td>0</td>
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<td></td>
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<tr>
<td>Anomalies of skin, hair &amp; nails</td>
<td></td>
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<td>17</td>
<td>0</td>
<td>0</td>
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<td>Cystic Fibrosis</td>
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<tr>
<td>Maple Syrup Urine Disease</td>
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<td>1</td>
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<td>Albinism</td>
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<td>Phenylketonuria</td>
<td></td>
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<tr>
<td>Acardiac Monster</td>
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<td>1</td>
<td>0</td>
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<td>Mermaid Fetus</td>
<td></td>
<td>1</td>
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<tr>
<td>Downs Syndrome</td>
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<td>15</td>
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</tr>
<tr>
<td>Patau Syndrome</td>
<td></td>
<td>2</td>
<td>0</td>
<td>0</td>
<td>2</td>
<td></td>
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<tr>
<td>Edwards Syndrome</td>
<td></td>
<td>2</td>
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<td>0</td>
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<td></td>
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<tr>
<td>Potters Syndrome</td>
<td></td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>4</td>
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<tr>
<td>Cornelia de Lange Syndrome</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
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<tr>
<td>Klippel-Trenaunay-Weber Syndrome</td>
<td></td>
<td>1</td>
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<tr>
<td>Pierre Robin Syndrome</td>
<td></td>
<td>1</td>
<td>1</td>
<td>0</td>
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<td></td>
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<tr>
<td>Homers Syndrome</td>
<td></td>
<td>2</td>
<td>2</td>
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</table>
Paediatric Assessment Unit

The Assessment Unit is concerned with the aftercare of infants who are considered to be at risk because of adverse factors affecting fetal or neonatal wellbeing. Its aims are to assist each infant to achieve his full potential for growth and development, and to ensure that by early intervention the effects of handicaps are minimised. Infants are recalled to the Clinic at key developmental ages and are followed to 2 and, in some cases, to 3 years of age. At each visit, in addition to physical examination, assessment of growth, of behavioural development, of diet, and of haematologic status are carried out. Neurological assessment and assessment by the physiotherapist are carried out where indicated. Figures for 1980 are shown with the figures for 1979 in parenthesis:

- Number of patients attending: 958 (886)
- Total number of visits: 1,394 (1,335)
- Additional visits for haematologic and dietetic assessment: 78 (51)
- Number of patients attending for physiotherapy: 276 (222)
- Referrals to other hospitals or units: 203 (136)

In general, the figures for 1980 are similar to those of the previous year. More patients attended physiotherapy, and the number of referrals to other hospitals and units went up from 136 to 203. These numbers reflect the increasing complexity of the cases and the more active management of potential handicaps. They include referral for specialist opinion in fields such as audiology and ophthalmology, psychology, neurology and neurosurgery as well as for E.E.G. and X-ray.

The following table illustrates the distribution of patients according to birth weight, with figures for 1979 in parenthesis:

<table>
<thead>
<tr>
<th>Birth Weight</th>
<th>1979</th>
<th>1980</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1.00 kilogram</td>
<td>4(3)</td>
<td></td>
</tr>
<tr>
<td>1.01-1.50 kilograms</td>
<td>62(52)</td>
<td></td>
</tr>
<tr>
<td>1.51-2.00 kilograms</td>
<td>91(80)</td>
<td></td>
</tr>
<tr>
<td>2.01-2.50 kilograms</td>
<td>165(112)</td>
<td></td>
</tr>
<tr>
<td>&gt;2.51 kilograms</td>
<td>623</td>
<td></td>
</tr>
<tr>
<td>Not weighed</td>
<td>13</td>
<td></td>
</tr>
</tbody>
</table>

Review of the work load of the Clinic over the last few years shows that patterns of morbidity are gradually changing. No longer do we see infants who have suffered birth trauma in the physical sense. The number of normal infants who suffer an acute asphyxiating experience during labour or delivery is also falling. However, with advances in paediatric intensive care, we are seeing increasing numbers of extremely premature low birth weight infants with all their attendant complications — the small infants who have survived ventilation and alimentation, recurrent apnoea and pneumothoraces, infection, anaemia and jaundice, to name but some of
their problems. These fragile babies require not only intensive initial care but long term management. Assessment in these infants requires careful distinction between the effects of prematurity itself and possible underlying defects of neurological abnormalities.

We are also seeing the survival of an increasing number of infants who have been subject to adverse factors during fetal life, and who go into labour in a compromised state. Small for gestational age infants and large deprived infants do not have the metabolic reserves to carry them through the stresses of even a normal labour. They survive because of fetal monitoring, and because of special methods of delivery and of post-natal care, and they present with evidence of neurological damage which has been incurred both in fetal life and during delivery. Examination of the maternal history often reveals a period of intra-uterine ill health and failure to grow as shown by clinical signs and by assessment of bi-parietal diameters, oestriol levels, maternal weight gain and C.T.G.'s. It is very probable that disturbance in brain growth and development also occurs during these periods to become overt later.

The time scale of morbidity in these cases is one that is altogether slower and less predictable than that associated with birth asphyxia. Early signs may be minimal, and may be limited to a poor feeding pattern and an abnormal cry. Many of these infants show a rapid increase in head growth after birth. Neurological dysfunction becomes evident at the 12 or 16 week examination and manifests as unusual disorders of tone and persistent abnormal primary responses. The patterns of neurological response are often bizarre and do not fit with already accepted patterns. If neurological signs become fixed these infants most frequently present with hemiplegic types of disorders. Some present as hyperactive infants with poor sleeping patterns and others have personality disorders or behavioural disorder such as head banging and cot rocking.

The introduction of computerised axial tomography and ultrasound as diagnostic aids have enabled perinatologists to look more closely at the events of parturition as they affect the brain. It is now clear that intracranial haemorrhage is much commoner than had previously been suspected, and that many infants survive even a significant intracranial bleed but may be left with areas of neuronal damage. As yet there is only a little knowledge of the degree to which repair or regeneration can occur. Nevertheless, there is an increasing awareness that the possibilities for compensation do exist, and that in the young brain other areas can take over function from a damaged area.

This is the basis for neuro-developmental training. Developmental attainment is a complex interweaving of forces, which in a normal individual proceeds in a regular and predictable fashion, each stage of development being an extension of the previous stage. But in the individual with an impaired central nervous system this process is disturbed and may be replaced by abnormal patterns of development. One of the important functions of the physiotherapist is to ensure that deviant patterns do not become established. For this reason timing of treatment to critical periods in development is of the utmost importance.

It is conceivable that the principles which have been found therapeutic in the field of motor development may also be applied in other areas. With experience in developmental assessment we are becoming conscious of more hidden areas of disordered development, particularly in the field of adaptive and early cognitive development. For example, the young child who has difficulty in spatial construction, or who lacks the concept of numbers or who is slow to develop colour recognition, has functional problems relating to different activities of the brain. It is possible that in the future timed intervention in these areas may be rewarding.
Neuromotor Dysfunction

Two hundred and seventy-six infants received physiotherapy during the year the total number of visits being 854. With the exception of a small number with miscellaneous conditions such as torticollis and metatarsus varus, the majority attended for neuro-developmental training. Our approach to the management of neuromotor dysfunction involving as it does training of the parents in the positioning and handling of their babies has been described in some detail in previous reports particularly 1978 and 1979. The results achieved through neuro-developmental training are very encouraging. Even infants with very adverse histories do remarkably well. A good genetic inheritance and a favourable environment are of paramount importance in the recovery phase, as is management of the infant during the stage when the central nervous system is "plastic".

However, there always remains the difficulty of predicting which babies will be unable to compensate and will show deterioration. Some appear to do well initially and then falter. In our experience contributory reasons may sometimes be environmental and may be found in the attitude of the parents, particularly the mother. Socio-economic conditions, intercurrent infection, and failure to establish good feeding patterns are also unfavourable factors. The situation is a two way process, and when a baby shows little progress it is difficult for the parents to maintain a hopeful attitude and the tendency is to become discouraged. Faced with lack of response on the part of the baby these parents need a great deal of outside support.

It is in this area that a study of growth patterns may be of value in predicting outcome. Retrospective analysis of patterns of growth and development of these babies shows that in many cases an upward swing which then falters would seem to indicate an attempt at recovery which is not successful. The ideal growth pattern is one where growth is proportionate and goes out along the centiles with regular increments. This pattern should be continuous from fetal through post-natal life. However, in some instances, because of maternal influence, the organism may be unable to attain or to maintain its full growth potential in-utero. After birth the organism will compensate by an increased rate of growth until the full level of potential is achieved, and this will show as a growth pattern which "rises through the centiles". The magnitude of the rise through the centiles will be an indication of the degree of growth restriction superimposed on the fetus.

On the other hand, if post-natal growth rate falls off and if the growth pattern is falling through the centiles, then one must assume that the organism is unable to sustain its own potential and must settle for a lower level. And this would appear to be what happens in many babies who have shown evidence of neuromotor dysfunction in the early stages. In other words, whereas recovery would seem to have taken place, it has been at the expense of failure on the part of the individual to realise full potential.

A total of 52 infants had neonatal seizures, either convulsions or twitching in association with cerebral signs. Eight were born in 1977, sixteen in 1978, sixteen in 1979 and twelve in 1980. There was considerable morbidity in this group of children, about half of whom have areas of developmental delay. Most showed evidence of neuromotor dysfunction at some time during the follow-up period, and 45 of the 52 received neuro-developmental training. Psychological assessment is carried out between 3 and 3½ years of age. It is a valuable indication of early cognitive development and parents find it helpful in planning for schooling. Seven children have been referred for long term management and will possibly have special educational needs.

Seventeen patients with a history of neonatal meningitis were also seen. Two had been born in 1977, two in 1978, seven in 1979 and six in 1980. Six
were preterm and eleven were term infants. Again this is a group with special problems and special needs.

A total of 13 cases were referred for long term management to the Central Remedial Clinic during 1980. A brief history of 8 cases is given here. Of the 5 remaining, 1 was of developmental delay associated with a family history of mental deficiency; 1 was a rubella syndrome with deafness and some developmental delay; and 3 were cases with varying complications following neonatal meningitis. We would like to take this opportunity of thanking Dr. Barry and his team for their co-operation and frequent reports.

<table>
<thead>
<tr>
<th>Case</th>
<th>Date of Birth</th>
<th>Gestation</th>
<th>Birth Weight</th>
<th>Problems</th>
<th>Management</th>
</tr>
</thead>
<tbody>
<tr>
<td>B.2801/M/79</td>
<td>09/10/1979</td>
<td>28 weeks</td>
<td>1.08 kg</td>
<td>Preterm jaundice and anemia</td>
<td>Follow-up in assessment clinic from 28 weeks. Initial progress good. Referred to Central Remedial Clinic at 18 months. Report: right hemiparesis. Alert and interested.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Case</th>
<th>Date of Birth</th>
<th>Gestation</th>
<th>Birth Weight</th>
<th>Problems</th>
<th>Management</th>
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</thead>
<tbody>
<tr>
<td>B.6340/M/79</td>
<td>03/10/1979</td>
<td>34 weeks</td>
<td>1.50 kg</td>
<td>Neonatal jaundice and anemia</td>
<td>Follow-up in assessment clinic from 46 weeks of age. Height and weight less than the 3rd centile. Head circumference on the 97th centile. Bilateral single palmar creases. Poor head control and neuromotor dysfunction with associated developmental delay. Physiotherapy. Referred to C.R.C. at 11 months of age. Early report: developmental delay.</td>
</tr>
<tr>
<td>B.4899/M/79</td>
<td>09/10/1979</td>
<td>37 weeks</td>
<td>2.2 kg</td>
<td>Pre-eclampsia toxaemia. Induced, meconium in liquor and type II dips. Delivered by Caesarean Section. Apgars 1 at one minute, 10 at five minutes. RDS. Initial progress in unit reasonably satisfactory. At 4 weeks of age developed signs of acute infection with patchy inflammation on chest X-ray, and E coli 044 in stools. Because of increasing head size investigated for hydrocephalus. C.A.T. scan negative. Assessment clinic from 46 weeks of age. Head circumference on the 97th centile. Bilateral single palmar creases. Poor head control and neuromotor dysfunction with associated developmental delay. Physiotherapy. Referred to C.R.C. at 7 months. Initial report: developmental delay but no gross motor signs. Report: cerebral palsy with valgus deformity and speech delay.</td>
<td></td>
</tr>
</tbody>
</table>
| B.140/M/80 | 01/10/1980 | 36 weeks | 2.13 kg | Pre-eclampsia toxaemia | Day two later right iliac fossa pain 7 abruption. A.R.M. and Syntocinon to induce. C.T.G. not in labour but type II dips. Emergency Caesarean Section. Apgars 3 at one minute, 8 at three minutes and 10 at five minutes. Intubated. At 2
days of age noted to be oedematous and sclerematous. Still oedematous at 10 days of age and the etiology was considered to be an infective process. Clostridii Welchii was isolated in the final sub-culture of the blood culture. At 10 days of age was transferred to Our Lady's Hospital, Crumlin. At this time he had a large heart with a murmur, was tachypneic and oedematous. Good recovery by 6 months. Assessment clinic follow-up from 27 weeks. Initially developmental progress delayed and irregular. Head growth was poor along the 3rd centile in relation to height and weight on the 25th centile. Tests for thyroid function were negative. Bone age at lower limit of normal. At 11 months of age developmental progress improved and at the same time head growth increased to 25th centile level. Referred to C.R.C. at 11 months. Reported as a passive obese child with delayed development.


Blood Group Incompatibility

In recent years only the more problem cases of blood group incompatibility are recalled to the assessment clinic. Some infants who live at long distances are followed in their local areas. A total of 36 infants with rhesus iso-immunization were seen for follow-up, 24 of whom had required exchange transfusions. Fifteen of the infants were born prematurely before 37 weeks gestation. In general these premature infants show somewhat irregular patterns of development with many minor deviations from normal, whereas the term infants tend to show normal patterns except in the area of speech development. As already reported we continue to note a significant incidence of speech delay and dysarthria even when the haemolytic components of the disease have been mild. Six patients had been born in 1977, 14 in 1978, 10 in 1979 and 6 in 1980, reflecting the falling incidence of rhesus disease. A total of 14 infants with ABO incompatibility and 2 siblings with Anti-Kell were also followed. These were mild cases and many were seen for reasons other than incompatibility. Three had required exchange transfusions.

During the past year much has been written on the subject of perinatal mortality and morbidity and on the prevention of handicap. The report of the Parliamentary Social Services Committee on perinatal and neonatal mortality\(^1\) is a significant one and makes interesting reading. Having been convinced that modern obstetrical techniques can to some extent compensate for the hazards to the fetus of adverse social circumstances, the Committee has advocated that certain measures should be implemented. The report lists the factors responsible for perinatal and neonatal deaths in two categories: lack of education, poverty, poor housing, possibly poor nutrition, unplanned pregnancy, smoking and excess alcohol on the one hand; and lack of antenatal care, low birth weight, asphyxia during and after delivery, congenital malformations, and birth injury on the other. Having been convinced that modern obstetrical techniques can to some extent compensate for the hazards to the fetus of adverse social circum-
stances, the Committee has spelt out a programme to put that policy into action. Firstly, medical intervention has to be aimed at the women at high risk; secondly, standards of equipment and staffing have to be improved, and thirdly, improvements are needed in the collection and analysis of data on the short term and long term results of medical intervention. In addition the report suggests that each region should set up a perinatal working party with the duty of monitoring obstetric and neonatal work, rationalizing services, and reviewing the perinatal deaths. Of particular interest to us it also recommends studies of the long term outcome for infants at high risk of mental and physical handicap. It has been described as a detailed and authoritative report which comes down unequivocably on the side of specialist skills and setting of minimum standards.

There is a current impression that, in the situation where there is a large volume of normal or near normal pregnancies, the mother at risk may not be receiving sufficient attention. The identification of the mother at risk is therefore important, and high in this category must come mothers who have already had an infant with neonatal morbidity. Women of small stature and light build are particularly well represented among assessment clinic mothers. Taking mean height in women to be 5' 4". 10 per cent of mothers attending during 1980 were 4' 11" or less, and 25 per cent were 5' 1" or less. Our experience has been that it is necessary to look at risk factors not in isolation, but in combination with each other. For example, the small mother may be at risk if she is carrying a large baby, but the risk will be compounded if she is in an older age group, has a history of urinary tract infection, is a heavy smoker, belongs to a lower socio-economic group, and has a history of a previous neonatal death.

For this reason we would like to see, what one might term, a pregnancy profile for each mother, which would embrace all risk factors including physical ones such as mother's height and weight (body mass), age, and father's height and age. Previous reproductive history and previous medical history and drug ingestion should also be recorded. If each factor were given a value, then a risk total could be calculated. Those mothers with a high risk total would then merit the greater surveillance.

Clinical Research

The breech project continued during the year. The study is a prospective and longitudinal one, and comprehensive in that it includes all infants presenting as a breech at term, born in this hospital over a period of 9 months. Dr. Anne Keane has completed the collection of the data and its analysis to two years of age.

Dr. Peter Gray undertook a special project to complete the follow-up of infants weighing less than 1.50 kilograms born during 1977. The data gathered is being utilised in a further research study of low birth weight infants.

A paper entitled "A Prospective Study of Growth, Development and Neurological Function in Infants Presenting as a Breech at Term" was presented at the meeting of the Irish and American Paediatric Society in Cork in September 1980.

We would like to record grateful appreciation to our secretary, Miss Bernadette Thompson, nurse, Mrs. Ryan, dietitian, Mrs. Yvonne Walsh and haematology technologist, Mrs. McKeown. In particular we thank Mrs. Coote and the other members of the Physiotherapy Department.

References:
Deliveries less than 28 weeks weighing 500 grams and over

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of infants</td>
<td>19</td>
</tr>
<tr>
<td>Incidence per cent</td>
<td>2.6</td>
</tr>
<tr>
<td>Dead born</td>
<td>5</td>
</tr>
<tr>
<td>Live born</td>
<td>14</td>
</tr>
<tr>
<td>Survived</td>
<td>7</td>
</tr>
</tbody>
</table>

Comment: The number of patients who delivered infants weighing over 500 grams before 28 weeks' gestation was similar to that reported last year. However, a most significant change occurred in the outcome in that 7 of the 14 live born infants survived. This figure represents a remarkable improvement on any figure recorded previously in this hospital and is a direct result of the improved intensive care available in the Neonatal Department. The birth weights of the surviving infants ranged from 730 grams to 1270 grams, with 4 of the infants weighing less than 1,000 grams. It is gratifying to record that 6 of the 7 surviving infants are, to date, developing satisfactorily. One baby, however, has been lost to follow-up despite every effort to maintain contact.

Dead Born

B.50851, Para 3\*\* Age 33. S.E. 3. Booked at 8 weeks gestation. Only one living child due to severe rhesus haemolytic disease. Plasmapheresis started at 10 weeks gestation. Despite this the fetus showed hydropic changes at 22 weeks and intrauterine transfusion was given at this time but IUD and spontaneous abortion one week later of male infant weighing 936 grams.


Live Born

B.49792, Para 1\*\*\*. Age 19. Gestation 26 weeks. Booked at 24 weeks. Admitted in premature labour at 26 weeks. F.H.N.H. Spontaneous breech delivery of live born male infant weighing 1.1 kilograms, born with Apgar scores of 1 and 5. Large retroplacental clot. Following admission to the Special Care Unit the baby was ventilated. On the 7th day he had a pulmonary haemorrhage which responded well to ventilatory adjustment. The infant also had a patent ductus arteriosis with associated congestive cardiac failure. As the baby had a coagulopathy the ductus was treated with fluid restriction only. The ductus appeared to close spontaneously with this at the age of 4 weeks and the baby was finally extubated at the age of 6 weeks.
Following this the baby’s course was uneventful and he was discharged at the age of 113 weeks weighing 2.5 kilograms with a head circumference of 34 cm. This infant did not return for follow-up despite every effort and it is noted that the mother had had another low birth weight infant during the previous 12 month period.

B.54517. Para 0\textsuperscript{12}. Age 20. Gestation 26 weeks. Booked at 19 weeks gestation. Admitted with vaginal bleeding at 25 weeks. Further bleeding following admission and premature labour at 26 weeks. Assisted breech delivery of female infant weighing 737 grams, born with Apgar scores of 2 and 5. Resuscitation was not initially undertaken because of "extreme prematurity". At 35 minutes of age she was admitted to the Special Care Unit and was resuscitated with ventilation as the baby had already established respiration with a good apex beat. The initial ventilatory progress was satisfactory but subsequently the baby went on to develop severe broncho-pulmonary dysplasia so she ultimately required ventilatory assistance for 126 weeks prior to extubation. Her course was also complicated by a labile patent ductus arteriosus. She was supported by total parenteral nutrition for the first 8 weeks, then she went on to naso-jejunal feeds. She was discharged home at the age of 18 weeks weighing 2.7 kilograms with a head circumference of 35.3 cm. She has subsequently been seen for review and, apart from one hospital admission for respiratory tract infection, is doing well.

B.3597. Para 3\textsuperscript{0}. Age 30. Gestation 23 weeks. Booked at 16 weeks gestation. Admitted with vaginal bleeding at 22 weeks. Premature labour one week and spontaneous delivery of male infant weighing 530 grams which died within a few hours.

B.43762. Para 4\textsuperscript{0}. Age 33. Gestation 27 weeks. Booked at 17 weeks gestation. Admitted a few days later with vaginal bleeding. Intermittent bleeding continued and premature labour at 27 weeks. Spontaneous delivery of male infant weighing 992 grams. Prior to delivery the fetal heart had not been recorded and on admission to the Special Care Unit, the infant was oedematous and foul smelling. Baby was resuscitated, its circulation supported and full ventilation given. Over the ensuing weeks he developed pneumothoraces and sepsis but at no time was there evidence of intraventricular haemorrhage. He was eventually taken off respiratory assistance, apart from an oxygen hood, at the age of 9 weeks. In addition he had problems with prolonged obstructive-type jaundice which eventually cleared spontaneously. The baby was discharged at the age of $\text{M months}$, i.e. term +9 days. At this time his weight was 2.71 kilograms and his head circumference was 35 cm. Initially his parents had difficulty. When last seen, at the age of $5^5$ months, he was doing well and no neurodevelopmental abnormality was detected.

B.46518. Para 1\textsuperscript{1}. Age 23. Gestation 27 weeks. Booked at 16 weeks gestation. Admitted with vaginal bleeding at 19 weeks. Subsequent premature rupture of membranes at 22 weeks followed by premature labour at 27 weeks. Breech delivery of live born male infant weighing 1.27 kilograms with Apgar scores of 1 and 5. Initial pH was 6.93. Infant was given full ventilation and antibiotics. By the fourth day the baby was weaned on to C.P.A.P. for his respiratory distress syndrome but this was only for a short time as he developed apnoea which required him to be ventilated again. He then went on to have problems with atelectasis and pneumothoraces prolonging his period of requirement of respiratory support for a period of 83 weeks before he was weaned on to an oxygen hood. In addition, during his course baby had seizures which were controlled with phenobarbitone. He was discharged home at the age of 12 weeks weighing 2.65 kilograms, with a head circumference of 34.5 cm. Follow-up has been uneventful and, in particular, he has no evidence of significant pulmonary sequelae.

B.45735. Para 1\textsuperscript{1}. Age 30. Gestation 27 weeks. Booked at 21 weeks gestation. Admitted with vaginal bleeding at 27 weeks. Progressed in labour and assisted breech delivery of live born male infant weighing 1.08 kilograms. Baby was admitted to the S.C.B.U. and ventilated. Initial pH 7.079 which readily corrected with ventilation. The initial respiratory disease was due to hyaline membrane disease and on clearing the baby had pulmonary atelectasis which required continued respiratory support. At the age of 6 weeks, the baby was finally extubated and put in an oxygen hood. In addition, during his course baby had seizures which were controlled with phenobarbitone. He was discharged home at the age of 12 weeks weighing 2.53 kilograms and head circumference 34.3 cm. Follow-up of this baby has been uneventful.

B.57106. Para 0\textsuperscript{0}. Age 26. Gestation 26 weeks. Booked at 15 weeks gestation. Admitted with vaginal bleeding at 26 weeks and progressed in labour. Spontaneous delivery of male infant weighing 960 grams, born with Apgar scores of 5 and 8. Following admission to the S.C.B.U. the infant was put on C.P.A.P. and shortly after this required full ventilation. His course was complicated by pneumothoraces. He also had a patent ductus arteriosus. It was not possible to use Indomethacin prior to the 17th day because of an elevated serum creatinine; however, when used the patent ductus closed on the 18th day. The baby was subsequently weaned from the ventilator over the next few days with the aid of theophylline. However, further recurrent atelectasis required the baby to have continued assistance with ventilation until he was extubated at the age of 8 weeks. Though the baby had occasional myclonic jerks, no evidence of intraventricular haemorrhage was detected on repeated ultrasound scans. This infant was fed with maternal breast milk but developed neonatal rickets. He did not respond
to vitamin D and was given cholecalciferol before his rickets healed. This is of particular interest as the infant was also receiving regular vitamin D doses throughout his course, in addition to breast milk, and there was no reason to believe that his mother was malnourished but it does suggest that there was inadequate vitamin D available for the growth and development of his rickets which may have contributed to his recurrent atelectasis. He was discharged home at the age of 13 weeks with a weight of 2.75 kilos and head circumference of 34 cm. Follow-up has been satisfactory to date.


Post-mortem: (i) Intraventricular cerebral haemorrhage; (ii) Pulmonary atelectasis.

B.55421. Para 0. Age 28. Gestation 27 weeks. Premature labour and delivery of female infant weighing 1.1 kilograms, with Apgar scores of 8 and 10. Baby was admitted to the S.C.B.U. following delivery which was precipitated by spontaneous rupture of membranes associated with a large retroplacental clot. Following delivery the baby required C.P.A.P. and subsequently ventilation was required intermittently. The baby developed a patent ductus arteriosis which closed successfully with indomethacin only to reopen later. The main problem with this infant was recurrent apnoea associated with atelectasis. This infant had one possible episode of aspiration pneumonia but overall had a relatively benign course with moderate respiratory distress syndrome. The baby was discharged home at the age of 11 weeks weighing 2.12 kilograms and head circumference of 33.5 cm. The baby has done well since discharge and is continuing under review.

N.B. 42754. Para 9. Age 32. S.E. 4. Gestation 27 weeks. Admitted with vaginal bleeding at 27 weeks gestation. Uterus bigger than dates and scan confirmed twin pregnancy. Cervix fully dilated 2 hours after admission. Both twins delivered by the breech. First twin female, weighed 1 kilogram. Following resuscitation the baby had evidence of respiratory distress and was intubated and given E.T.T. C.P.A.P. However, no ventilator was available and the baby was maintained on C.P.A.P. and the baby had evidence of respiratory failure. When Twin II died, Twin I was connected to a ventilator and the baby's condition generally improved. However, persistent metabolic acidosis was present and the baby was clinically felt to have had an intracranial haemorrhage. This was also associated with fresh blood from the endotracheal tube and the baby continued to deteriorate with death at 42 hours of age.

Post-mortem: (i) Massive intraventricular haemorrhage; (ii) Pulmonary immaturity and haemorrhages.

Second twin, male, weighed 950 grams and was born with an Apgar score of 6. Following delivery this infant had poor respiratory effort and was commenced on assisted ventilation immediately upon arrival in the S.C.B.U. The baby had extensive bruising and did not improve despite full resuscitation. Baby died at 4 hours of age.

Post-mortem: Pulmonary atelectasis and immaturity with bilateral medullary adrenal haemorrhages.

B.52609. Para 0. Age 19. (Innupta). Gestation 23 weeks. Booked at 12 weeks gestation with threatened abortion. Admitted at 23 weeks with vaginal bleeding and crampy abdominal pains. Spontaneous delivery the following day of live born male infant weighing 690 grams. No active measures were taken and the infant died at 3 hours of age.

Post-mortem: Pulmonary immaturity and atelectasis.


Post-mortem: Pulmonary immaturity and atelectasis.

Maternal Deaths

Number of cases: 1
Maternal mortality rate: 0.1/1,000

There was 1 maternal death during 1980 and the case history is summarised below.

B.48712. Para 0\(0^+\). Age 35. Gestation 41 weeks. Height 128 cm. Severe kyphoscoliosis and extremely short stature. Husband died during pregnancy. Normal antenatal course. Vertex not engaged at term, but pelvimetry surprisingly showed normal pelvic measurements. Labour induced 10 days past term, at which time the liquor was found to be meconium stained. Emergency Caesarian Section, three hours later, for fetal distress. Male infant 3.0 kilograms born with Apgar scores of 9 and 10. The operation itself went uneventfully but shortly afterwards the patient collapsed with signs of intraperitoneal bleeding. A laparotomy was performed and an arterial vessel was found bleeding from the posterior wall of the uterus, quite separate from the lower uterine incision. It was impossible to achieve complete haemostasis and a hysterectomy was performed. During this procedure the patient was transfused with 10 units of blood and 2 units of fresh frozen plasma because of an associated coagulation defect. She subsequently developed severe pulmonary oedema and required ventilation. Following this her condition improved and appeared to have been stabilised. She was transferred to a General Hospital the next day. Unfortunately, she subsequently developed a cardiac arrest to which she responded initially prior to developing a second cardiac arrest which led to her death 4 days after Caesarian Section.

Post-mortem:
(i) Bilateral pulmonary embolus.
(ii) Broncho-pneumonia

Bilateral pulmonary infarction was confirmed on histological examination of the lungs. In the opinion of the pathologist death was due to severe uterine haemorrhage followed by coagulation defect and shock.

Comment: From a clinical point of view it is difficult to say whether this death was directly due to the haemorrhage following Caesarean section or whether it occurred as a direct result of the pulmonary emboli. Insofar as the precipitating cause of this patient’s demise was haemorrhage secondary to Caesarean section it should be deemed avoidable. But in fact it seems likely that this complication had been overcome when the pulmonary emboli caused the patient’s death. This was a particularly tragic case in that the patient’s husband died early on in the pregnancy and for the remainder of the pregnancy the mother herself kept saying that she wished to join him.
Cardiac Department

Number of cases: 16
Incidence per cent: 0.19
Perinatal deaths: Nil

Sixteen patients with heart disease or with a history of heart disease were delivered during the year. They included the following cases:

- Patent ductus arteriosis (ligated): 2
- Atrial septal defect (repaired): 2
- Small haemodynamically insignificant VSD: 1
- Paroxysmal atrial fibrillation secondary to thyrotoxicosis: 1
- Isolated atrial fibrillation: 1
- Symptomatic ventricular ectopic beats: 1
- Mild pulmonary stenosis: 3
- Mild mitral regurgitation: 2
- Mild mitral stenosis: 1
- Mild combined mitral regurgitation and stenosis: 1
- Mild aortic incompetence: 1

No maternal or foetal death occurred. Four patients had normal hearts (ligated patent ductus 2; repaired ASD, 2).

Three patients diagnosed as having mild pulmonary stenosis had grade 3 systolic murmurs in the pulmonary area. They were classified as mild congenital pulmonary stenosis but some or all of these may have had no significant heart disease.

Only five patients had valve disease of rheumatic origin and all these patients were very mild. No patient presented with coronary heart disease.

Thus the results for 1980 confirm the trend of recent years of fewer cases of rheumatic disease and of mild cases when they do occur. There has been no suggestion of an increased incidence of coronary heart disease in child-bearing women and heart disease in general is becoming a rarity amongst them. No maternal death from heart disease has been reported from the Coombe Hospital for more than 12 years.

The reduction in incidence of heart disease amongst child-bearing women is manifested by the number of electrocardiograms performed in the Coombe Lying-in Hospital for in-patients and out-patients over the past 9 years. Thirty-eight electrocardiograms were performed in 1980. The average number of ECGs performed annually during the preceding 8 years was 60.

## COMPARATIVE TABLE FOR 10 YEARS

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<td>36</td>
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<td>18</td>
<td>16</td>
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<td>Incidence %</td>
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<td>0.4</td>
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<td>0.4</td>
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<td>0.21</td>
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<tr>
<td>Perinatal deaths</td>
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<td>Nil</td>
<td>Nil</td>
<td>1</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
</tr>
<tr>
<td>Mortality Rate</td>
<td>60.6</td>
<td>62.5</td>
<td>37.0</td>
<td>Nil</td>
<td>Nil</td>
<td>33</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
</tr>
</tbody>
</table>
Latent Diabetes Mellitus

Number of cases . . . . . . 71
Incidence per cent . . . . . . 0-9
Number of babies delivered 71
Stillbirths . . . . . . . . . . . 2
Neonatal deaths . . . . . . . Nil
Perinatal deaths . . . . . . . 2 Rate: 28.2/1,000

Comment: The number of patients with latent diabetes has decreased during the year. An increased awareness of this condition is necessary and on occasions the referring of these patients to the clinic has been unnecessarily delayed. In general, the latent diabetes is controlled by diet and regular visits with 2 hour post-prandial blood sugars. Induction at term is preferred. One of the two stillbirths (74) was an avoidable intrapartum death where a type II deceleration was not noted and acted upon; the other stillbirth (70) was due to a congenital abnormality.

PERINATAL DEATHS

Normal Infant


Post-mortem: Intra-partum asphyxia.

Congenital Malformation

Case No. 70

COMPARATIVE TABLE FOR TEN YEARS

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</tr>
</thead>
<tbody>
<tr>
<td>Number of cases</td>
<td>73</td>
<td>157</td>
<td>132</td>
<td>126</td>
<td>134</td>
<td>142</td>
<td>108</td>
<td>101</td>
<td>97</td>
<td>71</td>
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<tr>
<td>Incidence %</td>
<td>1.1</td>
<td>2.0</td>
<td>1.8</td>
<td>1.6</td>
<td>1.8</td>
<td>2.0</td>
<td>1.5</td>
<td>1.4</td>
<td>1.27</td>
<td>0.9</td>
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<td>Perinatal deaths</td>
<td>1</td>
<td>9</td>
<td>6</td>
<td>4</td>
<td>1</td>
<td>3</td>
<td>Nil</td>
<td>2</td>
<td>3</td>
<td>2</td>
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<tr>
<td>Mortality rate</td>
<td>13.7</td>
<td>56.9</td>
<td>44.4</td>
<td>31.7</td>
<td>7.4</td>
<td>21</td>
<td>Nil</td>
<td>19.2</td>
<td>31.2</td>
<td>28.2</td>
</tr>
</tbody>
</table>
Clinical Diabetes Mellitus

Number of cases . . . . 12
Incidence per cent . . . . 0.15
Caesarean Sections ... 2
Perinatal deaths . . . . 2 Rate: 166/1,000

Comment: 2 patients were diagnosed as clinical Diabetics during pregnancy under review; in each case the diagnosis was confirmed by an oral glucose tolerance test after the puerperium. Regrettably one case (43) was diagnosed only after intra-uterine death occurred. This patient had attended the hospital clinic on 3 occasions and despite repeated glycosuria had not had any blood sugar levels estimated. This, therefore, was an avoidable death, that should have been referred to the diabetic clinic at an earlier stage in pregnancy. 9 of the 10 known clinical Diabetics were insulin dependent prior to pregnancy. The distribution of cases according to the classification of White was: A (2), B (4), C (1), D (3), F.R (2).

The second intra-uterine death occurred in a Class F patient at 30 weeks gestation. The patient had marked pre-eclampsia and severe intra-uterine growth retardation had been observed for many weeks prior to intra-uterine death. However, the fetus was so small that earlier delivery would only have resulted in neonatal death and have given rise to an unnecessary operation.

PERINATAL DEATHS

Normal Infants

(43) B.55759. Para 0\textsuperscript{10}. Age 25. S.E.4. Gestation 28 weeks. First visit at 17 weeks gestation, - glycosuria +3. further visit 6 weeks later, - glycosuria +2. Subsequent visits at 23 and 25 weeks showed no glycosuria but there was progressive weight loss. U.T.I, treated with Nitrofurantoin. Admitted at 27 weeks with vomiting. Within 24 hours developed ketoacidosis and coma. Insulin commenced and improvement was rapid. However, the fetal heart disappeared on the morning of the 28th week of pregnancy. Spontaneous labour and delivery of stillborn female infant weighing 1.21 kilograms. Clinical diagnosis: I.U.D. due to diabetes.

Post-mortem: Intra-uterine anoxia.

(44) B.1969. Para 3\textsuperscript{12}. Age 30. S.E.3. Diabetic patient for 21 years. Gestation 30 weeks. Booked at 6 weeks gestation, which was confirmed by ultrasound scan. Insulin levels were controlled and regular attender. However, noted to have retarded intra-uterine growth from 24 weeks gestation onwards. Grossly dysmature and I.U.D. at 30 weeks. At this time the uterine fundus was only equal to that of a 24 week pregnancy. Spontaneous labour and delivery one week later of female macerated infant weighing 850 grams.

Post-mortem: Refused

COMPARATIVE TABLE FOR TEN YEARS

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<tr>
<td>Number of cases</td>
<td>--</td>
<td>9</td>
<td>10</td>
<td>9</td>
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<td>10</td>
<td>12</td>
<td>11</td>
<td>19</td>
<td>12</td>
</tr>
<tr>
<td>Incidence %</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.1</td>
<td>0.14</td>
<td>0.17</td>
<td>0.15</td>
<td>0.25</td>
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<tr>
<td>Perinatal deaths</td>
<td>Nil</td>
<td>2</td>
<td>1</td>
<td>1</td>
<td>Nil</td>
<td>2</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>2</td>
</tr>
<tr>
<td>Mortality</td>
<td>Nil</td>
<td>200</td>
<td>111</td>
<td>111</td>
<td>Nil</td>
<td>200</td>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>166</td>
</tr>
</tbody>
</table>
**Haemolytic Disease of the Newborn**

Number of cases . . . . . 71
Incidence per cent . . . . . 0.9
Number of babies delivered 71
Stillbirths . . . . . . . . . Nil
Neonatal death . . . . . . . . 1
Perinatal death . . . . . . . . 1 Rate: 14.0/1,000

*Comment:* The incidence of haemolytic disease of the newborn increased slightly during 1980. The higher incidence was due to an increased incidence of ABO incompatibility. However, the majority of babies were only mildly affected and only 1 baby died.

The types of haemolytic disease encountered during the year are summarised in the table together with a resume of the therapy required for each particular problem. It can be noted from the table that some of the babies with ABO incompatibility did not require any therapy, though their Coombs test was positive. In addition to the 54 babies given phototherapy for haemolytic disease, a total of 254 other babies required phototherapy for jaundice caused by other factors.

<table>
<thead>
<tr>
<th>Type of Haemolytic Disease</th>
<th>Number of babies affected</th>
<th>Number of babies exchanged</th>
<th>Number of exchange transfusions</th>
<th>Number of babies given simple transfusions</th>
<th>Number of babies given phototherapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhesus</td>
<td>22</td>
<td>11</td>
<td>20</td>
<td>6</td>
<td>18</td>
</tr>
<tr>
<td>Anti-A</td>
<td>26</td>
<td>1</td>
<td>1</td>
<td>1</td>
<td>19</td>
</tr>
<tr>
<td>Anti-B</td>
<td>19</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>16</td>
</tr>
<tr>
<td>Others</td>
<td>4</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td><strong>Total:</strong></td>
<td><strong>71</strong></td>
<td><strong>13</strong></td>
<td><strong>23</strong></td>
<td><strong>8</strong></td>
<td><strong>54</strong></td>
</tr>
</tbody>
</table>

There was only 1 perinatal death during 1980, and this was due to anti-Kell antibodies. These antibodies presumably developed as a result of a blood transfusion in the patient's previous pregnancy. This patient was rhesus positive and the development of anti-Kell antibodies since her previous pregnancy demonstrates the importance of screening all patients at first visit for antibodies even though their blood group is known to be rhesus positive. Approximately 2 per cent of patients who receive blood transfusions will subsequently develop antibodies which may cause haemolytic disease of the newborn. During the year anti-D immunoglobulin was administered to 676 puerperal patients and to 125 rhesus negative women who aborted.

In addition to the 71 cases described above, one patient who had severe rhesus haemolytic disease was commenced on plasmaphoresis at 10 weeks gestation. However, despite this the baby became hydropic and an intrauterine transfusion was performed at 22 weeks gestation. However intra-
uterine death occurred one week later and the patient was delivered of a dead born male infant weighing 936 grams. This death has been included in the chapter relating to deliveries under 28 weeks gestation.

PERINATAL DEATH

Normal Infant

(83) B. 18689. Para 2°. Age 28. S.E. 3. Gestation 30 weeks. Booked at 10 weeks gestation. Blood group A Rh. positive. Previous pregnancies (i) full term delivery followed by primary P.P.H. and blood transfusion, (ii) full term normal delivery, no complications. Present pregnancy uneventful until 29 weeks gestation when patient complained of oedema of legs, feeling tired and being uncomfortable. B.P. 140/100, no proteinuria, hydramnios. Scan showed huge placenta, Grade I-II praevia. Infant appeared hydropic with ascites. Mother found to have anti-Kell antibodies. A.P.H. and emergency L.S.C.S. with delivery of female infant weighing 2.2 kilograms, born with Apgar scores of 1 and 1. The baby was markedly hydropic and required intubation. Hydrops fetalis secondary to anti-Kell antibodies. Baby given 2 exchange transfusions and 3 top-up transfusions. Developed hypoglycaemia and hypercalcaemia which were treated with peritoneal dialysis. Baby failed to respond and died at 7 days of age. The mother was given 6 units of blood during L.S.C.S. and developed puerperal pyrexia due to bacteroides but made a good recovery. Post-mortem: (i) Massive intra cerebral haemorrhage; (ii) Massive extra medullary haematopoiesis related to haemolytic disease; (iii) traumatic emphysema and subpleural bulla.

COMPARATIVE TABLE FOR 10 YEARS

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<td>129</td>
<td>83</td>
<td>63</td>
<td>53</td>
<td>64</td>
<td>49</td>
<td>77</td>
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<tr>
<td>Incidence %</td>
<td>1.1</td>
<td>1.1</td>
<td>0.78</td>
<td>1.7</td>
<td>1.15</td>
<td>0.9</td>
<td>0.75</td>
<td>0.85</td>
<td>0.64</td>
<td>0.9</td>
</tr>
<tr>
<td>Perinatal deaths</td>
<td>12</td>
<td>17</td>
<td>6</td>
<td>8</td>
<td>4</td>
<td>3</td>
<td>4</td>
<td>15</td>
<td>1</td>
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</tr>
<tr>
<td>Mortality rate</td>
<td>150</td>
<td>195</td>
<td>101</td>
<td>62</td>
<td>47.6</td>
<td>47</td>
<td>74</td>
<td>15.6</td>
<td>65.4</td>
<td>14.0</td>
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</tbody>
</table>
Anaemia

(A Haemoglobin reading of less than 10 grams per cent on one occasion).

- Number of cases: 378
- Incidence per cent: 4.9
- Number of babies delivered: 394
- Stillbirths: 9
- Neonatal deaths: 7
- Perinatal deaths: 16 Rate: 40/1,000

Comment: The incidence of anaemia continued to fall during 1980. Sixteen of the 378 cases (4.1 per cent) occurred in association with multiple pregnancy. Six of the patients required treatment with blood transfusion; the remainder were treated with iron and folic acid alone. Twenty-seven of the 513 infants weighed less than 2.5 kilograms at birth. While the anaemia, per se, was not directly responsible for any of the perinatal deaths, one stillbirth (47) occurred as a direct result of jaundice and anaemia.

PERINATAL DEATHS

Normal Infants


Post-mortem: Maceration.


Post-mortem: Intra-uterine anoxia.


Post-mortem: Maceration.

(44) B.1969. Para 3\textsuperscript{+2}, Age 30. S.E. 3. Diabetic patient for 21 years. Gestation 30 weeks. Booked at 6 weeks gestation, which was confirmed by ultrasound scan. Insulin levels were controlled and regular attender. However, noted to have retarded intra-uterine growth from 24 weeks gestation onwards. Grossly dysmature and I.U.D. at 30 weeks. At this time the uterine fundus was only equal to that of a 24 week pregnancy. Spontaneous labour and delivery one week later of female macerated infant weighing 850 grams.

Post-mortem: Refused.

(47) N.B.53432. Para 0\textsuperscript{0}, Age 20. S.E. 5. Gestation 37 weeks. Transferred from country hospital at 37 weeks gestation with jaundice of one week's duration. F.H.H. on admission. Patient was jaundiced with high bilirubin levels, very low platelet levels and moderately raised transaminases. A provisional diagnosis of either leptospirosis, obstructive jaundice or viral
hepatitis was made. Two days following admission, patient went into spontaneous labour. F.H.N.H. Prophylactic forceps delivery of a stillborn female infant weighing 2.65 kilograms. Primary post-partum haemorrhage treated with platelets and blood. Patient improved following delivery. Three days later patient transferred to St. Vincent's Hospital. There liver biopsy performed and diagnosis of acute fatty liver made.

Post-mortem: Maceration.

(78) B.41239. Para 1°. Age 30. S.E. 2. Gestation 42 weeks. Booked at 8 weeks gestation. Uneventful A.N.C. except for anaemia. Admitted at 38 weeks gestation because of oblique breech. By 42 weeks cephalic presentation. Stable but high. Clinically big baby. PGE induction. Head did not descend in pelvis in second stage of labour and L.S.C.S. performed. Male infant weighing 4.71 kilograms, born with Apgar scores of 5 and 4. Baby was admitted to S.C.B.U. and following resuscitation the endotracheal tube was removed at 25 mins. Initial pH showed severe mixed metabolic respiratory acidosis, with a pH of 6.8. Following extubation the baby had poor respiratory effort with extreme hypotonia and staring associated with a fine tremor of face and feet. Treatment with Mannitol and Dexamethasone was commenced and the baby was slow to pass urine. At 24 hrs. of age the baby was noted to have being increasing respiratory distress associated with pneumonia. The baby was further supported with ventilation with some improvement. Neurological examination continued to be very abnormal. At 46 hours of age the baby was markedly hypoxic and despite every effort to improve ventilation the baby died.

Post-mortem: (i) Severe anoxic changes in the brain; (ii) Bilateral bronchial pneumonia; (iii) Pneumococcus was grown from the endotracheal tube and eye swab.

(80) B.25055. Para 2°. Age 34. S.E. 3. Gestation 29 weeks. Booked at 10 weeks gestation. Regular attender at the antenatal clinic. Admitted at 29 weeks in labour with transverse lie. L.S.C.S. Male infant weighing 1.55 kilograms born with Apgar scores of 8 and 9. Following admission to S.C.B.U. the baby was intubated and given C.P.A.P. Clinical kyphoscoliosis was confirmed with X-ray. Full septic work up was carried out in view of P.R.O.M. Chest X-ray was consistent with mild R.D.S. Antibiotics were commenced. Over the following 24 hours the baby became oedematous and at the age of 24 hours the infant collapsed. This was in association with severe metabolic acidosis with subsequent attacks of severe apnoea. The oedema increased and a pericardial effusion was aspirated. The baby had subsequent cardiac arrests and died at 3s days of age. Lumbar puncture performed at this time was bloodstained. All cultures were sterile apart from an eye swab which grew actinobacter species which was sensitive to the antibiotic used.

Post-mortem: (i) Massive intraventricular haemorrhage; (ii) Haemopericardium; (iii) Pulmonary and renal harmorrhages.

(83) B.18689. Para 2°. Age 28. S.E. 3. Gestation 30 weeks. Booked at 10 weeks gestation. Blood group A Rh. positive. Previous pregnancies (i) full term delivery followed by primary P.P.H. and blood transfusion, (ii) full term normal delivery, no complications. Present pregnancy uneventful until 29 weeks gestation when patient complained of oedema of legs, feeling tired and being uncomfortable. B.P. 140/100, no proteinuria, hydramnios. Scan showed huge placenta, Grade I-II praevia. Infant appeared hydropic with ascites. Mother found to have anti-Kell antibodies. A.P.H. and emergency L.S.C.S. with delivery of female infant weighing 2.2 kilograms, born with Apgar scores of 1 and 1. The baby was markedly hydropic and required intubation. Hydrops fetalis secondary to anti-Kell antibodies. Baby given 2 exchange transfusions and 3 top-up transfusions. Developed hypoglycaemia and hypercalcaemia which were treated with peritoneal dialysis. Baby failed to respond and died at 7 days of age. The mother was given 6 units of blood during L.S.C.S. and developed puerperal pyrexia due to bacterioides but made a good recovery.

Post-mortem: (i) Massive intra cerebral haemorrhage; (ii) Massive extra medullary haematoioesia related to haemolytic disease; (iii) traumatic emphysema and subpleural bulla.

Congenital Malformations

Cases: 55, 57, 67, 72, 94,102,106,108.

Comperative Table for 9 Years

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of cases</th>
<th>Incidence %</th>
<th>Perinatal deaths</th>
<th>Mortality rate</th>
</tr>
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<tbody>
<tr>
<td>1972</td>
<td>287</td>
<td>3.7</td>
<td>5</td>
<td>16.9</td>
</tr>
<tr>
<td>1973</td>
<td>468</td>
<td>6.2</td>
<td>16</td>
<td>33.2</td>
</tr>
<tr>
<td>1974</td>
<td>610</td>
<td>8.0</td>
<td>16</td>
<td>40.7</td>
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<tr>
<td>1975</td>
<td>728</td>
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<td>1976</td>
<td>649</td>
<td>9.2</td>
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<td>24</td>
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<tr>
<td>1977</td>
<td>550</td>
<td>7.8</td>
<td>9</td>
<td>16.1</td>
</tr>
<tr>
<td>1978</td>
<td>406</td>
<td>6.7</td>
<td>4</td>
<td>9.63</td>
</tr>
<tr>
<td>1979</td>
<td>505</td>
<td>4.9</td>
<td>10</td>
<td>19.5</td>
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<tr>
<td>1980</td>
<td>378</td>
<td>4.9</td>
<td>16</td>
<td>40</td>
</tr>
</tbody>
</table>

57
Bacteriuria

(A positive result is represented by a colony count of over 100,000 organisms per ml. in a pure culture whether symptomatic or asymptomatic.)

<table>
<thead>
<tr>
<th>Number of cases</th>
<th>577</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence per cent</td>
<td>7.6</td>
</tr>
<tr>
<td>Number of babies delivered</td>
<td>587</td>
</tr>
<tr>
<td>Stillbirths</td>
<td>10</td>
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<tr>
<td>Neonatal deaths</td>
<td>9</td>
</tr>
<tr>
<td>Perinatal deaths</td>
<td>19 Rate: 32/1,000</td>
</tr>
</tbody>
</table>

Comment: The incidence of bacteriuria dropped slightly in 1980 but was similar to that generally recorded over the years. Forty of the 587 (6.8 per cent) babies weighed less that 2.5 kilograms at birth. Fifty of the 577 mothers (8.6 per cent) developed anaemia, compared with the overall incidence of 4.9 per cent in the hospital population. None of the perinatal deaths was due to the urinary tract infection; in particular the diabetic coma noted in Case 43 was not related to a severe urinary tract infection.

PERINATAL DEATHS

Normal Infants


Post-mortem: Maceration.


Post-mortem: Not performed.


Post-mortem: Intra-uterine anoxia.


Post-mortem: Intra-uterine anoxia.

(27) B.19934. Para 8\(^{th}\) Age 32. S.E.3. Gestation 38 weeks. Booked at 21 weeks. Smoked 20 cigs./day. Admitted at 32 weeks with D.V.T. and started on Heparin. Discharged at 35...
weeks on Miniphep injections. Re-admitted at 38 weeks with abruptio placentae. F.H.H.
Emergency L.S.C.S. — fresh stillborn male infant weighing 3.17 kilograms. Retroplacental
clots.

Post-mortem: Intra-uterine anoxia secondary to placental abruption.

(38) B.52184. Para 1⁰ Age 37. S.E.4. Gestation uncertain. Previous pregnancy 1979:
at 7 24 weeks. Scan one month later suggested 35-36 weeks. Size clinically 28 weeks. Admitted,
B.P. 160/100. F.H.N.H. two days after admission. Spontaneous delivery of macerated female
infant weighing 1.81 kilograms.

Post-mortem: Refused.

(40) B.56225. Para 0⁰ Age 24. S.E.2. Gestation 40 weeks. First seen at 16 weeks gestation
when B.P. 140/90. Combined Antenatal Care Scheme. Normal antenatal record. Last seen at
40 weeks when B.P. recorded as 150/90. No albuminuria or oedema. Diminished F.M. but
F.H.H. Admission arranged but F.H.N.H. on admission in labour next day. Membranes
ruptured—thick meconium. Spontaneous delivery of fresh stillborn female infant weighing
3.03 kilograms. Cord loosely around neck.

Post-mortem: Intra-uterine anoxia.

(43) B.55759. Para 0⁰ Age 25. S.E.4. Gestation 28 weeks. First visit at 17 weeks gesta-
tion—glycosuria + 3. Further visit 6 weeks later—glycosuria + 2. Subsequent visits at 23 and
25 weeks showed no glycosuria but there was progressive weight loss. U.T.I. treated with
Nitrofuradantin. Admitted at 27 weeks with vomiting. Within 24 hours developed ketoacidosis
and coma. Insulin commenced and improvement was rapid. However, the fetal heart disap-
ppeared on the morning of the 28th week of pregnancy. Spontaneous labour and delivery of

Post-mortem: Intra-uterine anoxia.

(74) B.26676. Para 4⁰ Age 39. Gestation 40 weeks. First seen at antenatal clinic at 13
weeks gestation. Uneventful A.N.C. Seen in diabetic clinic as history of latent diabetes on
A.R.M. performed 3 days later. Nine hours later still not in established labour. Sudden fetal
distress and F.H.N.H. five minutes later. Spontaneous delivery of stillborn male infant
weighing 3.80 kilograms.

Post-mortem: Intra-partum asphyxia.

Regular attender at the antenatal clinic. Admitted at 29 weeks in labour with transverse lie.
L.S.C.S. Male infant weighing 1.55 kilograms born with Apgar scores of 8 and 9. Following
admission to S.C.B.U. the baby was intubated and given C.P.A.P. Clinical kyphoscoliosis
was confirmed with X-ray. Full septic work-up was carried out in view of P.R.O.M. Chest
X-ray was consistent with mild R.D.S. Antibiotics were commenced. Over the following 24
hours the baby became oedematous and at the age of 24 hours the infant collapsed. This was
in association with severe metabolic acidosis with subsequent attacks of severe apnoea. The
oedema increased and a pericardial effusion was aspirated. The baby had subsequent cardiac
arrests and died at a days of age. Lumoar puncture penormed at tnis time was oioosaainue.
All cultures were sterile apart from an eye swab which grew actinobacter species which was
sensitive to the antibiotic used.

Post-mortem: (i) Massive intraventricular haemorrhage.
   (in Haemopericardium.
   (ii) Pulmonary and renal haemorrhages.

**Congenital Malformations**

Cases 57, 85, 87,91,100,102,103,107,108.

**COMPARATIVE TABLE FOR 9 YEARS**

<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>Number of cases</td>
<td>413</td>
<td>616</td>
<td>465</td>
<td>507</td>
<td>413</td>
<td>420</td>
<td>606</td>
<td>694</td>
<td>577</td>
</tr>
<tr>
<td>Incidence %</td>
<td>5.3</td>
<td>8.1</td>
<td>6.1</td>
<td>7.0</td>
<td>5.9</td>
<td>6.0</td>
<td>8.1</td>
<td>9.2</td>
<td>7.6</td>
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<tr>
<td>Perinatal deaths</td>
<td>4</td>
<td>18</td>
<td>30</td>
<td>15</td>
<td>14</td>
<td>9</td>
<td>6</td>
<td>22</td>
<td>19</td>
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<tr>
<td>Mortality rate</td>
<td>9.5</td>
<td>28.6</td>
<td>61</td>
<td>29.3</td>
<td>34</td>
<td>21.3</td>
<td>9.69</td>
<td>31.1</td>
<td>32</td>
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</table>
**Abruptio Placentae**

<table>
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<tr>
<th>Number of cases</th>
<th>91</th>
</tr>
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<tbody>
<tr>
<td>Incidence per cent</td>
<td>1.18</td>
</tr>
<tr>
<td>Twins</td>
<td>3</td>
</tr>
<tr>
<td>Number of babies delivered</td>
<td>94</td>
</tr>
<tr>
<td>Stillbirths</td>
<td>15</td>
</tr>
<tr>
<td>Neonatal deaths</td>
<td>3</td>
</tr>
<tr>
<td>Perinatal deaths</td>
<td>18 Rate: 233/1,000</td>
</tr>
</tbody>
</table>

**Comment:** The incidence of abruptio placentae continued to increase, while the perinatal mortality changed little over the past five years. Two patients needed more than 5 units of blood. There was one case of maternal coagulation defect. Seven of the 18 perinatal deaths were considered to have avoidable factors (17, 18, 20, 22, 24, 71, 77). The 3 neonatal deaths were due to congenital malformations.

**PERINATAL DEATHS**

**Normal Infants**


*Post-mortem:* Not performed.


*Post-mortem:* Maceration.


*Post-mortem:* Not performed.


*Post-mortem:* Not performed.


*Post-mortem:* Refused.

Post-mortem: Maceration.


Post-mortem: Intra-uterine anoxia.


Post-mortem: Intra-uterine anoxia.


Post-mortem: Intra-uterine anoxia.


Post-mortem: Refused.


Post-mortem: Intra-uterine anoxia secondary to placental abruption.


Post-mortem: Intra-uterine anoxia.


Post-mortem: Intra-uterine anoxia.


Post-mortem: (i) Strangulation of the neck by a nuchal cord. (ii) Visceral congestion and oedema with pulmonary haemorrhage. (iii) Normal placenta.

(77) B.45670. Para 1+0. Age 23. S.E.3. Gestation 41 weeks. Normal antenatal record. Labour induced at 41 weeks. Some 2 hours later during strong contractions brisk vaginal haemorrhage with marked fetal bradycardia. Emergency L.S.C.S. Baby very flat, appeared exsanguinated, cord flacid. Presumptive diagnosis of ruptured vasa praevia. Female alive weighing 3.45 kilograms born with Apgar scores 1 and 0. While blood was being drawn from an 0 Neg. donor for immediate transfusion following delivery, the infant had a cardiac arrest. Within 4 hours of delivery the infant was transfused with 200ml. of fresh blood. Following this
and during the latter part of the transfusion the baby was noted to be oozing from puncture sites. This tendency was counteracted with protamine sulphate pending coagulation screen result. By 14 hours of age marked cerebral signs were noted and the baby remained anuric. Subsequently continued oozing and drop in haemoglobin led to exchange transfusion which was well tolerated with little improvement in oozing from puncture sites. The baby’s cerebral status deteriorated with seizures and at the age of 48 hours the coagulopathy was almost corrected, and urinary output also improved. The baby continued to require full ventilation and at the age of 3½ days had a cardiac arrest and was not resuscitated further.

Post-mortem: (i) Severe anoxic injury with cerebral haemorrhage.
(ii) Pulmonary haemorrhage and oedema.

**Congenital Malformations**

Cases 102, 103 and 106.

**COMPARATIVE TABLE FOR 10 YEARS**

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</thead>
<tbody>
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<td>52</td>
<td>62</td>
<td>68</td>
<td>40</td>
<td>56</td>
<td>45</td>
<td>76</td>
<td>84</td>
<td>91-</td>
</tr>
<tr>
<td>Incidence %</td>
<td>0.78</td>
<td>0.67</td>
<td>0.82</td>
<td>0.89</td>
<td>0.56</td>
<td>0.8</td>
<td>0.64</td>
<td>1.02</td>
<td>1.11</td>
<td>1.18</td>
</tr>
<tr>
<td>Perinatal deaths</td>
<td>17</td>
<td>19</td>
<td>19</td>
<td>26</td>
<td>12</td>
<td>18</td>
<td>9</td>
<td>13</td>
<td>14</td>
<td>18</td>
</tr>
<tr>
<td>Mortality rate</td>
<td>320</td>
<td>358</td>
<td>306.4</td>
<td>371</td>
<td>292</td>
<td>300</td>
<td>196</td>
<td>166</td>
<td>166</td>
<td>233</td>
</tr>
</tbody>
</table>
**Placenta Praevia**

Number of cases  43  
Incidence per cent  0.56  
Stillbirths  Nil  
Neonatal deaths  2  
Perinatal deaths  2  Rate: 46/1,000  

Method of delivery:  
L.S.C.S.  39  
Vaginal  4  

**Comment:** Despite the use of ultrasound in its diagnosis, the incidence of placenta praevia was one of the lowest in the last ten years. The two perinatal deaths were due to haemolytic disease (83) and multiple congenital abnormalities (108), rather than placenta praevia. Twenty patients required blood transfusion, with one patient requiring 11 units of blood. Ten of the live-born infants weighed less than 2.5 kilograms at birth and 8 survived.

**PERINATAL DEATHS**

*Normal infant*

(83) B. 18689. Para 2<sup>10</sup>. Age 28. S.E.3. Gestation 30 weeks. Booked at 10 weeks gestation. Blood group A Rh. positive. Previous pregnancies (i) full term delivery followed by primary P.P.H. and blood transfusion, (ii) full term normal delivery, no complications. Present pregnancy uneventful until 29 weeks gestation when patient complained of oedema of legs, feeling tired and being uncomfortable. B.P. 140/100, no proteinuria, hydramnios. Scan showed huge placenta, Grade I-II praevia. Infant appeared hydropic with ascites. Mother found to have anti-Kell antibodies, A.P.H. and emergency L.S.C.S. with delivery of female infant weighing 2.2 kilograms, born with Apgar scores of 1 and 1. The baby was markedly hydropic and required intubation. Hydrops fetalis secopndary to anti-Kell antibodies. Baby given 2 exchange transfusions and 3 top-up transfusions. Developed hypoglycaemia and hypercalceemia which were treated with peritoneal dialysis. Baby failed to respond and died at 7 days of age. The mother was given 6 units of blood during L.S.C.S. and developed puerperal pyrexia due to bacteroides but made a good recovery.

*Post-mortem:*  
(i) Massive intra cerebral haemorrhage.  
(ii) Massive extra medullary haematopoiesis related to haemolytic disease.  
(iii) Traumatic emphysema and subpleural bulla.

Case 108.

**COMPARATIVE TABLE FOR 10 YEARS**

<table>
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<tbody>
<tr>
<td>Number of cases</td>
<td>40</td>
<td>56</td>
<td>48</td>
<td>69</td>
<td>54</td>
<td>57</td>
<td>61</td>
<td>47</td>
<td>63</td>
<td>43</td>
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<tr>
<td>Incidence %</td>
<td>0.6</td>
<td>0.7</td>
<td>0.6</td>
<td>0.9</td>
<td>0.8</td>
<td>0.8</td>
<td>0.9</td>
<td>0.6</td>
<td>0.83</td>
<td>0.56</td>
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<tr>
<td>Perinatal deaths</td>
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<td>2</td>
<td>2</td>
<td>4</td>
<td>2</td>
<td>3</td>
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<td>5</td>
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<tr>
<td>Mortality rate</td>
<td>146</td>
<td>34</td>
<td>42</td>
<td>56</td>
<td>37</td>
<td>53</td>
<td>49</td>
<td>106</td>
<td>32</td>
<td>46</td>
</tr>
</tbody>
</table>
Antepartum Haemorrhage of Unknown Origin

Number of cases . . . . . . . . . 127
Incidence per cent . . . . . . . . . 1.64
Stillbirths . . . . . . . . . . . . . . 1
Neonatal deaths . . . . . . . . . . . . . Nil
Perinatal deaths. . . . . . . . . . . . . 1 Rate: 7.8/1,000

Method of delivery:

Caesarean section . . . . . . . . . . 23
Vaginal delivery. . . . . . . . . . . . . 104

Comment: The incidence of this problem remained unchanged for the last three years, while the number of perinatal deaths, in which antepartum haemorrhage appeared to be a contributory factor, decreased to only one (42). Seven of the 126 live-born infants weighed less than 2.5 kilograms and all of these survived. Thirteen of the 127 mothers delivered before 37 weeks gestation.

PERINATAL DEATH

Normal Infant


Post-mortem: Maceration.

COMPARATIVE TABLE FOR 10 YEARS

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Number of cases</td>
<td>94</td>
<td>193</td>
<td>167</td>
<td>188</td>
<td>145</td>
<td>136</td>
<td>158</td>
<td>124</td>
<td>129</td>
<td>127</td>
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<tr>
<td>Incidence %</td>
<td>1.4</td>
<td>2.5</td>
<td>2.2</td>
<td>2.5</td>
<td>2.0</td>
<td>1.9</td>
<td>2.2</td>
<td>1.6</td>
<td>1.7</td>
<td>1.6</td>
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<tr>
<td>Perinatal deaths</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>15</td>
<td>17</td>
<td>12</td>
<td>12</td>
<td>3</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Mortality rate</td>
<td>72</td>
<td>41</td>
<td>52</td>
<td>78</td>
<td>114</td>
<td>87</td>
<td>73</td>
<td>23</td>
<td>23</td>
<td>7.8</td>
</tr>
</tbody>
</table>
Department of Diagnostic Ultrasound

During 1980, 3,102 patients had 5,437 examinations. The number of patients scanned has increased only fractionally on the number seen in 1979, while the number of examinations increased by 4.4 per cent.

The indications for ultrasonic examinations were as follows:

- Gestational Age Assessment: 1,388
- Placental Localisation: 572
- Liquor volume assessment: 498
- Fetal abnormality: 495
- First trimester bleeding: 472
- Multiple pregnancy: 459
- Hypertensive disorders: 454
- Small for dates: 425
- Presentation: 220
- Bad obstetric history: 204
- Gynaecological tumours: 312
- Neonatal examinations: 191
- Pre shirodkar suture: 170
- Ectopic: 161
- Clinical/latent diabetes: 149
- Intra-uterine death: 126
- Localisation of I.U.C.D.: 112
- Gallstones: 74
- Missed abortion: 62
- Maternal kidneys: 57
- Hydrops: 35

The use of real-time scanning equipment has facilitated the assessment of fetal abnormality and the number of examinations performed in this category has increased by 95 per cent on the previous year. Furthermore, the mobility of this equipment has resulted in a 298 per cent increase in the number of neonatal examinations performed. Liquor volume assessment, as an adjunct to urinary oestriols and serial B.P.D. examinations in evaluating the at-risk fetus, increased six fold.

Dr. B. Stuart continued his research into fetal cardiovascular dynamics. Nurse S. Thornton, having attended a day release course organised by St. Vincent's Hospital, was successful in obtaining the Diploma of the College of Radiographers (Medical Ultrasound). To the best of our knowledge, she is the only midwife to hold this diploma.
Essential Hypertension

Pre-Eclampsia

Late Pregnancy Hypertension

**Essential Hypertension:** (A diastolic blood pressure reading of 90 mm. Hg. or greater on two or more occasions before the 20th week of pregnancy).

**Pre-Eclampsia:** (A diastolic blood pressure reading of 90 mm. Hg. or greater on two or more occasions after the 20th week of pregnancy accompanied by albuminuria or marked oedema, or both).

**Late Pregnancy Hypertension:** (A diastolic blood pressure reading of 90 mm. Hg. or greater on two or more occasions after the 20th week of pregnancy with no albuminuria or oedema).

<table>
<thead>
<tr>
<th></th>
<th>Essential Hypertension</th>
<th>Pre-Eclampsia</th>
<th>Late Pregnancy Hypertension</th>
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<tbody>
<tr>
<td>Number of cases</td>
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<td>229</td>
<td>317</td>
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<tr>
<td>Incidence percent</td>
<td>1.53</td>
<td>3.03</td>
<td>4.17</td>
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<tr>
<td>Primigravidae</td>
<td>25</td>
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<tr>
<td>Multigravidae</td>
<td>92</td>
<td>125</td>
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<td>Twins</td>
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<td>8</td>
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</tr>
<tr>
<td>Number of babies</td>
<td>120</td>
<td>237</td>
<td>322</td>
</tr>
<tr>
<td>Stillbirths</td>
<td>3</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Neonatal deaths</td>
<td>1</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Perinatal deaths</td>
<td>4(33/1,000)</td>
<td>6 (26/1,000)</td>
<td>2(6.3/1,000)</td>
</tr>
</tbody>
</table>

**Method of delivery:**

<table>
<thead>
<tr>
<th></th>
<th>Essential Hypertension</th>
<th>Pre-Eclampsia</th>
<th>Late Pregnancy Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous</td>
<td>88</td>
<td>162</td>
<td>280</td>
</tr>
<tr>
<td>Forceps</td>
<td>6</td>
<td>34</td>
<td>20</td>
</tr>
<tr>
<td>C.S.</td>
<td>23</td>
<td>33</td>
<td>14</td>
</tr>
<tr>
<td>Vacuum</td>
<td>1</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Breech</td>
<td>2</td>
<td>3</td>
<td>6</td>
</tr>
</tbody>
</table>

**Comment:** The incidence of the hypertensive disorders of pregnancy has not continued to decline as was the recent trend. The number of cases of essential hypertension and pre-eclampsia has increased while the number of cases of late pregnancy hypertension has fallen. However the perinatal mortality for each group has improved in comparison to the 1979 figures.

In the Essential Hypertension Group 2 of the 3 stillbirths (37 and 40) were small for gestational age and pre-term. Two patients (37 and 40) had avoidable factors: Case 37 was deemed avoidable because no placental function tests were performed even though the patient attended regularly and had a bad obstetric history; Case 40 should have been admitted to hospital sooner for detailed assessment. There were no avoidable factors in Case 44 who was a Grade F diabetic in whom earlier delivery was considered but rejected as the fetus was so dysmature. One neonatal death was due to a congenital malformation.

In the Pre-Eclampsia group there were 4 stillbirths (20, 21, 38 and 39); these do not include 1 patient (36) who developed eclampsia without any recorded evidence of pre-eclampsia. Avoidable factors were present in 3 of
the 4 stillbirths: in Case 30 earlier delivery might have produced a better outcome; Case 38 should have booked herself for confinement sooner; the hypertension was not acted upon in Case 39 for some time prior to admission to hospital. Two neonatal deaths (83 and 102) were unavoidable.

The perinatal mortality in Late Pregnancy Hypertension was low, as expected. The only still birth could have been avoided by better intra-partum management (75). The neonatal death was due to a congenital abnormality (100).

PERINATAL DEATHS

Normal Infants


Post-mortem: Not performed.


Post-mortem: Refused.


Post-mortem: Refused.


Post-mortem: Refused.


Post-mortem: Refused.


Post-mortem: Intra-uterine anoxia.

(44) B.1969. Para 3**. Age 30. S.E.3. Diabetic patient for 21 years. Gestation 30 weeks. Booked at 6 weeks gestation, which was confirmed by ultrasound scan. Insulin levels were controlled and regular attendant. However, noted to have retarded intrauterine growth from 24 weeks gestation onwards. Grossly dysmature and I.U.D. at 30 weeks. At this time the uterine fundus was only equal to that of a 24 week pregnancy. Spontaneous labour and delivery one week later of female macerated infant weighing 850 grams.

Post-mortem: Refused.

Post-mortem: Intra-partum asphyxia (cord prolapse).

(83) B. 18689. Para 2”. Age 28. S.E.3. Gestation 30 weeks. Booked at 10 weeks gestation. Blood group A Rh. positive. Previous pregnancies (i) full term delivery followed by primary P.P.H. and blood transfusion, (ii) full term normal delivery, no complications. Present pregnancy uneventful until 29 weeks gestation when patient complained of oedema of legs, feeling tired and being uncomfortable. B.P. 140/100, no proteinuria, hydramnios. Scan showed huge placenta. Grade I-II praevia. Infant appeared hydropic with ascites. Mother found to have anti-Kell antibodies. A.P.H. and emergency L.S.C.S. with delivery of female infant weighing 2.2 kilograms, born with Apgar scores of 1 and 1. The baby was markedly hydropic and required intubation. Hydrops foetalis secondary to anti-Kell antibodies. Baby given 2 exchange transfusions and 3 top-up transfusions. Developed hypoglycaemia and hypercalcaemia which were treated with peritoneal dialysis. Baby failed to respond and died at 7 days of age. The mother was given 6 units of blood during L.S.C.S. and developed puerperal pyrexia due to bacteriodes but made a good recovery.

Post-mortem: (i) Massive intra cerebral haemorrhage.
(ii) Massive extra medullary haemopoiesis related to haemolytic disease.
(iii) Traumatic emphysema and subpleural bulla.

Congenital Malformations

Cases 86, 100 and 102.
Eclampsia

Number of cases ............. 7
Incidence per cent ............. 0.09
Stillbirths .................. 2
Neonatal deaths ............... 0
Perinatal deaths ............... 2 Rate: 285/1,000

Method of delivery:
Spontaneous .................... 4
Caesarean Section .............. 3

Comment: Seven patients had eclamptic fits during 1980. Six of these patients had the fit before or during labour. One of the two perinatal deaths (36) was directly related to the convolution but the other death (20) had occurred prior to the eclamptic fit.

PERINATAL DEATHS

Normal Infants


Post-mortem: Not performed.


Post-mortem: Refused.

COMPARATIVE TABLE FOR 10 YEARS

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of cases</td>
<td>4</td>
<td>8</td>
<td>3</td>
<td>6</td>
<td>3</td>
<td>10</td>
<td>8</td>
<td>7</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>Incidence %</td>
<td>0.06</td>
<td>0.10</td>
<td>0.03</td>
<td>0.07</td>
<td>0.04</td>
<td>0.14</td>
<td>0.11</td>
<td>0.09</td>
<td>0.05</td>
<td>0.09</td>
</tr>
<tr>
<td>Perinatal deaths</td>
<td>1</td>
<td>Nil</td>
<td>1</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>Nil</td>
<td>1</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Mortality rate</td>
<td>250</td>
<td>Nil</td>
<td>333</td>
<td>166</td>
<td>333</td>
<td>200</td>
<td>125</td>
<td>Nil</td>
<td>250</td>
<td>285</td>
</tr>
</tbody>
</table>
Multiple Pregnancy

Number of twins . . . . . . . 115
Number of triplets . . . . . . . 1
Number of cases . . . . . . . . . . . . 116
Incidence per cent . . . . . . . . . . . . 1.5%
Number of babies delivered ... 233
Stillbirths . . . . . . . . . 2
Neonatal deaths . . . . . . . . . . . . 5
Perinatal deaths . . . . . . . . . . . . 7 Rate: 30/1,000

Comment: This is the highest number of multiple pregnancies recorded in the Coombe hospital in any one year. The perinatal mortality showed a reduction from the figure recorded in 1979 and the trend of perinatal mortality over the past ten years is shown in the ten year summary. 37 of the 116 mothers (32%) delivered prior to 37 weeks gestation. The two stillbirths (41 and 42) were both macerated and intra-uterine death had occurred many weeks prior to delivery. Both these deaths were deemed unavoidable. Two of the five neonatal deaths (99 and 101) were associated with gross congenital abnormalities while the other three all weighed less than 2500 g., two weighing less than 1500 g. One death (84) might have been avoided by earlier induction of labour and it is feasible that administration of steroids, prior to amniocentesis, might have provided a better outcome in case 81.

PERINATAL DEATHS

Normal Infants


Post-mortem: Maceration.

(42) B.20963. Para 5+0. Age 34. S.E.5. Gestation 40 weeks. Booked at 13 weeks gestation. Second trimester bleeding at 24 weeks. Discharged after 10 days. Seen frequently in clinic. Last visit at 40 weeks. Admitted 6 days later. Cervix fully dilated. Thick meconium staining. Spontaneous delivery of twins, 1st twin female alive in good condition, cord x4 around neck. Weight 2.64 kilograms. Undiagnosed second twin delivered in sac complete with placenta, male macerated, weight 907 grams.

Post-mortem: Maceration.

(79 and 81) B. 19241. Para 3+0. Age 34. S.E.2. Gestation 28 weeks. Booked at 13 weeks gestation. Multiple pregnancy diagnosed on scan. Acute hydramnios at 28 weeks and admitted. Trans-abdominal amniotomy but labour occurred. Forceps delivery of live male infants. Twin I, birth weight 1.25 kilograms. Apgar scores 0 and 3. The infant was intubated on delivery and given I.P.P.V. with slow improvement. Blood gases revealed severe mixed respiratory metabolic acidosis with adequate oxygenation. The baby's fontanelle was rather full and sutures splayed and C.S.F. was uniformly bloodstained. The baby died at 10 hours of age.
Twin II, clinically this twin was severely undernourished with weight lying on the 10th centile. The baby responded well to resuscitation having been flat at delivery and was ventilated. At 4 hours of age the baby’s colour disimproved and the ductus was noted to have opened. The baby shortly thereafter developed bilateral pneumothoraces which were drained with little improvement in the baby’s condition, which was associated with marked bradycardia. The baby died at 24 hours of age.

Post-mortem: Twin I — Large intraventricular haemorrhage extending through the mid brain, cerebellum and into sub-arachnoid space. Total atelectasis of both lungs; Twin II — Bilateral pulmonary atelectasis with early changes of hyaline membrane disease. The gastrointestinal tract was almost devoid of meconium.

(84) B. 10470. Para 3. Age 30. S.E.4. Gestation 41 weeks. Booked at 17 weeks gestation. Scan at 24 weeks because of gross hydramnios revealed multiple pregnancy. Two fetuses noted. Spontaneous onset of labour at 41 weeks with delivery of 3 infants. The first and second were normal females while the third was an acardiac monster. Triplet I: assisted breech delivery female infant weighing 2.31 kilograms, born with Apgar scores of 3 and 9. Meconium aspirated from trachea at delivery and bronchial lavage was carried out. Small for dates. Mild cerebral irritability with depressed anterior fontanelle. P.C.V. 75 per cent. Initial fluid restriction because of cerebral irritability which did not progress. On second day feeds were not tolerated and I.V. fluids were given. General condition less good. Full septic work-up performed and antibiotics commenced. Abdominal X-ray suggestive of necrotising enterocolitis with subsequent perforation. Following surgical consultation conservative management continued but later further deterioration and ventilatory support given. Urinary output dropped, with evidence of abdominal condition being less well localised. When baby stabilised she was transferred to another hospital for surgical intervention. However, operation was withheld and the abdomen drained. Subsequently, renal status deteriorated further, associated with cardiac arrhythmia secondary to hypercalcaemia which was corrected with peritoneal dialysis, using the drainage tube with restoration of urinary output. However, baby had sudden unexplained cardiac arrest on the 7th day. Triplet III: Acardiac monster.

Post-mortem: (i) Peritonitis mainly localised to site of perforation. (ii) Left renal papillary necrosis. (iii) Necrotising enterocolitis; (iv) Small left cerebral ventricular haemorrhage.

Congenital Malformations

Cases 99 and 101.
Breech Delivery

Number of mothers: 176
In incidence per cent: 2.28
Twins: 54
Number of babies delivered: 180
Stillbirths: 7
Neonatal deaths: 5
Perinatal deaths: 12 Rate: 66/1,000

Singleton Breech Delivery

Primigravidae:
Number of mothers: 29
In incidence per cent: 1.3
Stillbirths: 2
Neonatal deaths: 1
Perinatal deaths: 3 Rate: 103/1,000

Multigravidae:
Number of cases: 93
In incidence per cent: 1.7
Stillbirths: 5
Neonatal deaths: 3
Perinatal deaths: Rate: 85/1,000

Comment: The number of vaginal breech deliveries increased slightly during 1980, but this increase was entirely due to an increased incidence of breech presentation in association with multiple pregnancy. The format of presentation has been altered slightly in this year’s report in that the outcome of singleton breech delivery in both primigravidae and multigravidae is given separately, together with an analysis of perinatal deaths in singleton breech deliveries in relation to term or pre-term delivery.
Term Breech:
Table 1 includes all singleton vaginal breech deliveries weighing more than 2.5 kilograms. None of the 7 perinatal deaths were due to complications of labour or delivery: Cases 6 and 14 had died prior to the onset of labour and the other 5 perinatal deaths were all due to congenital malformations of the central nervous system.

Pre-term Breech:
Twenty-two singleton pre-term vaginal breech deliveries took place during 1980 and 4 of these babies died. However, as with the term breech deliveries none of the perinatal deaths were due to factors associated with labour or delivery. The two stillbirths (17 and 18) were associated with antepartum haemorrhage and intra-uterine death occurred before the onset of premature labour. Both neonatal deaths (92 and 97) were due to fetal abnormalities.

<table>
<thead>
<tr>
<th>No.</th>
<th>S.B.</th>
<th>N.N.D.</th>
<th>F/A</th>
<th>I.U.D.</th>
<th>P.N.M.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Term:</td>
<td>100</td>
<td>5</td>
<td>2*</td>
<td>5</td>
<td>2</td>
</tr>
<tr>
<td>Pre-term:</td>
<td>22</td>
<td>2</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Total:</td>
<td>122</td>
<td>7</td>
<td>4</td>
<td>7</td>
<td>4</td>
</tr>
</tbody>
</table>

TABLE 1
ANALYSIS OF PERINATAL DEATHS FOLLOWING SINGLETON BREECH DELIVERY

PERINATAL DEATHS
Normal Infants


Post-mortem: Maceration.


Post-mortem: Maceration.


Post-mortem: Not performed.


Post-mortem: Maceration.

(84) B.10470. Para 3⁺⁰. Age 30. S.E.4. Gestation 41 weeks. Booked at 17 weeks gestation. Scan at 24 weeks because of gross hydramnios revealed multiple pregnancy. Two fetuses noted. Spontaneous onset of labour at 41 weeks with delivery of 3 infants. The first and second were normal females while the third was an acardiac monster. Triplet 1: assisted breech delivery female infant weighing 2.31 kilograms, born with Apgar scores of 3 and 9. Meconium aspirated from trachea at delivery and bronchial lavage was carried out. Small for dates. Mild cerebral irritability with depressed anterior fontanelle. P.C.V. 75%. Initial fluid restriction because of cerebral irritability which did not progress. On second day feeds were not tolerated...
and I.V. fluids were given. General condition less good. Full septic work-up performed and antibiotics commenced. Abdominal X-ray suggestive of necrotising enterocolitis with subsequent perforation. Following surgical consultation conservative management continued but later further deterioration and ventilatory support given. Urinary output dropped, with evidence of abdominal condition being less well localised. When baby stabilised she was transferred to another hospital for surgical intervention. However, operation was withheld and the abdomen drained. Subsequently, renal status deteriorated further, associated with cardiac arrhythmia secondary to hypercalcaemia which was corrected with peritoneal dialysis, using the drainage tube with restoration of urinary output. However, baby had sudden unexplained cardiac arrest on the 7th day.

**Post-mortem:**

(i) Peritonitis mainly localised to site of perforation.
(ii) Left renal papillary necrosis.
(iii) Necrotising enterocolitis.
(iv) Small left cerebral ventricular haemorrhage.

**Congenital Malformations**

Cases: 66, 67, 69, 88, 90, 92 and 97.
External Version

Number of cases . . . . . . . . . . . 54
Incidence per cent . . . . . . . . . . . 0.7
Number of babies delivered . . . . . 54
Stillbirths . . . . . . . . . . . . . . . . 3
Neonatal deaths . . . . . . . . . . . . . . . . . . . . . . 1
Perinatal deaths . . . . . . . . . . . . . . . . . . . 4 Rate: 74/1,000

Comment: The incidence of external version dropped in comparison to previous years. The presentation remained cephalic in 49 of the 54 cases but reverted to breech on 5 occasions. One stillbirth (22) was directly due to external version which was performed without any general anaesthetic.

PRESENTATION AT DELIVERY

<table>
<thead>
<tr>
<th>Cephalic</th>
<th>Breech</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaginal Delivery</td>
<td>L.S.C.S.</td>
</tr>
<tr>
<td>46</td>
<td>3</td>
</tr>
</tbody>
</table>

PERINATAL DEATHS

Normal Infants


Post-mortem: Maceration.


Post-mortem: Refused.

Congenital Malformations

Cases 56 and 89.
Amniocentesis

Total Patients 45
Total amniocenteses 150

Indications for amniocentesis:
- Rhesus iso-immunisation 130
- L/S ratio 20

While the number of amniocenteses performed in the management of rhesus iso-immunisation has remained static, there was a considerable fall in the number of amniocenteses performed to assess lung maturity. All cases had ultrasonic localisation of the placenta prior to amniocentesis and in many instances the procedure was performed under ultrasonic guidance. No complication is known to have ensued from amniocentesis.
Varicose Vein Clinic

During the year 112 patients were seen at the clinic with an average of 3.1 visits per patient. These figures reveal a fall in the number of patients seen compared with previous years. Some 36 patients were treated by compression sclerotherapy while 7 patients were treated primarily for superficial thrombophlebitis. One patient was admitted from the clinic with deep vein thrombosis. Three patients were referred for surgical treatment postnatally.

<table>
<thead>
<tr>
<th>Age</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 20 years</td>
<td>0</td>
</tr>
<tr>
<td>20-24</td>
<td>5</td>
</tr>
<tr>
<td>25-29</td>
<td>33</td>
</tr>
<tr>
<td>30-34</td>
<td>47</td>
</tr>
<tr>
<td>35-39</td>
<td>18</td>
</tr>
<tr>
<td>40 +</td>
<td>9</td>
</tr>
</tbody>
</table>

Parity (recorded in 107)

<table>
<thead>
<tr>
<th>Parity</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>1</td>
<td>18</td>
</tr>
<tr>
<td>2</td>
<td>22</td>
</tr>
<tr>
<td>3</td>
<td>28</td>
</tr>
<tr>
<td>4</td>
<td>18</td>
</tr>
<tr>
<td>5+</td>
<td>16</td>
</tr>
</tbody>
</table>

The attendance at the Varicose Vein Clinic is dependant on referral from the antenatal clinics and the figures presented in no way reflect an accurate measurement of the problem. Several factors are involved in the poor referral rate but a higher referral rate is essential if the problem is to be adequately treated.
### Induction of Labour

<table>
<thead>
<tr>
<th>Number of cases</th>
<th>Incidence per cent</th>
<th>Multiple pregnancy</th>
<th>Number of babies delivered</th>
<th>Stillbirths</th>
<th>Neonatal deaths</th>
<th>Perinatal deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>1,593</td>
<td>21</td>
<td>29</td>
<td>1,622</td>
<td>19</td>
<td>3</td>
<td>22</td>
</tr>
</tbody>
</table>

Rate 13.6/1,000

### TABLE I—Parity

<table>
<thead>
<tr>
<th>Prim.</th>
<th>Mult.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cases</td>
<td>505</td>
</tr>
<tr>
<td>Incidence per cent</td>
<td>22.8</td>
</tr>
<tr>
<td>Total babies</td>
<td>514</td>
</tr>
</tbody>
</table>

### TABLE II—Indications for Induction

<table>
<thead>
<tr>
<th>Prim.</th>
<th>Mult.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Past 40 weeks gestation</td>
<td>293 (13.2%)</td>
</tr>
<tr>
<td>P. E.T./Hypertension</td>
<td>119 (5.4%)</td>
</tr>
<tr>
<td>Retarded intrauterine growth</td>
<td>18</td>
</tr>
<tr>
<td>Distance from hospital ...</td>
<td>5</td>
</tr>
<tr>
<td>Diabetes/Latent Diabetes</td>
<td>6</td>
</tr>
<tr>
<td>Poor weight gain</td>
<td>5</td>
</tr>
<tr>
<td>Poor obstetrical history ...</td>
<td></td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>9</td>
</tr>
<tr>
<td>Incipient labour</td>
<td>15</td>
</tr>
<tr>
<td>Antibodies</td>
<td></td>
</tr>
<tr>
<td>Social ...</td>
<td>2</td>
</tr>
<tr>
<td>Maternal Age ...</td>
<td>3</td>
</tr>
<tr>
<td>1st or 2nd trimester bleeding</td>
<td>2</td>
</tr>
<tr>
<td>Multiple pregnancy</td>
<td>3</td>
</tr>
<tr>
<td>Miscellaneous ...</td>
<td>25</td>
</tr>
<tr>
<td>Total (mothers)</td>
<td>505</td>
</tr>
</tbody>
</table>

Figures in brackets refer to the incidence in all patients of that parity.

### TABLE III—Mode of Delivery

<table>
<thead>
<tr>
<th>Prim.</th>
<th>Mult.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous vertex</td>
<td>347 (67.7%)</td>
</tr>
<tr>
<td>Forceps/Vacuum</td>
<td>128 (25.0%)</td>
</tr>
<tr>
<td>Breech ...</td>
<td>4</td>
</tr>
<tr>
<td>Caesarean section</td>
<td>35* (6.8%)</td>
</tr>
<tr>
<td>Total (babies) ...</td>
<td>514</td>
</tr>
</tbody>
</table>

*Two primigravid patients delivered by section had twins. One Para 2 twin spontaneously, but underwent abdominal delivery for the second.
### TABLE IV—CAESAREAN SECTION

<table>
<thead>
<tr>
<th>Indications for Induction</th>
<th>Prim.</th>
<th>Mult.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Past 40 weeks gestation</td>
<td>21 (7.0%)</td>
<td>21 (3.8%)</td>
</tr>
<tr>
<td>P. E.T./Hypertension</td>
<td>7 (5.9%)</td>
<td>3 (1.8%)</td>
</tr>
<tr>
<td>Retarded intra-uterine growth</td>
<td>1 (5.6%)</td>
<td>2 (6.0%)</td>
</tr>
<tr>
<td>Diabetes/Latent Diabetes</td>
<td></td>
<td>2 (6.9%)</td>
</tr>
<tr>
<td>Rhesus incompatibility</td>
<td></td>
<td>3 (17.6%)</td>
</tr>
<tr>
<td>Failing oestriols</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Reduced liquor</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td></td>
<td>3 (12.0%)</td>
</tr>
<tr>
<td>Previous stillbirth</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Incipient labour</td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Multiple pregnancy</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Previous infertility</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>Reduced liquor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reduced liquor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total (babies)</td>
<td>35*</td>
<td>38</td>
</tr>
</tbody>
</table>

Figures in brackets show the incidence of Caesarean section in patients induced for that particular indication.

*This figure includes 2 mothers, both of whom had twins, so that the total number of primigravid mothers sectioned was 33.

**Comment:** The incidence of induction of labour fell by 2 per cent when compared with 1979. This fall was entirely due to a reduction in the number of multiparous patients induced: the incidence of primigravid induction remained the same.

The indications for induction were similar to previous years. However, there was a reduction of 1.4 per cent in the number of primigravidae induced for uncomplicated postmaturity and of 2.5 per cent in multiparous patients started off for this reason. The title "Miscellaneous" unfortunately hides the fact that in 18 cases no indication was coded for the induction, and thorough scrutiny of the patients' notes failed to reveal one. It seems incredible that a procedure as serious as induction of labour should be undertaken without any obvious reason.

Another worrying factor about 1980 was the increase in the operative delivery rate amongst induced patients. No less than 6.8 per cent of primigravidae and 3.4 per cent of multiparous patients (Table III) ultimately required delivery by Caesarean section. The increase was unfortunately due to more sections being required for patients induced for uncomplicated post-maturity and, although the rates were no higher than in 1978 it is a pity to see a reversal of the favourable trend shown in 1979. While one can accept that a section may be required in some patients induced for complications such as rhesus incompatibility and retarded intra-uterine growth, one is less willing to accept it for simple post-maturity. The issue is not so clear however, as while the opponents of induction may blame the induction itself for the eventual section, its proponents may well take the view that had the induction been undertaken earlier the section might not have been required. Perhaps one answer would be never to induce anyone for uncomplicated post-maturity without first ripening the cervix by means of intra-vaginal prostaglandin.

Of the 19 stillbirths, 18 were either dead before the induction was undertaken or suffered from serious congenital malformation (Cases 4, 12, 19, 20, 22, 25, 30, 31, 33, 34, 36, 37, 48, 53, 55, 56, 63 and 66). Case 74 was an avoidable intra-partum death in whom a Type II deceleration was not noted and acted upon.

Of the neonatal deaths, Case 96 died from congenital malformations. Case 77 was induced at 11 days past term for uncomplicated post-maturity: a sudden gush of blood accompanied by fetal bradycardia was diagnosed as abruptio placentae; this diagnosis was proved incorrect at Caesarean Section where there was no evidence of retro-placental clot, but the baby was
pale and hypotonic. It is not possible to tell whether the fetal vessel was
torn at induction or ruptured later, although the latter seems the more
likely.

The mother in Case 78 was 5’ 10” tall, had already delivered a baby
weighing 4.4 kilograms and was absolutely certain of her dates. At term
+ 14 days the cephalic presentation was 4/5ths palpable and an intravenous
prostaglandin infusion was started. Six hours later the cervix was 4 cms.
dilated and hind water rupture produced clear liquor. Two hours later the
cervix was 8 cms. and the forewaters were broken with no evidence of
meconium. Some 2 hours later the cervix was fully dilated but the head had
not yet engaged and was in the occipito-posterior position. No attempt was
made at vaginal delivery. Unfortunately, the clear liquor may have caused
a false sense of security in that continuous monitoring of the fetus was not
undertaken. This patient should probably have been delivered by elective
Caesarean section.

PERINATAL DEATHS

Normal Infants

(74) B.26676. Para 4**. Age 39. Gestation 40 weeks. First seen at antenatal clinic at 13
weeks gestation. Uneventful A.N.C. Seen in diabetic clinic as history of latent diabetes on
A.R.M. performed 3 days later. Nine hours later still not in established labour. Sudden fetal
distress and F.H.N.H. five minutes later. Spontaneous delivery of stillborn male infant
weighing 3.80 kilograms.

Post-mortem: Intra-partum asphyxia.

Labour induced at 41 weeks. Some 2 hours later during strong contractions brisk vaginal
haemorrhage with marked fetal bradycardia. Emergency L.S.C.S. Baby very flat, appeared
exsanguinated, cord flacid. Presumptive diagnosis of ruptured vasa praevia. Female alive
weighing 3.45 kilograms born with Apgar scores 1 and 0. While blood was being drawn from
an 0 Neg. donor for immediate transfusion following delivery, the infant had a cardiac arrest.
Within 4 hours of delivery the infant was transfused with 200 ml. of fresh blood. Following
this and during the latter part of the transfusion the baby was noted to be oozing from puncture
sites. This tendency was counteracted with protamine sulphate pending coagulation screen
result. By 14 hours of age marked cerebral signs were noted and the baby remained anemic.
Subsequently continued oozing and drop in haemoglobin led to exchange transfusion which
was well tolerated with little improvement in oozing from puncture sites. The baby's cerebral
status deteriorated with seizures and at the age of 48 hours the coagulopathy was almost
corrected, and urinary output also improved. The baby continued to require full ventilation
and at the age of 3| days had a cardiac arrest and was not resuscitated further.

Post-mortem: (i) Severe anoxic injury with cerebral haemorrhage.
(ii) Pulmonary haemorrhage and oedema.

Uneventful A.N.C. except for anaemia. Admitted at 38 weeks gestation because of oblique
breech. By 42 weeks cephalic presentation. Stable but high. Clinically big baby. PGE
induction. Head did not descend in pelvis in second stage of labour and L.S.C.S. performed.
Male infant weighing 4.71 kilograms, born with Apgar scores of 5 and 4. Baby was admitted
to S.C.B.U. and following resuscitation the endotracheal tube was removed at 25 mins. Initial
pH showed severe mixed metabolic respiratory acidosis, with a pH of 6.8. Following extubation
the baby had poor respiratory effort with extreme hypotonia and staring associated with a fine
tremor of face and feet. Treatment with Mannitol and Dexamethasone was commenced and
the baby was slow to pass urine. At 24 hours of age the baby was noted to be having increasing
respiratory distress associated with pneumonia. The baby was further supported with vential-
lation with some improvement. Neurological examination continued to be very abnormal. At
46 hours of age the baby was markedly hypoxic and despite every effort to improve ventilation
the baby died.

Post-mortem: (i) Severe anoxic changes in the brain.
(ii) Bilateral bronchial pneumonia.
(iii) Pneumococcus was grown from the endotracheal tube and eye swab.

The following normal infants had died before induction took place: cases 4, 12, 19, 20, 22,
25, 30, 31, 33, 34, 36, 37 and 48.

Congenital Malformations

Cases 53, 55, 56, 63, 66, and 96.
Management of Labour

This chapter refers to the management of all patients who went into spontaneous labour after 28 weeks gestation. Chapters dealing with transverse and oblique lie in labour, persistent posterior position of the occiput and transverse arrest, disproportion, as well as face and brow presentation, which in previous years were described separately, have also been included even when labour was induced.

The policy of active management of labour was maintained during 1980, and spontaneous labour was accelerated by A.R.M., oxytocin infusion, or both procedures, on 2,353 occasions (41 per cent). Continuous fetal heart rate monitoring was used in the management of 1,277 (17 per cent) patients: 1,097 (86 per cent) delivered vaginally and 180 (14 per cent) were delivered by Caesarean Section.

Seventy-four of the monitored patients had a Caesarean Section performed because of fetal distress, an incidence of 5.7 per cent amongst the monitored patients. Despite the increased use of continuous fetal heart rate monitoring during labour, the incidence of Caesarean Section for fetal distress actually decreased during 1980, possibly as a result of an increased use of fetal scalp pH measurements. Epidural anaesthesia was administered during labour to 401 patients: 202 (50%) delivered spontaneously; 148 (38%) were delivered with the aid of forceps; 26 (6%) were delivered by Caesarean Section; 25 (6%) were delivered by the aid of vacuum extraction. The fetal heart rate was monitored continuously in 254 (63%) of the 401 cases.

Prolonged labour

(Labour lasting longer than 12 hours)

<table>
<thead>
<tr>
<th></th>
<th>Number of cases</th>
<th>Incidence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primigravidae</td>
<td>137</td>
<td>6.1%</td>
</tr>
<tr>
<td>Multiparae</td>
<td>230</td>
<td>4.2%</td>
</tr>
<tr>
<td>Labour accelerated</td>
<td>294</td>
<td>80%</td>
</tr>
</tbody>
</table>

Comment: The duration of labour exceeded 12 hours in 367 patients who started labour spontaneously. In addition, the induction/delivery interval was longer than 12 hours in 187 of the 1,593 patients in whom labour had been induced. There were 6 perinatal deaths: 3 were due to congenital malformations (66, 67 and 96); in 2 instances intra uterine death occurred prior to the onset of labour (12 and 48); 1 multiparous (74) was an avoidable intra partum death in whom a Type II deceleration was not noted and acted upon.
Transverse and oblique lie in labour

Number of cases: 26
Incidence per cent: 0.34

Comment: Five of the 26 cases were associated with labour before the end of the 37th completed week, four with a parity of 5 or more and 2 with multiple pregnancy. In 3 cases there was an associated prolapse or presentation of the cord. The lie was not responsible for either of the perinatal deaths (80 and 91).

Persistent posterior position of the occuput and transverse arrest

Number of cases: 170
Incidence per cent: 2.2
Deep transverse arrest: 51
Persistent occipito-posterior: 119

Comment: The incidence of these mal-presentations increased from 1 per cent to 2.2 per cent between 1979 and 1980. This was probably related to the increased use of epidural analgesia; 29 per cent of cases were associated with epidural analgesia compared with only 13 per cent in 1979. Forty of the 170 cases had labour induced, and labour was prolonged in 43 instances. Fifty per cent of cases were delivered by Neville Barnes Forceps, 19 per cent by vacuum extraction, and 6 per cent by Caesarean section. Four babies weighed less than 2.5 kilograms at 37 and 38 weeks respectively. None of the perinatal deaths (15, 82, 99 and 106) was due to the malposition.

Disproportion

Number of cases: 25
Incidence per cent: 0.33
Perinatal death: 1
Primigravidae: 15
Multigravidae: 10
Management:
Elective L.S.C.S.: 14
Trial of labour: 11

Comment: There were 15 new cases of disproportion during 1980 and 11 of these cases were treated by trial of labour. The one perinatal death (78) would have been more appropriately managed by elective Caesarean section.

Face and brow presentation

Face:
Number of cases: 9
Incidence per cent: 0.1
Stillbirths: 3
Neonatal deaths: Nil
Perinatal deaths: 3
Method of delivery:
- Spontaneous
- Forceps

Brow:
- Number of cases: 2
- Incidence per cent: 0.02
- Perinatal deaths: Nil

Comment: The 9 cases of face presentation included 3 anencephalic infants who were all stillborn (54, 59 and 68). Two of the remaining 6 infants were delivered with the aid of forceps, while four delivered spontaneously. The two brow presentations were delivered by Caesarean section. The malpresentation was not responsible for any of the perinatal deaths.

PERINATAL DEATHS

Normal Infants


Post-mortem: Maceration.

(15) B. 49146. Para 0\textsuperscript{1}. Age 29. S.E.I. Gestation 41 weeks. Irregular cycle. F.M. ceased at 41 weeks. Spontaneous labour and forceps delivery of macerated male infant weighing 3.06 kilograms.


Post-mortem: Intra-partum asphyxia.

(78) B.41239. Para 1\textsuperscript{0}. Age 30. S.E.2. Gestation 42 weeks. Booked at 8 weeks gestation. Uneventful A.N.C. except for anaemia. Admitted at 38 weeks gestation because of oblique breech. By 42 weeks cephalic presentation. Stable but high. Clinically big baby. PGE induction. Head did not descend in pelvis in second stage of labour and L.h.C.S. performed. Male infant weighing 4.71 kilograms, born with Apgar scores of 5 and 4. Baby was admitted to S.C.B.U. and following resuscitation the endotracheal tube was removed at 25 mins. Initial pH showed severe mixed metabolic respiratory acidosis, with a pH of 6.8. Following extubation the baby had poor respiratory effort with extreme hypotonia and staring associated with a fine tremor of face and feet. Treatment with Mannitol and Dexamethasone was commenced and the baby was slow to pass urine. At 24 hrs. of age the baby was noted to be having...
increasing respiratory distress associated with pneumonia. The baby was further supported with ventilation with some improvement. Neurological examination continued to be very abnormal. At 46 hours of age the baby was markedly hypoxic and despite every effort to improve ventilation the baby died.

**Post-mortem:**
(i) Severe anoxic changes in the brain.
(ii) Bilateral bronchial pneumonia.
(iii) Pneumococcus was grown from the endotracheal tube and eye swab.

(80) B.25055. Para 2\textsuperscript{a}. Age 34. S.E.3. Gestation 29 weeks. Booked at 10 weeks gestation. Regular attender at the antenatal clinic. Admitted at 29 weeks in labour with transverse lie. L.Ś.C.S. Male infant weighing 1.55 kilograms born with Apgar scores of 8 and 9. Following admission to S.C.B.U. the baby was intubated and given C.P.A.P. Clinical kyphoscoliosis was confirmed with X-ray. Full septic work-up was carried out in view of P.R.O.M. Chest X-ray was consistent with mild R.D.S. Antibiotics were commenced. Over the following 24 hours the baby became oedematous and at the age of 24 hours the infant collapsed. This was in association with severe metabolic acidosis with subsequent attacks of severe apnoea. The oedema increased and a pericardial effusion was aspirated. The baby had subsequent cardiac arrests and died at 31 days of age. Lumbar puncture performed at this time was bloodstained. All cultures were sterile apart from an eye swab which grew actinobacter species which was sensitive to the antibiotic used.

**Post-mortem:**
(i) Massive intraventricular haemorrhage.
(ii) Haemopericardium.
(iii) Pulmonary and renal haemorrhages.

(82) N.B.55896. Para 2\textsuperscript{a}. Age 33. S.E.3. Gestation 29 weeks. No significant past history. Transferred from a country hospital in premature labour at 29 weeks. S.R.O.M. 2 hours earlier, clear liquor. After a labour lasting 11 hours spontaneous delivery of live female infant weighing 1.19 kilograms, born with Apgar scores of 1 and 7. Infant transferred to S.C.B.U. and commenced on nasopharyngeal C.P.A.P. and shortly thereafter a nasotracheal tube was passed and full ventilation commenced. With this there was some improvement but the peripheries remained grey in colour. An arterial gas revealed a severe metabolic acidosis with pH=7. Base Excess —20 while gases were normal. Further support was given with bicarbonate and fresh frozen plasma to no avail and the infant died 51 hours following delivery.

**Post-mortem:**
(i) Hyaline membrane disease.
(ii) Subarachnoid haemorrhage.

**Congenital Malformations**
**Prolapse and Presentation of the Cord**

Number of cases ..................  32
Incidence per cent ................  0.43
Twins ............................  1
Number of babies delivered ......  33
Stillbirth ........................  1
Neonatal deaths ..................  2
Perinatal deaths ..................  3 Rate: 90.9/1,000
Method of delivery:
   - Caesarean section ............  24
   - Spontaneous vertex ...........  5
   - Breech ........................  3
   - Ventouse ........................  1

**Comment:** Six of the cases occurred in primigravidae and 3 were associated with transverse lie in labour. Both neonatal deaths (96 and 100) were due to congenital malformations but the stillbirth (75) could have been avoided by better intrapartum care.

**PERINATAL DEATHS**

*Normal Infant*

(75) B.6131, Para 5<sup>40</sup>. Age 33, S.E.5. Gestation 41 weeks. Booked at 15 weeks. Admitted at 37 weeks with P.E.T. Head too high for A.R.M. Spontaneous onset of labour at 41 weeks — prolapse of cord at 8 cms. Spontaneous delivery of fresh stillborn male infant weighing 2.70 kilograms.

*Post-mortem:* Intra-partum asphyxia (cord prolapse).

**Congenital Malformations**

Cases 96 and 100.

**COMPARATIVE TABLE FOR 10 YEARS**

<table>
<thead>
<tr>
<th></th>
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<th></th>
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<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cases</td>
<td>17</td>
<td>23</td>
<td>25</td>
<td>25</td>
<td>20</td>
<td>29</td>
<td>34</td>
<td>26</td>
<td>20</td>
<td>32</td>
</tr>
<tr>
<td>Incidence %</td>
<td>0.5</td>
<td>0.25</td>
<td>0.29</td>
<td>0.3</td>
<td>0.3</td>
<td>0.28</td>
<td>0.41</td>
<td>0.5</td>
<td>0.35</td>
<td>0.43</td>
</tr>
<tr>
<td>Perinatal deaths</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>5</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Mortality rate</td>
<td>55.5</td>
<td>74.0</td>
<td>40</td>
<td>200</td>
<td>130.4</td>
<td>125</td>
<td>135.1</td>
<td>38.4</td>
<td>45.5</td>
<td>90.9</td>
</tr>
</tbody>
</table>

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**Forceps Delivery**

<table>
<thead>
<tr>
<th>Number of cases</th>
<th>562</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence per cent</td>
<td>7.3</td>
</tr>
<tr>
<td>Twins</td>
<td>.11</td>
</tr>
<tr>
<td>Number of babies delivered</td>
<td>569</td>
</tr>
<tr>
<td>Stillbirths</td>
<td>4</td>
</tr>
<tr>
<td>Neonatal deaths</td>
<td>4</td>
</tr>
<tr>
<td>Perinatal deaths</td>
<td>8</td>
</tr>
</tbody>
</table>

**Indications:**

<table>
<thead>
<tr>
<th>Indication</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Failure to advance</td>
<td>304</td>
</tr>
<tr>
<td>Fetal distress</td>
<td>192</td>
</tr>
<tr>
<td>Prematurity</td>
<td>19</td>
</tr>
<tr>
<td>A.P.H</td>
<td>16</td>
</tr>
<tr>
<td>Epidural</td>
<td>14</td>
</tr>
<tr>
<td>Maternal distress</td>
<td>14</td>
</tr>
<tr>
<td>P.E.T./Eclampsia</td>
<td>5</td>
</tr>
<tr>
<td>Hypertension</td>
<td>4</td>
</tr>
<tr>
<td>Cardiac disease</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Type of anaesthetic used</th>
<th>Count</th>
</tr>
</thead>
<tbody>
<tr>
<td>Local infiltration</td>
<td>414</td>
</tr>
<tr>
<td>Lumbar epidural</td>
<td>148</td>
</tr>
</tbody>
</table>

**Comment:** The incidence of forceps delivery has remained remarkably constant at 7.3 per cent. As in previous years the main indications were delay in the second stage and fetal distress. The number of perinatal deaths was slightly higher than in 1979 but none of the deaths was due to the forceps delivery. The 4 stillbirths had died prior to the onset of labour; 3 of the 4 neonatal deaths were due to major congenital abnormalities, whilst the fourth was due to intra-ventricular haemorrhage associated with extreme prematurity. There was no case of traumatic intra-cranial haemorrhage. Twenty babies weighed less than 2.5 kilograms; their weights and gestation ranged from 915 grams at 29 weeks to 2.49 kilograms at 40 weeks.
PERINATAL DEATHS

Normal Infants

(15) B.49146. Para 0\textsuperscript{+1} Age 29. S.E.I. Gestation 41 weeks. Irregular cycle. F.M. ceased at 41 weeks. Spontaneous labour and forceps delivery of macerated male infant weighing 3.06 kilograms.


Post-mortem: Intra-uterine anoxia.

(47) N.B.53432 Para 0\textsuperscript{+0} Age 20. S.E.5. Gestation 37 weeks. Transferred from country hospital at 37 weeks gestation with jaundice of one week's duration. F.H.H. on admission. Patient was jaundiced with high bilirubin levels, very low platelet levels and moderately raised transaminases. A provisional diagnosis of either leptospirosis, obstructive jaundice or viral hepatitis was made. Two days following admission, patient went into spontaneous labour. F.H.N.H. Prophylactic forceps delivery of a stillborn female infant weighing 2.68 kilograms. Primary post-partum haemorrhage treated with platelets and blood. Patient improved following delivery. Three days later patient transferred to general hospital. There liver biopsy performed and diagnosis of acute fatty liver made.

Post-mortem: Maceration.


Post-mortem: Maceration.

(79) B.19241. Para 3\textsuperscript{+0} Age 34. S.E.2. Gestation 28 weeks. Multiple pregnancy diagnosed on scan. Acute hydramnios at 28 weeks and admitted. Trans-abdominal amniotomy but labour occurred. Forceps delivery of live male infants. Twin 1, birth weight 1.25 kilograms. Apgar scores 0 and 3. The infant was intubated on delivery and given I.P.P.V. with slow improvement. Blood gases revealed severe mixed respiratory metabolic acidosis with adequate oxygenation. The baby's fontanelle was rather full and sutures splayed and C.S.F. was uniformly bloodstained. The baby died at 10 hours of age.

Post-mortem: Large intraventricular haemorrhage extending through the mid brain, cerebellum and into sub-arachnoid space. Total atelectasis of both lungs.

Congenital Malformations

Cases 99,102 and 106.
**Vacuum Extraction**

Number of cases .......................... 58
Incidence per cent ........................ 0.75
Number of babies delivered ................. 58
Perinatal deaths ............................ Nil

*Comment:* Fifty eight babies were delivered by vacuum extraction during the year, an increase of 18 on the figures for 1979. Forty (69 per cent) were primigravidae. The main indications were second stage delay associated with malrotation of the occiput (46.5 per cent) and fetal distress (40 per cent). Local anaesthesia was used in 60 per cent of cases and epidural analgesia in the remaining 40 per cent. Labour was induced in 19 cases (33 per cent) and was prolonged in a further 2 cases (36 per cent). Three babies weighed less than 2.5 kilograms. There were no perinatal deaths.
Caesarean Section

<table>
<thead>
<tr>
<th>Number of cases</th>
<th>Incidence per cent</th>
<th>Twins</th>
<th>Number of babies delivered</th>
<th>Stillbirths</th>
<th>Neonatal deaths</th>
<th>Perinatal deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>12</td>
</tr>
</tbody>
</table>

Rate: 19.8/1,000

Main indications in each case:

- Repeat elective
- Fetal distress
- Breech
- Failure to advance
- Placenta praevia
- Disproportion
- Cord prolapse
- Transverse lie in labour
- Retarded intra-uterine growth
- Unstable lie
- Failed induction
- Abruptio placentae
- Pre-eclampsia
- Essential hypertension
- Bad history
- Rhesus
- A.P.H. (unknown origin)
- Previous vaginal surgery
- Eclampsia
- Metroplasty

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Comment: There was a slight rise in the incidence of Caesarean section during 1980. However, even though more patients were monitored continuously in labour the number of Caesarean sections performed for fetal distress actually decreased. This may have been due to the availability of scalp pH measurement, though 22 of the cases in whom Caesarean section was performed for fetal distress were not monitored during labour. There were 3 stillbirths (27, 52 and 71) and an avoidable factor was present in one case (71). Four of the 9 neonatal deaths were due to congenital malformations (87, 91, 94 and 98), 1 was due to iso-immunisation with anti-Kell antibodies (83), 1 was associated with bleeding from ruptured vasa previa (77) and 1 with prematurity (80). Avoidable factors were present in the other 2 cases (76 and 78). There was no maternal death associated with Caesarean section.

PERINATAL DEATHS

Normal Infants


Post-mortem: Intra-uterine anoxia secondary to placental abruption.


Post-mortem: (i) Strangulation of the neck by a nuchal cord.
   (ii) Visceral congestion and oedema with pulmonary haemorrhage.
   (iii) Normal placenta.

(76) B.43120. Para 2\(^{+1}\) S.E.5. Gestation 38 weeks. Booked at 23 weeks. R.I.U.G. noted at 37 weeks and confirmed by scan. Booked for further scan but was admitted in labour at 38 weeks with slow irregular fetal heart and meconium stained liquor. L.S.C.S. performed. Female infant weighing 2.72 kilograms, flat at delivery. At delivery the baby had no apex and cardiac massage was given followed by intubation. Intermittent positive pressure was used and an apex beat was obtained. On admission to the S.C.B.U. the baby had occasional spontaneous respirations and required reintubation. Initial pH was 6.86 at 30 minutes of age. Anti cerebral oedema therapy was initiated and the baby remained critically ill. Further resuscitation was not undertaken. The baby died on the 2nd day.

Post-mortem: Non specific, and could be attributed to intra-uterine asphyxia.

(77) B.45670. Para 1\(^{+1}\) Age 23. S.E.3. Gestation 41 weeks. Normal antenatal record. Labour induced at 41 weeks. Some 2 hours later during strong contractions brisk vaginal haemorrhage with marked fetal bradycardia. Emergency L.S.C.S. Baby very flat, appeared exsanguinated, cord flacid. Presumptive diagnosis of ruptured vasa praevia. Female alive weighing 3.45 kilograms born with Apgar scores 1 and 0. While blood was being drawn from an 0 Neg. donor for immediate transfusion following delivery, the infant had a cardiac arrest. Within 4 hours of delivery the infant was transfused with 200ml. of fresh blood. Following this
and during the latter part of the transfusion the baby was noted to be oozing from puncture sites. This tendency was counteracted with protamine sulphate pending coagulation screen result. By 14 hours of age marked cerebral signs were noted and the baby remained aneuric. Subsequently continued oozing and drop in haemoglobin led to exchange transfusion which was well tolerated with little improvement in oozing from puncture sites. The baby's cerebral status deteriorated with seizures and at the age of 48 hours the coagulopathy was almost corrected, and urinary output also improved. The baby continued to require full ventilation and at the age of 3s days had a cardiac arrest and was not resuscitated further.

**Post-mortem:**
(i) Severe anoxic injury with cerebral haemorrhage.
(ii) Pulmonary haemorrhage and oedema.

(78) B.41239. Para 1<sup>st</sup> Age 30. S.E.2. Gestation 42 weeks. Booked at 8 weeks gestation. Uneventful A.N.C. except for anaemia. Admitted at 38 weeks gestation because of oblique breech. By 42 weeks cephalic presentation. Stable but high. Clinically big baby. PGE induction. Head did not descend in pelvis in second stage of labour and L.S.C.S. performed. Male infant weighing 4.71 kilograms, born with Apgar scores of 5 and 4. Baby was admitted to S.C.B.U. and following resuscitation the endotracheal tube was removed at 25 mins. Initial pH showed severe metabolic respiratory acidosis, with a pH of 6.8. Following extubation the baby had poor respiratory effort with extreme hypotonia and staring associated with a fine tremor of face and feet. Treatment with Mannitol and Dexamethasone was commenced and the baby was slow to pass urine. At 24 hrs. of age the baby was noted to be having increasing respiratory distress associated with pneumonia. The baby was further supported with ventilation with some improvement. Neurological examination continued to be very abnormal. At 46 hours of age the baby was markedly hypoxic and despite every effort to improve ventilation the baby died.

**Post-mortem:**
(i) Severe anoxic changes in the brain.
(ii) Bilateral bronchial pneumonia.
(iii) Pneumococcus was grown from the endotracheal tube and eye swab.

(80) B.25055. Para 20 Age 34. S.E.3. Gestation 29 weeks. Booked at 10 weeks gestation. Regular attender at the antenatal clinic. Admitted at 29 weeks in labour with transverse lie. L.S.C.S. Male infant weighing 1.55 kilograms born with Apgar scores of 8 and 9. Following admission to S.C.B.U. the baby was intubated and given C.P.A.P. Clinical kyphoscoliosis was confirmed with X-ray. Full septic work up was carried out in view of P.R.O.M. Chest X-ray was consistent with mild R.D.S. Antibiotics were commenced. Over the following 24 hours the baby became oedematous and at the age of 24 hours the infant collapsed. This was in association with severe metabolic acidosis with subsequent attacks of severe apnoea. The oedema increased and a pericardial effusion was aspirated. The baby had subsequent cardiac arrests and died at 3| days of age. Lumbar puncture performed at this time was bloodstained. All cultures were sterile apart from an eye swab which grew actinobacter species which was sensitive to the antibiotic used.

**Post-mortem:**
(i) Massive intraventricular haemorrhage.
(ii) Haemopericardium.
(iii) Pulmonary and renal haemorrhages.

(83) B.18680. Para 2<sup>nd</sup> Age 28. S.E.3. Gestation 30 weeks. Booked at 10 weeks gestation. Blood group A Rh. positive. Previous pregnancies (i) full term delivery followed by primary P.P.H. and blood transfusion, (ii) full term normal delivery, no complications. Present pregnancy uneventful until 29 weeks gestation when patient complained of oedema of legs, feeling tired and being uncomfortable. B.P. 140/100, no proteinuria, hydramnios. Scan showed huge placenta, Grade I-II praevia. Infant appeared hydropic with ascites. Mother found to have anti-Kell antibodies. A.P.H. and emergency L.S.C.S. with delivery of female infant weighing 2.2 kilograms, born with Apgar scores of 1 and 1. The baby was markedly hydropic and required intubation. Hydrops fdiusus secondary to anti-Kell antibodies. Baby given 2 exchange transfusions and 3 top-up transfusions. Developed hypoglycaemia and hypercaleaemia which were treated with peritoneal dialysis. Baby failed to respond and died at 7 days of age. The mother was given 6 units of blood during L.S.C.S. and developed puerperal pyrexia due to bacteroides but made a good recovery.

**Post-mortem:**
(i) Massive intra cerebral haemorrhage.
(ii) Massive extra medullary haematopoiesis related to haemolytic disease.
(iii) Traumatic emphysema and subpleural bulla.

**Congenital Malformations**

Cases 52, 87, 91, 94 and 98.
# Labour Following Previous Caesarean Section

<table>
<thead>
<tr>
<th>Method of delivery:</th>
<th>Number of babies delivered</th>
<th>Stillbirths</th>
<th>Neonatal deaths</th>
<th>Perinatal deaths</th>
<th>Rate: 14.1/1,000</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous</td>
<td>204</td>
<td>3</td>
<td>1</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>L.S.C.S</td>
<td>40</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forceps</td>
<td>34</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breech</td>
<td>4</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Comment: Two hundred and seventy eight patients who had previously had a Caesarean section performed, laboured. Forty patients (14.3 per cent) required repeat emergency Caesarean section. Thirty-four patients were delivered by forceps (12.2 per cent) and there were two hundred and four spontaneous vaginal deliveries (73 per cent). Labour was induced in fifty cases (18 per cent) and was prolonged, it is disturbing to note, in thirty cases (10.8 per cent). There were no cases of scar rupture during 1980. There were 3 stillbirths and 1 neonatal death, none of which could be associated with labour. Seventeen babies weighed less than 2.5 kilograms, ranging in weight and gestation from 1.32 kilograms at 39 weeks to 2.49 kilograms at 40 weeks.

## PERINATAL DEATHS

### Normal Infants


**Post-mortem:** Maceration.


**Post-mortem:** Maceration.

### Congenital Malformations

Cases 58 and 91.
**Thrombosis**

Ante-Natal:
- Superficial: 9
- Deep Venous Thrombosis: 3

Puerperal:
- Superficial: 43
- Deep Venous Thrombosis: 4

*Comment:* The incidence of superficial thrombophlebitis was similar to that recorded previously. These cases were treated topically. Deep venous thrombosis occurred quite infrequently with only 7 treated cases among 7,596 mothers. The low incidence may be related to the routine use of plasma expanders during all Caesarean sections. Although 2 of the 59 patients who developed thrombosis had stillborn infants (22 and 27) the thrombosis was in no way related to the perinatal deaths.

**PERINATAL DEATHS**

*Normal Infants*


*Post-mortem:* Maceration.


*Post-mortem:* Intra uterine anoxia secondary to placental abruption.
Manual Removal of Placenta

Number of cases ........................................... 109
Incidences per cent ...................................... 1.4
Twins ......................................................... 3

Method of delivery:
- Spontaneous ............................................. 84
- Breech ..................................................... 12
- Forceps .................................................... 11
- Vacuum ................................................... 2

Comment: The number of manual removals of the placenta has not altered over the past ten years. Eight of the 109 cases were associated with primary post-partum haemorrhage. The overall morbidity was low and only one of the 109 patients developed a puerperal genital infection.

COMPARATIVE TABLE FOR 10 YEARS

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of cases</td>
<td>104</td>
<td>140</td>
<td>140</td>
<td>129</td>
<td>118</td>
<td>155</td>
<td>109</td>
<td>124</td>
<td>104</td>
<td>109</td>
</tr>
<tr>
<td>Incidence %</td>
<td>1.6</td>
<td>1.8</td>
<td>1.9</td>
<td>1.7</td>
<td>1.6</td>
<td>2.2</td>
<td>1.6</td>
<td>1.6</td>
<td>1.4</td>
<td>1.4</td>
</tr>
</tbody>
</table>
**Postpartum Haemorrhage**

<table>
<thead>
<tr>
<th>Number of cases</th>
<th>129</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence per cent</td>
<td>1.7</td>
</tr>
<tr>
<td>Twins</td>
<td>3</td>
</tr>
</tbody>
</table>

Aetiology:

**MODE OF DELIVERY**

<table>
<thead>
<tr>
<th>Placental fragments</th>
<th>S.V.D.</th>
<th>V.E.</th>
<th>C.S.</th>
<th>Forceps</th>
<th>Breech</th>
<th>Twins</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole Placenta</td>
<td>47</td>
<td>1</td>
<td>4</td>
<td>3</td>
<td>2</td>
<td>2</td>
<td>59</td>
</tr>
<tr>
<td>Traumatic</td>
<td>10</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>4</td>
<td>—</td>
<td>14</td>
</tr>
<tr>
<td>Uterine atony</td>
<td>37</td>
<td>2</td>
<td>1</td>
<td>6</td>
<td>1</td>
<td>1</td>
<td>48</td>
</tr>
</tbody>
</table>

Total: 102 3 5 13 3 3 129

| Primary | 36 | 2 | 4 | 10 | 1 | 2 | 55 |
| Secondary | 66 | 1 | 1 | 3 | 2 | 1 | 74 |

**Comment:** The incidence of postpartum haemorrhage has changed little over the last ten years. Labour was induced in 35 of the 129 cases (27 per cent) and had been prolonged (greater than 12 hours) in 11 patients. Twenty-four of the 129 patients (18 per cent) had previously had 4 or more children. Eleven patients required 4 or more units of blood, 3 of these requiring 14 or more units. There was one case of ruptured uterus which required hysterectomy to control the bleeding.

**COMPARATIVE TABLE FOR 10 YEARS**

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of cases</td>
<td>97</td>
<td>183</td>
<td>168</td>
<td>191</td>
<td>177</td>
<td>145</td>
<td>165</td>
<td>125</td>
<td>151</td>
</tr>
<tr>
<td>Incidence %</td>
<td>1.5</td>
<td>1.7</td>
<td>2.2</td>
<td>2.5</td>
<td>2.4</td>
<td>2.1</td>
<td>2.3</td>
<td>1.7</td>
<td>2.0</td>
</tr>
<tr>
<td>Twins</td>
<td>—</td>
<td>—</td>
<td>1</td>
<td>9</td>
<td>5</td>
<td>1</td>
<td>6</td>
<td>4</td>
<td>3</td>
</tr>
</tbody>
</table>
Puerperal Pyrexia

Number of cases ........................................ 150
Incidence per cent ...................................... 1.9

Genital Infection ........................................... Number of cases 62
Method of delivery:
- Spontaneous .............................................. 38
- Caesarean section ........................................ 13
- Forceps ..................................................... 9
- Breech .................................................... 2

B. Non-Genital Infection .................................... Number of cases 88
- Respiratory tract infection ............................ 24
- Urinary tract infection .................................. 17
- Wound infection .......................................... 11
- Breast lesions ............................................ 10
- Other causes ............................................. 26

Comment: There was a slight increase in the number of cases of puerperal pyrexia in comparison to those recorded in 1979. Fifty-five of the patients who were delivered by Caesarean section developed puerperal pyrexia, 13 of the cases being due to genital infection and 42 to non-genital. Thirteen of the 62 cases of genital tract infection occurred following induction of labour; 8 occurred following prolonged labour and 1 was associated with manual removal of the placenta. The organisms most commonly isolated were Bacteroides, Anaerobic streptococci, E.coli, and Streptococci.

COMPARATIVE TABLE FOR 9 YEARS

<table>
<thead>
<tr>
<th>Year</th>
<th>Number of cases</th>
<th>Incidence %</th>
</tr>
</thead>
<tbody>
<tr>
<td>1972</td>
<td>71</td>
<td>0.9</td>
</tr>
<tr>
<td>1973</td>
<td>153</td>
<td>2.0</td>
</tr>
<tr>
<td>1974</td>
<td>81</td>
<td>1.1</td>
</tr>
<tr>
<td>1975</td>
<td>91</td>
<td>1.2</td>
</tr>
<tr>
<td>1976</td>
<td>82</td>
<td>1.2</td>
</tr>
<tr>
<td>1977</td>
<td>130</td>
<td>1.9</td>
</tr>
<tr>
<td>1978</td>
<td>111</td>
<td>1.5</td>
</tr>
<tr>
<td>1979</td>
<td>112</td>
<td>1.5</td>
</tr>
<tr>
<td>1980</td>
<td>150</td>
<td>1.9</td>
</tr>
</tbody>
</table>
Physiotherapy and Mothercraft

OBSTETRICAL

During 1980 there was an ever increasing rise in primigravidae attendance at the ante-natal classes. Figures for the year have shown that approximately 50 per cent of primigravid mothers delivered in the hospital attended the physiotherapy classes beforehand. Of the 1,362 notified, 1,245 did so: 481 were public; 446 semi-private and 225 were private. The attendance at the Coombe classes totalled 7,901. In addition, many other patients attended evening classes outside the hospital, either because they were working, or because of geographical considerations.

Our course consisted of a series of eight classes, the first being an enrolment followed by a lecture given by one of the Assistant Masters. The classes concluded with a visit to the delivery suite so that the primigravid mothers might see where their babies will be born and hopefully dispel any fears that they may have had. This visit is generally found most encouraging. Each class lasted \( \frac{2}{3} \) hours; one hour was given over to Physiotherapy and the remaining \( \frac{1}{3} \) hour to Mothercraft. The physiotherapist outlines the ailments of pregnancy and the three stages of labour and teaches breathing and relaxation used during labour. The midwife in her half hour covers all aspects of Mothercraft. Particular attention is paid to promoting the importance of breast feeding and during the year 2,394 of the 7,596 (32 per cent) mothers breast fed.

More mothers attended the Refresher class every Friday morning. These were multiparae who wanted to refresh their memories on the ante natal classes that most had attended with their first pregnancies. During these classes we talked to 335 multiparae. Post-natally patients who wished were encouraged to attend classes in the gymnasium. Post-natal leaflets were distributed in the wards and the exercises shown on the leaflets were instructed in the classes. A total of 2,174 attended the Post-natal classes during 1980.

Mr. Roger Waugh, the representative from Richardson/Merril continued to show three films (Ready for Baby. Where Love ends and The Joy of Nursing) on the first Thursday of every month. One thousand one hundred and twenty nine husbands and wives attended during 1980.

GYNAECOLOGICAL

Routinely we continued to administer pre and post-operative physiotherapy to all patients undergoing major surgery with regard to breathing exercises, and foot and ankle exercises for the prevention of unnecessary chest, or circulatory, problems.

The Physiotherapy department itself remained steady in the number of heat treatment to perineums, wounds, abscesses and 206 applications of infra-red were carried out. Three hundred and eighty nine shortwave diathermy treatments were performed. These were mostly for backache or pelvic conditions. However there were no treatments of vaginal faradism as this form of treatment for those with incontinence, or weak pelvic floor muscles, is now considered too stressing on the patient. It is now preferred to treat those patients conservatively with simple pelvic floor exercises.
ORTHOPAEDIC PAEDIATRIC PHYSIOTHERAPY

Babies with subluxated, or click, hips were referred to Physiotherapy and were fitted with a pelvic harness by the physiotherapist and then reviewed fortnightly as outpatients. Bad talipes equino varus were strapped sometimes twice and three times daily. Other babies with torticollis necks were referred to Physiotherapy where the mothers were instructed in stretching and exercises for the neck to correct the problem. These babies were also reviewed fortnightly until the problem had been corrected. A total of 738 orthopaedic treatments were carried out in 1980.

PAEDIATRIC CHEST PHYSIOTHERAPY

Both in-patient and out-patient chest Physiotherapy was carried out on babies with chest conditions such as pneumonia, bronchitis, respiratory distress syndrome and cystic fibrosis. This totalled 3,883 treatments during 1980.

NEURO-DEVELOPMENTAL PHYSIOTHERAPY

This aspect of Physiotherapy is gaining more and more importance and the numbers of babies referred for treatment constantly increased in 1980. With the extra help of Mrs. Coote who works solely with the "At Risk" babies, the three full time physiotherapists carried out 1,975 assessments and treatments on those babies that required specialised exercises. The majority of these were out-patients.

COURSES ATTENDED

In October Mrs. Riana O'Cofaigh and Miss Ann O'Loughlin attended a weekend Seminar for physiotherapists in Obstetrics and Gynaecology in the Wilton Hospital in Cork. One of the lectures was given by Mrs. Joy Coote on the "At Risk Baby" and the Physiotherapy treatment. Examples and slides used for her lecture consisted entirely of "Coombe babies".

In 1980 we bade farewell to two of our Physiotherapists; Mrs. Edwina Hamill, who had worked in the hospital for seven years, and Miss Ann Sheehan who had been with us for two years. They were replaced by Ann O'Loughlin and Sunny Kavanagh respectively.
Department of Pathology

The total units of work carried out during the past year was 303,186. The breakdown into the various categories is as follows:

- Bacteriology: 95,000
- Histology: 5,970
- Cytology: 10,859
- Biochemistry: 81,789
- Blood Grouping: 43,550
- Haemathology: 66,018

Total: 303,186

There was a total of 107 autopsies performed during the year; 93 of these were in the perinatal category and they are set out in the table as follows:

<table>
<thead>
<tr>
<th>Category</th>
<th>Total Number of Cases</th>
<th>Number of Autopsies</th>
<th>Percentage Autopsy Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Still-births</td>
<td>75</td>
<td>64</td>
<td>85%</td>
</tr>
<tr>
<td>Neonatal Deaths</td>
<td>33</td>
<td>29</td>
<td>88%</td>
</tr>
<tr>
<td>Total:</td>
<td>108</td>
<td>93</td>
<td>86%</td>
</tr>
</tbody>
</table>

Congenital anomalies incompatible with life accounted for 36 of the deaths in the autopsy series. These include anomalies of the central nervous system (11), cardiac (4), renal tract (7), Exomphalos (1), Sirenomelia (1), Acardiac Monster (1), Chromosomal (2), Diaphragmatic Hernia (2), Multiple Haemangiomata (1).

During the year 20 new cases of malignant disease were encountered, comprising endometrium (8), Ovary (3), Cervix (4), Bartholin’s gland (1), Vulva (1), Urethra (1), Pelvis (1), Choriocarcinoma (1). In addition, there were 15 new cases of in-situ carcinoma of the cervix.
Eighteen pregnancies have to-date occurred amongst the 90 infertile patients, so that the pregnancy rate for women attending the clinic was 20 percent.
Social Service Department

The following is an analysis of the work of this Department for 1980:

Advice to Unmarried Mothers ........................................... 493
Housing ................................................................. 275
Extra Nourishment ....................................................... 269
Baby Clothes ........................................................... 223
Advice on Domestic Problems ......................................... .117
Care of Children ......................................................... 81
Samaritan Fund ......................................................... 57
Home Visits ............................................................. 52
Adult Clothing and Bedding ............................................ 27

During the year 493 single patients were interviewed. Of these 30 married shortly after their first visit. Sixteen had no ante-natal care, 4 had stillborn infants and there were 4 neonatal deaths and 7 miscarriages. Of the remainder 333 kept their babies and 85 had them adopted; 5 were confined elsewhere and 11 were admitted to a Special Home prior to confinement. In 60 cases they were cohabiting and 14 were follow-up contacts of 1979.

The trend for the Single mother to keep her baby is increasing. There are cases where one would have reservations as to the advisability of this as regards the welfare of the child. At present however until the child is shown to have been neglected it cannot be taken into care or protective custody.

A problem that emerged during the year and which seems to be likely to become more widespread is the drug problem. Co-operation is maintained with Jervis Street Drug Centre in an effort to help these patients to become drug free prior to their confinement. However the apparent easy availability of drugs in the Community makes this task difficult.

During the year a Casework Service was given to 511 patients. This frequently involved contact with the patient over a period of three or more months.

In 117 cases the problem was due to a difficult domestic or marital situation. Of these, 46 patients were either separated or deserted. In 81 instances the problem was linked with child welfare. Poor living conditions and overcrowding have a detrimental effect on the health of a pregnant patient. They can also be a contributory factor in connection with the baby's re-admission to the Hospital. Unfortunately the housing situation is still difficult as the points required for an applicant to be successful have, like inflation, increased.

Our thanks are due to the members of the Linen Guild for their continued concern for our patients, as manifested by their generous supply of layettes and Christmas Hampers.
Extern Services

Deliveries

Number of cases over 28 weeks maturity: 1
Perinatal Deaths: Nil

Comment: The District Service was called out to look after one unbooked patient who had had no ante-natal care whatever. She had a normal delivery of a full term live-born female infant and was admitted to the hospital following delivery.

Paediatric Home Visiting

A total of 5,887 home visits took place during 1980. It is hoped that it might be possible to reduce this particular work load in the department by improving the facilities which may be available in liaison with the Community Health Services and this aspect is being looked into.

Flying Squad

The Flying Squad was called out on 3 occasions during 1980. Two of these patients were obstetrical patients, while the third call was a Paediatric call to Cavan where a lady had delivered triplets at 28 weeks gestation. This latter call was the first time a Paediatric Flying Squad call had been requested and it was gratifying that the hospital was able to send two experienced medical staff as well as experienced nursing personnel to deal with this particular problem. It was also gratifying to state that 2 of the 3 triplets survived following intensive care in the Paediatric Unit for many weeks.

Both the obstetrical flying squad calls were due to post partum haemorrhage. One was for an unbooked patient who had no ante-natal care and had delivered at home; she was brought to the Coombe Hospital and, following transfusion made a satisfactory recovery. The second call took place to a County Hospital where a patient had had a hysterectomy for a severe post partum haemorrhage. In fact, on arrival the patient's condition was stable but she was transferred to the Coombe Hospital where she was maintained on conservative management for a week prior to re-transfer to her own hospital.
### Department of Radiology

<table>
<thead>
<tr>
<th>Obstetrical/Gynaecological</th>
<th>General</th>
<th>Paediatric</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maturity</td>
<td>Chest</td>
<td>Chest</td>
</tr>
<tr>
<td>Position</td>
<td>Abdomen</td>
<td>45 Abdomen</td>
</tr>
<tr>
<td>Fetal Abnormality</td>
<td>Spine</td>
<td>160 Spine</td>
</tr>
<tr>
<td>Multiple</td>
<td>Skull &amp; Pit. Fossa</td>
<td>90 Skull</td>
</tr>
<tr>
<td>Pelvimetry</td>
<td>Sinuses</td>
<td>2 Sinuses</td>
</tr>
<tr>
<td>Salpingography</td>
<td>Hips</td>
<td>4 Hips</td>
</tr>
<tr>
<td></td>
<td>Pelvis</td>
<td>8 Pelvis</td>
</tr>
<tr>
<td></td>
<td>Bones</td>
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<tr>
<td></td>
<td>I.V.P.</td>
<td>57 I.V.P.</td>
</tr>
<tr>
<td></td>
<td>Port. Chest</td>
<td>22 Trachea</td>
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<td></td>
<td>Cholecystogram</td>
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<td></td>
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<tr>
<td></td>
<td>Port. Skull</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Port. Mict. Cystogram</td>
<td>1</td>
</tr>
</tbody>
</table>

| Total                     | 445     | 721        | 3,586      |

**Comment:** The work load in the Radiology Department was generally speaking similar to that recorded in 1979, although a further fall in the number of obstetrical X-rays took place due to the continued increased use of ultrasound scanning.
Research and Publications

The research work being carried out by Dr. B. Stuart, under the finance of the Florence and William Blair Bell research fellowship from the Royal College of Obstetricians and Gynaecologists was concluded during 1980. The results of the studies were presented by Dr. Stuart at the British Congress of Obstetrics and Gynaecology, in Edinburgh, as well as being presented to the Blair Bell research society in London.

Dr. J. Murphy also concluded his research into aetiological aspects of cervical neoplasia which was being performed in conjunction with the University of Birmingham and was being supported by The Irish Cancer Society.

A prospective study into the Progress and Outcome of a Breech Labour, commenced over two years ago, was also concluded during 1980 and the results will be published shortly.

A new study was commenced in conjunction with Dr. J. P. McKenna into The Role of The Adrenal Gland in patients with amenorrhoea and infertility.

The hospital continued to employ 2 research doctors and 1 research sister and it is planned to extend the research into the neonatal unit in 1981.

PUBLICATIONS BY MEMBERS OF THE MEDICAL STAFF, 1980


