FOETAL ANOXIA

BY

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A Clinical and Laboratory Study

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"And in the womb of a mother was I moulded into flesh:
Within the space of ten months, . . ."
Wisdom of Solomon, VII, 1, 2.

I wish to thank the Council of the College for the honour they have conferred upon me in the award of a Blair-Bell Memorial Lectureship.

My subject is perhaps appropriate, as, in 1928, the late Professor Blair-Bell, in co-operation with colleagues in Liverpool, published the first recorded readings of the oxygen content of the blood in the umbilical vessels in the human foetus.

The foetus depends for its growth and development on an adequate supply of nutrients from the mother. Oxygen is unique amongst those, as it cannot be stored and the supply must be ample and continuous. Even a moderate deficiency over a short period may seriously injure the foetus (Ingalls et al., 1950). Near term the human foetus can survive a total lack of oxygen for a period of about 30 minutes, provided its circulation is intact, and lesser degrees of anoxia for longer periods. It is doubtful, however, if it will always escape completely unharmed, as the higher cerebral centres are less resistant than the more primitive centres directly responsible for survival (Darke, 1944; Brinkman, 1953).

OXYGEN SUPPLY TO THE HUMAN FOETUS

Several important factors are concerned in maintaining the supply of oxygen to the foetus. The maternal arterial blood must be adequately saturated with oxygen and the maternal general circulation normal. The blood vessels to, and in the uterine wall must grow and develop normally in response to the pregnancy. The placenta should be normally grown and the deciduo-placental site fully developed and healthy. The layers of the foetal villus between the two bloods should function normally, and the circulation in the umbilical cord must not be impeded. Maternal blood pours into the intervillous spaces and oxygen is transferred by diffusion to the foetal blood.

Circulation of the maternal blood through the placenta in late pregnancy and labour depends on regular uterine contractions. Inadequate or prolonged contractions or a high resting tone may interfere with this circulation. In the foetal vessels circulation depends mainly on the action of the foetal heart.

All those factors govern the amount of oxygen reaching the foetus, and disease processes of many kinds may interfere. Even, however, in the absence of disease, the oxygen supply undergoes a series of changes as pregnancy progresses. This was first demonstrated by Barcroft and his colleagues in the goat (1934) and the sheep (1939).

Very little is known about the levels of oxygen supply to the human foetus before the onset of labour. What information there is, is confusing, and the conclusions drawn are contradictory (Eastman, 1930; Haselhorst and Stromberger, 1930, 1931; Dieckmann and Kramer, 1944). No studies have been made of possible variations of oxygen supply during normal pregnancy, or in association with abnormal clinical states.

I felt that it would be worth while to study the oxygen levels in the blood of the human foetus at abdominal and vaginal delivery in the latter
half of pregnancy, before and after labour, and in normal and abnormal cases. The problem is really one of human physiology, but it seemed an ideal investigation for a clinician to undertake, as a detailed experience of the clinical features of pregnancy and labour is essential in the interpretation of the results.

Methods

For all cases studied there was a detailed clinical history and the length of gestation was accurately known. Spinal or local anaesthesia was used for all operative deliveries studied, because inhalation anaesthetics interfere with most techniques of blood-gas analysis (Shaw and Downing, 1935). They may, in addition, interfere with the oxygen supply to the foetus (Smith, 1939; Taylor et al., 1951).

I took all blood samples myself and was present during delivery in all cases. Thus, difficulty in delivery could be assessed, the state and response of the baby noted, and samples could be obtained accurately and quickly. All oxygen estimations were made as quickly as possible and always within 6 hours. The method used, that of Roughton and Scholander (1943), could be applied at short notice, and was accurate with close agreement of duplicate readings.

At an early stage in the work, it became apparent that there was a close relationship between haemoglobin and oxygen levels in the cord blood, and Dr. Elizabeth Turnbull was appointed as a Research Fellow to assist in the haematological side of the enquiry.

Oxygen Levels before the onset of Labour

An account of the changes in the oxygen levels in the blood of the vessels of the umbilical cord of the human foetus in the second half of normal pregnancy has been recently published (Walker and Turnbull, 1953). The readings given there were obtained at abdominal delivery under spinal anaesthesia before the onset of labour.

In normal pregnancy the oxygen content of the blood in the umbilical vein appears to fall slowly from 14 vols. per cent at or about the 30th week till the 39th or 40th week when it is about 12 vols. per cent. The fall thereafter is very rapid, and by the 43rd week the content is less than 8 vols. per cent. Since the foetus normally takes 6 to 7 vols. per cent the fall of the content in the artery parallels that in the vein. At the 43rd week the foetus is just able to get enough oxygen from the blood for its needs in a resting state, and returns little or no oxygen to the placenta. There is, therefore, at this stage, no reserve to compensate for any other factor which might interfere with the oxygen supply. When the supply begins to fall, the foetus attempts to maintain the content by increasing the oxygen-carrying power of its blood, that is, it produces more haemoglobin. This attempt at acclimatisation might succeed if the oxygen supply were stable at a new low level, but it is only of limited value to the foetus, as the supply at source continues to decrease and the haemoglobin vainly keeps on rising till, at the 43rd week, it may be as high as 140 per cent. As the content continues to fall and the haemoglobin continues to rise, the blood is becoming less and less saturated with oxygen, as more haemoglobin is carrying less oxygen. The per cent saturation, that is, the amount of oxygen carried relative to the amount which could be carried, is therefore the best measure of the efficiency of the oxygen supply, as it will only begin to fall when the haemoglobin rises in response to anoxia. The per cent saturation is also a better measure than the content of the oxygen available to the foetal tissues, since the tension or head of pressure in the blood is related closely to the per cent saturation. For these reasons, therefore, the oxygen supply reaching the foetus before and after labour will be discussed mainly with reference to the per cent saturation with oxygen of the blood in the umbilical vessels. Content and capacity findings are available in the relevant tables.

The per cent saturation of the blood in the umbilical vein in the latter half of normal pregnancy is 70 per cent at or about the 30th week, 60 per cent at 39 or 40 weeks, and falls to under 30 per cent at 43 weeks. All babies are not, however, even as well oxygenated as this, and the blood of some infants in apparently normal pregnancy is only 30 to 40 per cent saturated even at 40 weeks. A deficient oxygen supply may therefore be seen at any time in the last few weeks of pregnancy, but by the 42nd to 43rd week the supply is dangerously low in almost all
foetuses. Where the pregnancy is complicated by pre-eclampsia, or if abortion has threatened earlier in the pregnancy, the oxygen supply may fail sooner than in normal cases (Walker and Turnbull, 1953).

Oxygen Levels in the Cord Blood after Labour

Oxygen levels in the cord blood at the moment of birth have been studied by many investigators (Eastman, 1930; Haselhorst and Stromberger, 1930; Noguchi, 1937). It is generally agreed that the well-oxygenated infant begins life with its blood about 50 per cent saturated with oxygen. I have shown, however, that the per cent saturation with oxygen of the blood in the umbilical vessels before the onset of labour varies with the week of delivery, and at 42 to 43 weeks most infants begin labour with the blood in the umbilical vein less than 40 per cent saturated.

After easy spontaneous delivery the oxygen levels in the cord blood are seen in Table I and Fig. 1. These cases were delivered without analgesia, and there was no evidence of the passage of meconium by the foetus. In Fig. 1 the per cent saturation of the blood in the umbilical vein of the individual cases is plotted against the range of readings found by Walker and Turnbull (1953) to be normal before the onset of labour. It is seen that the readings after delivery all fall close to the range of readings found before labour begins provided com-

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Menstrual Age (weeks)</th>
<th>Duration Labour (hours)</th>
<th>Oxygen Capacity Vols. per cent</th>
<th>Oxygen Content in Vols. per cent</th>
<th>Per cent Saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>412/51</td>
<td>... 38</td>
<td>... 19</td>
<td>23.8</td>
<td>11.9 0.7</td>
<td>50.0 2.4</td>
</tr>
<tr>
<td>2001/52</td>
<td>... 39</td>
<td>... 38</td>
<td>19.8</td>
<td>11.8 5.4</td>
<td>60.0 28.0</td>
</tr>
<tr>
<td>2821/52</td>
<td>... 39</td>
<td>... 9</td>
<td>22.4</td>
<td>12.7 5.4</td>
<td>57.0 24.0</td>
</tr>
<tr>
<td>1642/52</td>
<td>... 39</td>
<td>... 17</td>
<td>21.8</td>
<td>12.4 6.2</td>
<td>56.9 28.5</td>
</tr>
<tr>
<td>827/51</td>
<td>... 41</td>
<td>... 12</td>
<td>22.0</td>
<td>12.2 –</td>
<td>55.3 —</td>
</tr>
<tr>
<td>806/51</td>
<td>... 41</td>
<td>... 7</td>
<td>23.0</td>
<td>11.7 6.3</td>
<td>50.9 27.6</td>
</tr>
<tr>
<td>805/51</td>
<td>... 41</td>
<td>... 12</td>
<td>23.1</td>
<td>12.2 6.6</td>
<td>52.8 28.8</td>
</tr>
<tr>
<td>813/51</td>
<td>... 41</td>
<td>... 14</td>
<td>20.6</td>
<td>8.9 3.6</td>
<td>42.8 17.4</td>
</tr>
<tr>
<td>2728/52</td>
<td>... 42</td>
<td>... 13</td>
<td>23.6</td>
<td>10.4 4.5</td>
<td>44.0 19.0</td>
</tr>
<tr>
<td>1597/51</td>
<td>... 43</td>
<td>... 11</td>
<td>23.8</td>
<td>3.2 0.2</td>
<td>13.4 0.9</td>
</tr>
</tbody>
</table>

Mean 22.4 10.7 4.3 48.3 19.6

Comparisons are made at the same week of gestation. It is only to be expected that a physiological process like normal labour should have little effect on the oxygen supply to the foetus.

After prolonged or difficult labour, where the foetus is born in a severe state of asphyxia, very low levels of oxygen are found in the cord blood (Eastman, 1932; Wilson et al., 1937), but no

**Fig. 1**

Per cent saturation with oxygen of blood in the umbilical vein at spontaneous delivery (without evidence of meconium staining).
FOETAL ANOXIA

Per cent saturation with oxygen of blood in the umbilical vein after prolonged or difficult labour (without evidence of meconium staining).

Evidence is available of the likely effect of difficult labour itself.

In Table II are seen the oxygen levels in the blood of the cord vessels in a series of infants born after labours lasting from 11 to 89 hours, and ending with operative delivery under spinal anaesthesia. There was, however, no evidence of meconium in the liquor. In Fig. 2 the readings of the per cent saturation with oxygen of the blood in the umbilical vein are plotted against the range of readings found to be normal before the onset of labour. It is seen that there is little or no difference when compared with the levels found before the onset of labour. In the absence of meconium, therefore, the oxygen supply to the foetus during prolonged or difficult labour does not appear to be interfered with to any extent.

During prolonged or difficult labour, however, the foetus frequently shows clinical evidence of distress, the liquor is heavily stained with meconium, and at birth the infant's skin may be covered with a golden-green film. The oxygen levels in such cases are usually very low.

**Recent meconium staining.** The oxygen levels in a series of cases where the foetus had recently passed meconium in labour or was found to be covered with meconium at delivery are seen in Table III. In Fig. 3 the readings of the per cent saturation of the blood in the umbilical vein are plotted against the range found to be normal before the onset of labour. Two important points are seen.

(a) The per cent saturation of the blood in the umbilical vein is at or below 30 per cent no matter the stage of gestation.

(b) This level of 30 per cent, the "distress level", as I call it, is around the level of oxygen supply at which most foetuses in the 43rd week normally begin labour.

The information shown by Tables II and III and Figs. 2 and 3 suggests that prolonged or difficult labour may have only little effect on the

| Case No. | Menstrual Age (weeks) | Type of Delivery | Duration Labour (hours) | Oxygen Capacity Vols. per cent | Oxygen Content Vols. per cent | Per cent Saturation
<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Vein</td>
<td>Artery</td>
<td>Vein</td>
</tr>
<tr>
<td>2969/50</td>
<td>. . 39</td>
<td>Low Forceps</td>
<td>17</td>
<td>24.2</td>
<td>13.9</td>
<td>5.9</td>
</tr>
<tr>
<td>525/51</td>
<td>. . 40</td>
<td>Low Forceps</td>
<td>19</td>
<td>21.6</td>
<td>11.6</td>
<td>1.0</td>
</tr>
<tr>
<td>1651/51</td>
<td>. . 40</td>
<td>Diff. Mid Forceps</td>
<td>55</td>
<td>23.1</td>
<td>8.6</td>
<td>2.8</td>
</tr>
<tr>
<td>2820/52</td>
<td>. . 40</td>
<td>Diff. Mid Forceps</td>
<td>89</td>
<td>22.2</td>
<td>12.1</td>
<td>6.0</td>
</tr>
<tr>
<td>2765/50</td>
<td>. . 41</td>
<td>Mid Forceps</td>
<td>11</td>
<td>19.8</td>
<td>10.0</td>
<td>4.0</td>
</tr>
<tr>
<td>3124/50</td>
<td>. . 41</td>
<td>LUCS</td>
<td>21</td>
<td>24.2</td>
<td>13.3</td>
<td>7.3</td>
</tr>
<tr>
<td>506/51</td>
<td>. . 43</td>
<td>Diff. Mid Forceps</td>
<td>46</td>
<td>24.8</td>
<td>6.8</td>
<td>3.6</td>
</tr>
<tr>
<td>3027/52</td>
<td>. . 43</td>
<td>LUCS</td>
<td>34</td>
<td>23.4</td>
<td>6.5</td>
<td>0.9</td>
</tr>
</tbody>
</table>

| Mean     | . . 22.9              | 10.4            | 3.9                     | 45.5             | 16.8             |
### Table III

**Oxygen in the Umbilical Vessels when there is Recent Meconium Staining**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Menstrual Age (weeks)</th>
<th>Type of Delivery</th>
<th>Duration of Labour (hours)</th>
<th>Degree of Meconium Staining</th>
<th>Oxygen Capacity Vols. per cent</th>
<th>Oxygen Content Vols. per cent</th>
<th>Per cent Saturation</th>
</tr>
</thead>
<tbody>
<tr>
<td>2683/50</td>
<td>. . 40</td>
<td>LUCS</td>
<td>32</td>
<td>Light</td>
<td>21.8</td>
<td>6.6</td>
<td>30.2</td>
</tr>
<tr>
<td>667/53</td>
<td>. . 41</td>
<td>LUCS</td>
<td>94</td>
<td>Light</td>
<td>27.2</td>
<td>7.5</td>
<td>31.4</td>
</tr>
<tr>
<td>2993/50</td>
<td>. . 41</td>
<td>LUCS</td>
<td>12</td>
<td>V. Heavy</td>
<td>22.2</td>
<td>3.9</td>
<td>17.6</td>
</tr>
<tr>
<td>1596/53</td>
<td>. . 42</td>
<td>LUCS</td>
<td>Elect.</td>
<td>V. Heavy</td>
<td>24.6</td>
<td>7.7</td>
<td>31.4</td>
</tr>
<tr>
<td>5/52</td>
<td>. . 43</td>
<td>LUCS</td>
<td>114</td>
<td>V. Heavy</td>
<td>25.2</td>
<td>2.0</td>
<td>20.8</td>
</tr>
<tr>
<td>1447/51</td>
<td>. . 43</td>
<td>LUCS</td>
<td>18</td>
<td>V. Heavy</td>
<td>23.2</td>
<td>4.3</td>
<td>18.7</td>
</tr>
<tr>
<td>2291/51</td>
<td>. . 43</td>
<td>LUCS</td>
<td>49</td>
<td>Light</td>
<td>25.8</td>
<td>5.4</td>
<td>20.8</td>
</tr>
<tr>
<td>991/51</td>
<td>. . 43</td>
<td>LUCS</td>
<td>62</td>
<td>Moderate</td>
<td>25.6</td>
<td>3.0</td>
<td>11.6</td>
</tr>
<tr>
<td>3054/52</td>
<td>. . 43</td>
<td>LUCS</td>
<td>35</td>
<td>Moderate</td>
<td>25.6</td>
<td>6.6</td>
<td>25.8</td>
</tr>
<tr>
<td>1506/51</td>
<td>. . 43</td>
<td>S.D.</td>
<td>16</td>
<td>Light</td>
<td>24.6</td>
<td>7.5</td>
<td>30.6</td>
</tr>
<tr>
<td>3001/51</td>
<td>. . 43</td>
<td>LUCS</td>
<td>Elect.</td>
<td>Light</td>
<td>25.6</td>
<td>8.2</td>
<td>32.1</td>
</tr>
</tbody>
</table>

Mean . . 24.7 5.7 1.3 23.2 4.8

**Summary of Oxygen Findings**

1. The oxygen supply to the human foetus in a clinically normal pregnancy falls gradually up to the 40th week and rapidly thereafter.
2. Most foetuses at 40 weeks are well oxygenated and have nearly 100 per cent reserve, but in some the supply is low, and the reserve is correspondingly reduced.
3. The number of foetuses with a poor oxygen supply increases in the next few weeks until, by the 43rd week, all foetuses have a low oxygen supply and practically no reserve. They are forced at the best to clear all oxygen from their blood to obtain enough for their resting needs.
4. The well-oxygenated baby can stand the effect of labour of any kind without becoming short of oxygen, but the foetus, already with a deficient supply, is likely to experience deficiency of oxygen during labour and to declare this deficiency by passing meconium.
5. When meconium is passed, the saturation of the blood in the umbilical vein is at or below 30 per cent, and the foetus is not obtaining enough oxygen to survive indefinitely.

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**FIG. 3**

Per cent saturation with oxygen of blood in the umbilical vein where the foetus has recently passed meconium.
OBSTETRIC DEATH, STILLBIRTH, AND FIRST WEEK NEONATAL DEATHS
PER 1,000 TOTAL BIRTHS — 11,051 BOOKED (ALL PARITIES)

Obstetric Death, Stillbirth, and first-week Neonatal Death Rates per 1,000 total births.

CLINICAL EFFECTS OF ANOXIA

Since oxygen deficiency becomes more common and more severe after term, one might expect to find, after that time, an increasing incidence of foetal deaths due to anoxia, and a greater incidence of clinical evidence of distress during labour.

Obstetric Death

In Fig. 4 are seen the obstetric death, stillbirth and first-week neonatal death rates per 1,000 total births in relation to the duration of pregnancy in 11,051 booked patients of all parities, delivered from 1948 to 1952. The obstetric death rate is 11.7 and 11.5 for deliveries in the 40th and 41st weeks, but rises steadily thereafter till for deliveries at or after the 43rd week it is 28.5. The rise in rate after 41 weeks is due mainly to a rise in stillbirths as has been shown by others (McKiddie, 1949; Gibberd, 1952), but it is seen that the first-week death rate rises from 3.8 at the 41st week to 7.1 for deliveries in the 43rd and subsequent weeks. The risk of neonatal death is definitely greater for the post-mature infant than for the infant delivered during the 40th or 41st weeks.

Fig. 5 shows that the rise in the death rate after the 40th and 41st weeks is due primarily to a rising incidence of unexplained deaths, that is, death of the foetus where no clinical abnormality of pregnancy or labour was present which might
explain why the baby died. Deaths of this type are uncommon at 40 to 41 weeks, but by 43 weeks in primigravidae and in multigravidae they are the highest single cause of death. In primigravidae, there is also a rise in incidence of deaths considered to be directly due to difficult labour (in infants presenting by the vertex). The death rate from "other causes" is also raised in the 42nd and 43rd weeks. This is not surprising as the anoxic foetus is less likely to be able to withstand the further anoxic effects of pre-eclampsia, cord prolapse or breech delivery.

In the above series, it is possible to calculate death rates in each week as the duration of pregnancy of most patients delivered over the 5-year period 1948-1952 is accurately known. Exclusion of obstetric deaths in cases where the gestational age is uncertain does not influence the shape of the curves (most are in premature foetuses). The actual number of deaths, however, is small, and some of the curves are of doubtful statistical significance (see Note on p. 179). In view of this, all stillbirths and first-week deaths occurring in 10,785 primigravidae and 12,723 multigravidae booked for and delivered in the Aberdeen Maternity Hospital from 1938 to 1951 have been studied and a clinical cause assigned to each death. The proportion of deaths from each cause in relation to the total deaths occurring at each week of gestation has been calculated. Death rates at each week could not be calculated as accurate information is not available of the gestational age of all infants delivered over those years.

Table IV shows that the proportion of "unexplained deaths" (in babies over 5½ pounds) is seen to rise steadily as pregnancy is prolonged, until by the 42nd and subsequent weeks at least one-third of all deaths are "unexplained". Of all obstetric deaths in both primigravidae and multigravidae, about the same proportion is unexplained (14·4 and 12·8 per cent).

At postmortem, 70 per cent of these unexplained deaths show the lesions of anoxia alone, and in most of the remainder the likely lesions of anoxia are masked by maceration. In a study of causes of stillbirth occurring in Queen Charlotte's Hospital in 1948 to 1950, Gibberd (1952) has shown that the stillbirth rate for delivery from the 41st to the 45th week is nearly 3 times that for delivery at the 37th to 40th week, and that the excess after term is due entirely to a great rise in the incidence of unexplained anoxic deaths. Most other investigators consider that the excess mortality after term is due mainly to prolonged or difficult labour (Ballantyne and Browne, 1922; Clayton, 1941, 1953; Rathburn, 1943; Latto, 1951). In this connexion I would like to stress that, if a foetus dies of anoxia early in the first stage of a labour which lasts for 3 days and ends with a difficult forceps delivery, the death must surely be classified as "unexplained", and not as due to prolonged labour or traumatic delivery.

In Table V, however, is seen the proportion of deaths assessed as directly due to difficult labour in the 13-year series. It will be noted that in primigravidae the proportion of deaths (14·0 per cent) from this cause is much higher than in multigravidae (4·3 per cent). The proportion occurring in each week of gestation is fairly steady up to the end of the 41st week, but after that time, in primigravidae, 37·1 per cent of all deaths are due to this cause.

### Table IV

<table>
<thead>
<tr>
<th>Week of Delivery</th>
<th>37</th>
<th>38-39</th>
<th>40</th>
<th>41</th>
<th>42+</th>
<th>Uncertain</th>
<th>Total</th>
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<tr>
<td><strong>Primigravidae</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per cent Deaths Unexplained</td>
<td>1·7</td>
<td>11·3</td>
<td>16·7</td>
<td>24·4</td>
<td>32·4</td>
<td>15·0</td>
<td>14·4</td>
</tr>
<tr>
<td>Total Deaths</td>
<td>173</td>
<td>71</td>
<td>30</td>
<td>45</td>
<td>105</td>
<td>40</td>
<td>464</td>
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<tr>
<td><strong>Multigravidae</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Per cent Deaths Unexplained</td>
<td>1·8</td>
<td>19·6</td>
<td>11·5</td>
<td>30·2</td>
<td>35·6</td>
<td>11·1</td>
<td>12·8</td>
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<tr>
<td>Total Deaths</td>
<td>228</td>
<td>56</td>
<td>26</td>
<td>53</td>
<td>59</td>
<td>45</td>
<td>467</td>
</tr>
</tbody>
</table>

**Journal of Obstetrics and Gynaecology**
Obstetric death due to difficult labour is of two main types. The first, "traumatic", usually seen in association with mechanical difficulty and associated with tearing of the tentorium and haemorrhage; the second, "anoxic", usually associated with prolonged dysfunction labour and showing at autopsy the lesions of anoxia alone. At 40 weeks 13·3 per cent of all obstetric deaths are "traumatic", 3·3 per cent "anoxic". After the 41st week 13·3 per cent of deaths are "traumatic", but 23·8 per cent are "anoxic". The rise in rate and proportion of deaths due to difficult labour after term is thus seen to be due mainly to a rise in deaths from anoxia. Death from anoxia in association with difficult labour is extremely uncommon until after term, and 83 per cent of all such deaths occur after the end of the 41st week.

The Incidence of Difficult Labour

The rise in incidence of deaths directly due to difficult labour is to some extent explained by the increasing incidence of difficult labour after term. Normal labour is defined as spontaneous vertex delivery in under 24 hours, and all other labours are "difficult". Major difficult labours are those with undue prolongation of labour with deterioration of maternal condition, or Caesarean section or difficult forceps extraction because of the nature of the labour itself. (Cases of malpresentation, Caesarean section or forceps for foetal distress, maternal disease, etc., are excluded.) In Table VI it is seen that the incidence of difficult labour in primigravidae rises steadily from the 37th to the 44th weeks. The rise is due entirely to an increasing incidence of a major degree of difficulty. At the 40th week only 3·1 per cent of all cases have a major degree of difficulty in labour or delivery, but by the 44th week the figure rises to 13·7 per cent. This rise is due mainly to an increase in uterine dysfunction of severe degree, but mechanical difficulty also increases and is made clinically more important by the less efficient contractions.

The importance of a rising incidence of difficult labour after term has been noted previously by Rathburn (1943), DeLee and Greenhill (1947), Beruti and Roust (1948), Reenkola (1948), Stewart (1952), and Clayton (1953).

Clinical Evidence of Foetal Distress

The findings shown earlier in this paper confirm the importance of anoxia as a cause of obstetric death after the 40th week of gestation, but all anoxic foetuses do not die, and more information can be obtained from a study of foetal distress. For the purposes of this investigation I have defined foetal distress as gross irregularity of the foetal heart, or slowing to or

<table>
<thead>
<tr>
<th>Week of Delivery</th>
<th>. . .</th>
<th>-37</th>
<th>38-39</th>
<th>40</th>
<th>41</th>
<th>42</th>
<th>43</th>
<th>44+</th>
<th>Uncertain</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primigravidae</td>
<td>. . .</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>15·5</td>
<td>16·7</td>
<td>15·5</td>
<td>37·1</td>
<td></td>
<td></td>
<td></td>
<td>14·0</td>
<td></td>
</tr>
<tr>
<td>Multigravidae</td>
<td>. . .</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>4·3</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>5·0</td>
<td>11·5</td>
<td>11·1</td>
<td>13·6</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TABLE VI

Per cent Incidence of Difficult Labours (Vertex Delivery) in Primigravidae

<table>
<thead>
<tr>
<th>Week of Delivery</th>
<th>. . .</th>
<th>-37</th>
<th>38-39</th>
<th>40</th>
<th>41</th>
<th>42</th>
<th>43</th>
<th>44+</th>
<th>Uncertain</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>All Difficult Labour</td>
<td>. . .</td>
<td>15·4</td>
<td>19·9</td>
<td>24·2</td>
<td>29·8</td>
<td>32·3</td>
<td>35·5</td>
<td>34·5</td>
<td>21·8</td>
<td>25·9</td>
</tr>
<tr>
<td>All Major</td>
<td>. . .</td>
<td>0·8</td>
<td>3·5</td>
<td>3·1</td>
<td>6·7</td>
<td>9·4</td>
<td>13·0</td>
<td>13·7</td>
<td>2·7</td>
<td>5·7</td>
</tr>
<tr>
<td>Minor</td>
<td>. . .</td>
<td>14·6</td>
<td>16·4</td>
<td>21·1</td>
<td>23·1</td>
<td>22·9</td>
<td>22·5</td>
<td>20·8</td>
<td>19·1</td>
<td>20·2</td>
</tr>
<tr>
<td>Cases at Risk</td>
<td>. . .</td>
<td>254</td>
<td>573</td>
<td>693</td>
<td>813</td>
<td>501</td>
<td>200</td>
<td>58</td>
<td>412</td>
<td>3,504</td>
</tr>
</tbody>
</table>
40th week (in primigravidae). When distress is due to prolonged labour or to pressure on the foetal head in the pelvis (combined in Fig. 7) the rising incidence after term is due to a great extent to the rising incidence of difficult labour after that time. Distress due to cord complications rises in incidence since the anoxic foetus is less able to withstand interference with the cord circulation. Distress occurring late in the second stage (slowing of the foetal heart) with the head on the perineum is somewhat more frequent after that time. The most marked and constant rise, however, is in those cases of distress clinically unexplained, and after the 41st week this is the greatest single “cause” of distress (10·5 and 10·3 per cent of all cases at the 43rd and 44th week respectively).

In multigravidae, distress rates rise also after the 40th week, but in view of the greatly diminished incidence of difficult labour it is not valid to compare the total incidence of distress with that in primigravidae. In Fig. 8 the incidence of cases of distress clinically “unexplained” is seen in primigravidae and in multigravidae. The behaviour and incidence with prolonged

below 100 beats per minute, or the passage of meconium in presentations other than breech. According to the oxygen findings, it would appear that the foetus will pass meconium only when it is short of oxygen. Aetiologically, cases of distress therefore fall into two main groups, those where meconium is passed, and those where heart signs are present alone.

The incidence of foetal distress in all labours is seen in Fig. 6. It is low before and at the 40th week, at which time only 8·4 per cent of babies show distress. The rate rises steadily till at the 44th week evidence of distress is shown in 25·9 per cent of labours. In “normal” labours the rate is only a little lower, the rise beginning in the 41st week and by the 44th week it is 21·1 per cent. The distress rate in cases of difficult labour (vertex presenting) is, however, high. At 40 weeks 13 per cent of foetuses undergoing difficult vertex labour show clinical evidence of distress, and, at the 43rd week, 39·4 per cent.

Incidence by Clinical Cause. Each clinical history has been studied and a clinical cause assigned to the distress. In Fig. 7 it is seen that the incidence from all causes is low up till the
FOETAL ANOXIA

171

a great increase in the incidence of cases showing meconium staining which, though clinically unexplained, is due probably to a marked deficiency in the oxygen supply to the foetus.

THE CLINICAL PROBLEM

Experimental and statistical evidence suggests strongly that the fall in the oxygen supply to the human foetus in late pregnancy is the main factor in the rising stillbirth rate after 40 weeks, and in the greatly increased incidence of foetal distress in labour. The passage of meconium (in presentations other than breech) is evidence that the foetus is short of oxygen. In view, therefore, of the high incidence of meconium staining in labour after the 40th week, many infants must suffer long periods of relative anoxia before delivery. Even if they ultimately survive, they may be damaged. For example, spastic diplegia is known to occur not uncommonly after such a syndrome where there has been no question of trauma at delivery.

Many clinicians have believed for a long time that “post-maturity” is potentially dangerous. I hope that this analysis and the oxygen findings will show that the problem is that of the failing oxygen supply in late pregnancy, a problem which is already present at the 40th week in a few cases, but becomes more important and more frequent the further pregnancy is prolonged. As pregnancy becomes prolonged after term, the “pregnancy process” begins to regress. The maternal weight drops, the maternal uterine blood flow lessens, the foetus becomes progressively short of oxygen, and its weight gain slows and may cease. In the extreme case pregnancy as a physiological process is over, but labour fails to occur and the foetus cannot escape death. This is the clinical history of an extreme case.

2751/48. This was a primigravida, aged 35, and with a menstrual cycle of 34/25-28. Pregnancy was normal, specialist antenatal care being given from the 8th week. The weight curve in the last few weeks is seen in Fig. 9.

At the 44th week, the uterus was wrapped round the child, the foetal head was well into the maternal pelvis. On pelvic examination the cervix was thinned out over the head, the external os was high and in the sacral position, soft, and one finger could easily be inserted. Elective Caesarean section was performed under spinal anaesthesia. At operation no liquor was found, but the
Clinically, however, the main problem is in primigravidae, and becomes important after the end of the 41st week. What then is the size of that problem?

Twenty-one per cent of all primigravidae are delivered after the end of the 41st week. In this 21 per cent are:

- 51 per cent of all unexplained deaths (mature).
- 83 per cent of all anoxic deaths in difficult labours.
- 40 per cent of all traumatic deaths in difficult labours.
- 43 per cent of all major difficult labours.
- 36 per cent of all Caesarean sections for difficult labour.

Have we got to take this whole 21 per cent into serious consideration, or is there any further guide to aid in selection of cases more likely to be affected than others?

I agree that in this extreme form the syndrome is uncommon, but all prolonged pregnancies show lesser degrees of this picture.

How then can the clinician attack the problem of late pregnancy anoxia? In the present state of our knowledge it is impossible to select those cases in primigravidae likely to show a deficient oxygen supply to the foetus at the 40th or 41st weeks, partly because nearly 50 per cent of all pregnant women are delivered during those 14 days. There are, however, certain factors like pre-eclampsia or threatened abortion early in pregnancy which predispose to an early fall in the oxygen supply. Patients with these clinical abnormalities are better delivered at least by the end of the 40th week, and may with advantage be delivered a week or so earlier.

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Gravida is no more likely to go post-mature. If the mother is over 25, there is a much greater risk that her foetus will die of anoxia or will die because of difficult labour. The foetus is also much more likely to be called upon to face a difficult labour. All risks increase the longer pregnancy is prolonged. The problem of obtaining the first child alive is particularly important in the older primigravida in view of the greater value attached to the child by the older woman and a less likely chance of a family of reasonable size if the first child is stillborn.

**Clinical Management of Difficult Labour**

With the further clinical guidance given by a study of maternal age, I would suggest the following broad principles in management of labour to minimize the risk of death due directly or indirectly to anoxia.

Prevention of death from birth trauma is primarily a problem of obstetric skill at delivery, but is complicated after the 41st week by the falling oxygen supply to the foetus. A foetus, already anoxic and passing meconium, or already suffering local cerebral anoxia due to pressure, and manifesting this by slowing of its heart rate, may be killed by forceps delivery which a foetus, not already anoxic, would easily withstand. Careful assessment of the likely degree of difficulty should be made before delivery is begun, and especially in the older woman Caesarean section may be preferable.

Death due to anoxia in prolonged labour is usually preceded by a long period during which the liquor is stained with meconium. Frequently the membranes are intact, however, and meconium may not be visible. In cases of prolonged labour after the 41st week, it may help to tap the hindwaters. If the liquor is stained with meconium the foetus is already anoxic. The presence of meconium in the liquor indicates that the foetus has been short of oxygen, but unless it is becoming thicker or more yellow it does not, unfortunately, indicate whether the supply is deteriorating even further, or whether it is remaining just under distress levels. The foetal heart rate is not a good guide in such cases, as it frequently stops without any preceding period of slowing or irregularity. In the management of such cases it helps to consider the age of the mother. If she is over 30, it is probably best to perform Caesarean section for the sake of the child as soon as the presence of meconium is noted. In young primigravidae, dysfunction labour is uncommon and foetal distress is seen less often, but even here, if labour is unduly prolonged and there is passage of meconium, Caesarean section should be performed earlier.
than the duration of labour itself, or the condition of the mother, might indicate. During the waiting period, oxygen should be given to the mother by B.L.B. mask, as this will almost certainly improve the oxygen supply to the foetus (Landais, 1892; Waters and Harris, 1931).

**Anaesthesia.** It is important to choose the safest anaesthetic for operative delivery of the already anoxic foetus. In the present state of our knowledge it appears that, for the foetus, spinal, local or caudal is best (Taylor et al., 1951; Watts et al., 1951; Batten, 1943). Any fall in maternal blood pressure which might occur can be controlled with Methidrine, and oxygen can be given to the mother until the child is delivered (Batten, 1943). During the induction period of most general anaesthetics, there is a time when the maternal oxygen supply is impaired (McClure et al., 1939, 1948). If there should be difficulty in induction and maternal cyanosis should occur, the saturation of the mother's arterial blood drops only to some 80 per cent, but the oxygen tension drops by half, and very little oxygen would be transferred to the foetus during this time. Further, the infant born after the use of general anaesthesia may be handicapped by the presence of depressant anaesthetic gases in its own blood (Smith and Barker, 1942; Taylor et al., 1951).

**Resuscitation.** I would like briefly to discuss the problem of resuscitation of the anoxic infant. Even though the rise in the obstetric death rate after term is due mainly to a rise in stillbirths, many of the babies who are lost in the first few days could be saved by expert resuscitation at birth. When operative delivery is undertaken because of foetal distress, expert resuscitation may make the difference between success and failure. The natural person to resuscitate the infant is the paediatrician (if specially trained in resuscitation methods), who will care for it in the first few days of life. Since 1948 we have had the benefit of an increased paediatric staff who have taken a special interest in this problem and in the last few years very few babies, delivered after the 40th week, have been lost neonatally. The techniques of resuscitation are outwith the scope of this discussion, but I would like to mention the importance of emptying the stomach of the infant who has been anoxic at birth. Anything up to 20 ml. of sticky meconium-stained mucus may be present and, if not removed, may well be inhaled during the respiratory difficulties of the first few days (Ahvenainen, 1953).

**The Problem of Unexplained Deaths**

In Table VII are seen the causes of obstetric death in primigravidae after the 41st week of gestation. It is taken from the 1939-51 series and demonstrates the risks of prolonged pregnancy where obstetric care has not been specially directed at the problem.

<table>
<thead>
<tr>
<th>Cause of Obstetric Death after the 41st week of Pregnancy in 10,758 Primigravidae</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Difficult Labour (Vertex)</td>
<td>Anoxia 23.8</td>
</tr>
<tr>
<td>Malpresentation: Cord</td>
<td>Trauma 13.3</td>
</tr>
<tr>
<td>Deformity</td>
<td></td>
</tr>
<tr>
<td>Unexplained (Mature)</td>
<td></td>
</tr>
<tr>
<td>Toxaemia</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td></td>
</tr>
</tbody>
</table>

(105 cases)

It will be seen that the suggestions outlined for the management of difficult labour might save approximately half of the babies lost after the 41st week. Better care in labour, however, cannot influence death rates from the other causes, and especially those "unexplained". Of all those unexplained deaths shown here, 30 per cent occur before the onset of labour, 50 per cent occur during labour, and 20 per cent are neonatal. The large majority of the intrapartum deaths occur early in the first stage of labour without warning, or in the second stage without sufficient warning to allow the child to be saved. Neonatal deaths were preceded by a short period during which meconium was passed in the second stage of a short and easy labour. Rapid delivery might save a very few of those babies but the incidence of "unexplained" deaths could only be reduced to any degree if labour and delivery could be achieved at a period of gestation at
which the oxygen supply to the foetus is still good. If labour could be safely and successfully induced at this time, the number of deaths from anoxia due to prolonged labour would also, of course, greatly diminish without recourse to Caesarean section.

**Induction of Labour**

It has been suggested that induction of labour might fail to influence the obstetric death rate in prolonged pregnancy, or might, in fact, create new dangers (Solth and Müller, 1949). In our experience, surgical induction of labour by artificial rupture of the hindwaters seems to be a safe and easy procedure, and does not appear to predispose to prolonged labour. In view of this, the indications for induction have been widened in recent years, and, especially since the work of McKiddie in 1948-49, induction has been performed in many instances for post-maturity alone.

In Table VIII is seen the induction rate in primigravidae since 1948 according to the week in which delivery occurred. From 1948 to 1951 induction was performed for the more commonly accepted indications, for example, pre-eclampsia, but it will be noted that already we were beginning to induce labour more often after the 42nd week. In 1952, the induction rates doubled as indications were widened. In the early weeks, for example, milder cases of pre-eclampsia were induced, but in the 42nd and 43rd weeks many cases were being induced for "post-maturity". The results from the point of view of foetal mortality were very good (see Table IX). A study however, of the incidence of difficult labour suggested that the number of cases of major difficult labour was becoming less, and this seemed to be associated with the rising incidence of induction after the 40th week. It was decided, therefore, as a controlled clinical procedure, to increase the number of inductions after the 41st week, not only because of the likely influence on the obstetric death rate, but to see if cases induced at that time would have an easier and shorter labour.

In the Aberdeen population 21 per cent of primigravidae are delivered after the 41st week. It was obviously difficult to induce all cases. The obstetric death rates from unexplained causes and from difficult labour, and the incidence of difficult labour itself, are considerably less amongst younger women.

If the high risk group is defined as those cases who are aged 25 and over, and who go past the 41st week, it amounts to only 8 per cent of all primigravidae. It was therefore decided that all primigravidae over the age of 25 should be admitted to hospital and surgical induction performed if labour had not begun some 5 to 7 days after the estimated date of delivery, provided those dates seemed accurate and the infant was of reasonable size. Routine induction, therefore,

**Table VIII**

*Incidence (per cent) Surgical Induction of Labour in Primigravidae*

<table>
<thead>
<tr>
<th>Week of Delivery</th>
<th>Uncertain</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1948-51</td>
<td>7.5</td>
<td>7.5</td>
</tr>
<tr>
<td>Total Cases</td>
<td>3504</td>
<td></td>
</tr>
<tr>
<td>1952</td>
<td>14.0</td>
<td></td>
</tr>
<tr>
<td>Total Cases</td>
<td>860</td>
<td></td>
</tr>
<tr>
<td>January to June, 1953</td>
<td>23.4</td>
<td></td>
</tr>
<tr>
<td>Total Cases</td>
<td>461</td>
<td></td>
</tr>
</tbody>
</table>
applied to only 8 per cent of our booked primigravidae. Artificial rupture of the hindwaters was performed in those cases without regard for the position or condition of the cervix, or the position or station of the head.

**RESULTS OF TREATMENT**

*In Aberdeen Maternity Hospital*

Over the last few years in booked primigravidae there has been a general improvement in results due to a better understanding of the problem, improved standards of care in labour, improved methods of resuscitation and, recently, following more frequent induction of labour. The figures shown for the first half of 1953 are based on a very small number of cases, but they are promising and the improvement has been progressive.

The obstetric death rate (Table IX) for deliveries after the 41st week fell by 15 per 1,000 total births soon after the importance of late pregnancy anoxia was first realized, and by another 10 per 1,000 during the period when the incidence of induction was increasing.

The effect on major difficult labour appears to confirm our impression that induction does not predispose to difficult labour, and that it may, in fact, prevent its occurrence (Table X). In the first half of 1953, the incidence of major difficult labour in deliveries after the 41st week was the same as the incidence in deliveries in the 40th week.

The incidence of Caesarean section has not fallen (Table XI). Recently Caesarean section for difficult labour has become less frequent, but the fall has been more than balanced by a rise in the number of Caesarean sections performed for foetal distress. Up to 1952 it is possible that a slight increase in the Caesarean section rate, due mainly to the indications of foetal distress and advancing maternal age, may have played the major part in lowering the death rates of postmature infants, but if the incidence of major difficult labour should persistently remain low as a consequence of more frequent induction of labour, the overall Caesarean section rate after the 41st week will remain at its present low level.

**Table XI**

**Caesarean sections after the end of the 41st week of Pregnancy in Primigravidae**

<table>
<thead>
<tr>
<th>Years</th>
<th>Difficult Labour (per cent)</th>
<th>All Other Reasons (per cent)</th>
<th>Total Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1948-49</td>
<td>..</td>
<td>2.5</td>
<td>1.0</td>
</tr>
<tr>
<td>1950-51</td>
<td>..</td>
<td>3.0</td>
<td>1.4</td>
</tr>
<tr>
<td>1952-June, 1953</td>
<td>2.3</td>
<td>2.7</td>
<td></td>
</tr>
<tr>
<td>January to June, 1953</td>
<td>3.3</td>
<td>3.3</td>
<td></td>
</tr>
</tbody>
</table>

When routine induction is performed, "foetal distress", manifest by heavy meconium staining of the liquor, may be found. Especially in the older primigravidae the decision is usually made to perform an elective Caesarean section in the interests of the child. The typical picture is seen in the following case.

Mrs. H., a primigravida, aged 37 years, had been married for 12 years. Routine antenatal care was undertaken from the 8th week of pregnancy by a senior consultant, and the pregnancy was clinically normal. There was a steady maternal weight gain till the 39th week of pregnancy, from which time the weight remained unchanged till the 41st week and there was a loss of 2 pounds by the 42nd week. Routine surgical induction of labour was performed at the beginning of the 42nd week, and 15 ounces of liquor, dark green and thick with meconium, were drained from the uterus. Elective lower segment Caesarean section was performed under spinal anaesthesia. The child, a female, weighing 8 pounds 2 ounces, was in good condition at birth, responded to routine resuscitation, and survived without incident.

The oxygen capacity of the foetal blood was high, being 24.6 vols. per cent. The saturation of the blood in the umbilical vein was 31 per cent, and of the blood in the artery 6 per cent. This foetus was already short of oxygen and had no reserve to allow it to withstand labour without serious and probably fatal anoxia.

*In Aberdeen City*

A very high standard of work and careful management of each case is necessary to obtain
the best results. Evidence of the effect of improving standards of obstetric care is seen in Fig. 11 which illustrates the trends in the stillbirth rate in the City of Aberdeen for the years from 1939 to 1952. Each stillbirth has been studied and a clinical cause assigned to the death.

The situation in Aberdeen is favourable for such a clinical investigation as, in recent years, about 95 per cent of primigravidae and 70 per cent of multigravidae are booked for confinement under the care of specialists in hospital. Booked cases attend the antenatal clinic, and each clinic is conducted under the supervision of a consultant obstetrician, so that the standard of observation and of recording is uniformly high. A very good liaison exists with domiciliary midwives and general practitioners, so that cases under their care are referred quickly if abnormalities develop, and full and accurate information is available if a stillbirth should occur, so that the causes of death in domiciliary cases can also be accurately assessed. A fall of 50 per cent has occurred in the stillbirth rate from about 40 per 1,000 before the war to 20 in the last few years. The interplay of the many factors responsible for this has been discussed elsewhere (Baird et al., 1953; Baird and Walker, 1954), but I wish to concentrate on deaths of mature infants from birth trauma (of all forms) and “unexplained” causes. For the sake of simplicity the rates for both causes are combined in a single line in the diagram since they run parallel and close to each other.

The fall in the period 1939 to 1945 was due probably to the increasing proportion of cases confined in hospital, as a result of expansion of accommodation. The rise in 1946 to 1948 occurred mainly because of a great increase in the birth rate affecting first, second and third births, and due to an accumulation of war-postponed pregnancies. The patients were, therefore, older and in addition many more were confined in their own homes. As the stillbirth rate in both cause groups rises with maternal age, and as the emergencies of labour cannot be dealt with as quickly, and as effectively, under domiciliary conditions, a rise in the rate in that period is only to be expected. The fall since 1949 is due to the fact that nearly all primigravidae are confined in hospital (the falling birth rate made this possible) and to the new methods of management described. Deaths from birth trauma at all stages of gestation have become uncommon; in 1952, for example, there were no

![Stillbirth Rates per 1,000 Total Births in Aberdeen City, 1939-1952 by Clinical Cause](image-url)
obstetric deaths from birth trauma in deliveries of 1,000 booked primigravidae. The improvement in “unexplained deaths” is due almost exclusively to the elimination of stillbirths after the 40th week of gestation. Further fall in the stillbirth rate is likely to be slow, as other causes are not so amenable to surgical skill, and there is convincing evidence that they are related to biological and environmental factors, the removal of which would be difficult and slow.

DISCUSSION

Since the end of the war, comprehensive research has been conducted in Aberdeen into the causes and prevention of stillbirth and early neonatal death. My own particular interest, since joining the staff of the University Department in January, 1948, has been to study causes of death in babies delivered just before, at, or after term. Of special interest were those cases where anoxia was responsible for death of a foetus fully grown and otherwise normal.

In this paper I have stressed the importance of a falling oxygen supply as pregnancy becomes prolonged as a causal factor in many of those deaths. Quantitative estimations of oxygen lend support to those who, on clinical grounds, have held that post-maturity is potentially dangerous. Unfortunately, of course, the oxygen supply to the foetus cannot be measured as a practical guide to treatment in individual cases, and so treatment must be based on careful clinical assessment.

I have mentioned a few of the clinical guides that have been found helpful in planning the conduct of each case. The maternal weight is taken routinely at antenatal visits, and there are, of course, many variations of weight curves in individual pregnancies. However, a steadily rising curve with a halt and then a fall of some pounds at or after term is probably a significant sign in the type of case we are discussing. This valuable sign may be masked by fluid retention due to mild pre-eclampsia which is, of course, common at this time. Gradual disappearance of the liquor amnii and the picture of a firmer and more easily palpable foetus, as described by Wrigley (1946) and Mills (1953), does occur. Adequate liquor does not, however, preclude a dangerous degree of anoxia, as exemplified by the second case history.

As a result of our experience in the last few years, we have formulated certain general principles of management which we think are a great help when used in conjunction with a careful clinical assessment of each case.

(1) Induce labour before 40 weeks in cases of pre-eclampsia, or in those in which bleeding has occurred in the early months of pregnancy.

(2) Primigravidae under 25 with otherwise normal pregnancies are allowed to go into labour spontaneously, but induction is performed if, after term, maternal weight is falling steadily, the liquor appears to be lessening, or pre-eclampsia develops.

(3) If foetal distress develops during labour and especially if meconium is present in the liquor, oxygen is given to the mother and the case assessed on clinical grounds. Especially in the older primigravida, and if a further period of prolonged labour is to be expected, Caesarean section is seriously considered on the indication of “foetal distress”. With the already anoxic foetus, Caesarean section is often preferred if forceps delivery would be difficult and might put an undue strain on the foetus.

(4) In primigravidae over 25 labour is induced at the end of the 41st week. If, at induction, the liquor is heavily stained with meconium, elective Caesarean section is performed. Slowing of the foetal heart may not occur, or occurs too late to be relied upon as a sign of uncomplicated anoxic anoxia alone. It is unusual in primigravidae to find meconium staining at routine induction at this stage of pregnancy, and so, in practice, Caesarean section will not often be necessary.

(5) In multigravidae somewhat similar action is taken. Since most labours are short and easy, Caesarean section is rarely called for, but induction at the end of the 41st week is frequently performed. Where the previous child has been stillborn, delivery is usually achieved before term.

It may seem to some disappointing and surprising that what should be a natural and easy function requires so much care if the best results are to be obtained. There are several reasons for this.
FOETAL ANOXIA

Firstly, in modern society there is no natural selection of women most efficient in reproduction and the higher the standard of obstetrics the less the selection.

Secondly, as stressed by Baird (1952a, b), and Baird and Ilsley (1953), youth and optimal health are necessary for easy and safe childbearing, and of special importance in this paper is the demonstration that the incidence of "unexplained death" of the mature foetus and of death directly due to difficult labour are lowest in the age group 15 to 19 and increase steadily with increasing age.

The liability to post-maturity is just as great in primigravidae under 20 as in any other age group, so that, if post-maturity is regarded as "abnormal", then the young woman is no more efficient than older women in this respect. Nevertheless the consequences for the child are very much less serious in the younger woman, partly because of a much lower incidence of difficult labour. A study of foetal distress rates shows, however, that in the young woman oxygen supply to the foetus does not deteriorate so quickly, suggesting that the uterine blood flow is more efficient, and that there is a greater reserve.

I have shown that, as pregnancy is prolonged beyond 40 weeks, the foetus suffers from an increasing shortage of oxygen, and under certain conditions this can prove fatal. It is not at all clear why this deterioration should occur. There is still a great deal to be learned about the physiology of normal pregnancy and the effects and mechanism of deviations from the normal. It would appear that, in most women, prolonged pregnancy is pathological, but it is impossible to say why labour should fail to occur or when, in the given case, a delayed onset of labour becomes particularly dangerous to the foetus.

Meantime, therefore, we must attempt to assess the dangers to the foetus in the given case, and base treatment on all the information at our disposal without, if possible, increasing the risks to the mother.

Our experience of induction of labour and indications for Caesarean section have been very briefly described. The results are encouraging, but a longer time will be necessary before the role of induction of labour can be more clearly defined and the risks of the procedure properly evaluated. In our experience, induction as performed on the indications discussed appears to lessen the risks to the foetus without increasing the risk to the mother.

NOTE

The tables from which Figs. 4, 5, 6, 7, 8, and 10 were constructed have been omitted at the request of the editor.

In Fig. 4 the rise in the obstetric death and stillbirth rates from the 41st to the 43rd week is significant, P.<.01. The rise in the neonatal death rate is not significant. In Fig. 5 the rise in the rate of "unexplained mature" deaths from 41st to 43rd weeks in primigravidae is significant, P.<.05. The rise in the "difficult vertex" rate just fails to reach the 5 per cent level of significance on the one tail test. The increases in "unexplained mature" and "difficult vertex" rates after term in multigravidae because of small numbers are not statistically significant.

In Fig. 6 the difference between rates at 40 and 43 weeks is highly significant in each graph. In Fig. 7 the difference between rates of "cause unknown" at 40 and 43 weeks is highly significant, and rates for prolonged labour plus pressure on the head are significant, P.<.001.

In Fig. 8 both graphs are highly significant.

In Fig. 10 rates for primigravidae are significantly greater at the 30-34 age group than at the 20-24 age group. At the same age groups in multigravidae the differences are not statistically significant.

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