SOME ASPECTS OF THE MANAGEMENT OF THE PREGNANT DIABETIC

Senior Registrar in Obstetrics and Gynaecology to King's College Hospital and the Woolwich group of hospitals.

Increasing numbers of diabetic patients are becoming pregnant and what was a rare occurrence is now becoming much less so. Furthermore the passage of the next decade will see more and more children born of diabetic mothers, themselves entering the childbearing age group. If the influence of heredity on the development of diabetes is accepted, and a family history of the disease is frequently found, such patients should show an increased incidence of diabetes which in turn will serve to perpetuate the problem.

The Effect of Pregnancy upon Diabetes

Under modern conditions of management the maternal mortality of pregnant diabetics has fallen to very low levels; Black and Miller (1958) reported a maternal mortality of less than 0.6 per cent. in 3,200 cases reported in the literature in the last ten years. Such deaths as have been reported in different series have been most often due to obstetric accidents occurring during the early years of the series. For the more severe diabetic the undertaking of pregnancy may be said to be hazardous only where vascular complications of the disease are present. The advisability of therapeutic termination of pregnancy must be seriously considered during the early months in patients with hypertension, renal disease or retinopathy.

From the point of view of management of the diabetes the significant changes occurring in pregnancy are a disturbance of carbohydrate metabolism causing changes in the patient's insulin requirement and secondly a lowering of the renal threshold.

Disturbance of the Carbohydrate Metabolism

During pregnancy the carbohydrate balance of the expectant mother is upset, with a lowered glucose tolerance favouring the development of acidosis (Eastman, 1946; Hurwitz and Jensen, 1946; Weiden, 1948). Diabetes accentuates the disturbance which manifests itself in a changed insulin requirement. In most patients the change is toward increased insulin requirement (Oakley, 1953; Given et al., 1950; Hall and Tillman, 1951, and others). The increase usually starts from about 12 weeks onwards and may result in the number of units needed to maintain control being doubled or trebled. In a proportion of patients the requirement is diminished and in a very few it remains unchanged. Whatever the change it is usually temporary and a return to pre-pregnancy conditions ensues rapidly during or following delivery (Peel and Oakley, 1950).

Lowering of the Renal Threshold

Diabetics more than normal pregnant patients experience a lowering of the renal threshold which comes on between 12 to 20 weeks and lasts sometimes until term. The significance of this lies in the fact that control of the diabetes by testing for glycosuria may be rendered impossible and furthermore if the loss of sugar in the urine is sufficiently great ketosis may be induced. The importance of testing for ketonuria and of frequent blood sugar estimations is apparent in this type of case.

The Effect of Diabetes upon Pregnancy

In considering the effect of diabetes upon pregnancy the striking feature is the greatly increased foetal mortality. In a series of cases published by Whirrigh Williams (1909) before the advent of insulin therapy the foetal mortality was 41 per cent. and it is only recently that this high figure has been improved on (Table 1). The foetal loss is due to a high rate of intrauterine death in the latter weeks of pregnancy and to the high neonatal death rate among the delivered infants. The two are connected in that termination of a diabetic pregnancy at a stage designed to avoid intrauterine death of the foetus will inevitably be associated with an increased proportion of immature infants and an increased neonatal death rate. This is shown in the accompanying table (Table 2) modified from Bachman (1952), which also indicates the overall saving of foetal
life by termination of the pregnancy up to six weeks before full term is reached.

Although the series are not comparable in other respects the point is certainly established and management is based on delivery at or before the end of the 37th week. By way of confirmation Peel and Oakley (1950) have shown that of 183 babies lost in the collected series no fewer than 63 (34.4 per cent) died in utero during the last four weeks of pregnancy. The neonatal death rate falls steadily toward term but does not fall to anywhere like normal levels and hence there is no justification in allowing these pregnancies to proceed after the 37th week. If all are delivered at that date fewer babies will die in the neonatal period than would be lost from death in utero if they were left undelivered until term.

The reasons for the high foetal mortality are complex and are still by no means completely understood. Considerable controversy still exists as to what is important in decreasing foetal mortality but certain proven facts have emerged as a result of the publication of large series of cases by White, Peel and Oakley, Pedersen and Brandstrup, Stephens and others.

**The Factors Influencing Foetal Mortality**

**Control of Diabetes**

The one common denominator that has emerged from the most successful series of cases is that the highest foetal survival rates can only be obtained by careful control of the diabetic state. Control is only attained by close supervision of the patient by a physician with special experience in diabetes. The best way of achieving close control particularly in the last six to eight weeks of the pregnancy is to admit the patient to hospital. Without good control of the diabetes, consistently good

---

### Table 1

**The Incidence of Foetal Mortality in Diabetic Pregnancies (Excluding Abortion)**

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Period</th>
<th>No. of Cases</th>
<th>Stillbirths %</th>
<th>Neonatal deaths %</th>
<th>Total %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barns and Morgan</td>
<td>1949</td>
<td>1926-48</td>
<td>40</td>
<td>32.5</td>
<td>22.5</td>
<td>55.0</td>
</tr>
<tr>
<td>Oakley and Peel: Q Series</td>
<td>1949</td>
<td>1942-49</td>
<td>458</td>
<td>29.6</td>
<td>10.7</td>
<td>40.3</td>
</tr>
<tr>
<td>Given et al.</td>
<td>1950</td>
<td>?</td>
<td>113</td>
<td></td>
<td></td>
<td>27.2</td>
</tr>
<tr>
<td>Bastiaanse and Sindram</td>
<td>1951</td>
<td>1938-51</td>
<td>95</td>
<td></td>
<td></td>
<td>21.0</td>
</tr>
<tr>
<td>Barns and Morgan</td>
<td>1952</td>
<td>1947-52</td>
<td>24</td>
<td>12.5</td>
<td>4.2</td>
<td>16.7</td>
</tr>
<tr>
<td>Whiteley et al.</td>
<td>1953</td>
<td>1941-52</td>
<td>72</td>
<td>9.7</td>
<td>9.7</td>
<td>19.4</td>
</tr>
<tr>
<td>Jones</td>
<td>1953</td>
<td>1927-51</td>
<td>162</td>
<td>1.6</td>
<td>8.4</td>
<td>9.0</td>
</tr>
<tr>
<td>Nelson et al.</td>
<td>1953</td>
<td>1950-52</td>
<td>128</td>
<td>5.4</td>
<td>10.7</td>
<td>21.2</td>
</tr>
<tr>
<td>White</td>
<td>1953</td>
<td>1936-53</td>
<td>550</td>
<td></td>
<td></td>
<td>11.0</td>
</tr>
<tr>
<td>Tolstoi et al.</td>
<td>1953</td>
<td>1950-53</td>
<td>72</td>
<td>5.4</td>
<td>10.7</td>
<td>22.1</td>
</tr>
<tr>
<td>Pedersen</td>
<td>1954</td>
<td>1946-52</td>
<td>149</td>
<td></td>
<td></td>
<td>27.0</td>
</tr>
<tr>
<td>Medical Research Council</td>
<td>1955</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hormone Treated Cases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Hormone Treated Cases</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Long et al.</td>
<td>1954</td>
<td>1942-48</td>
<td>62</td>
<td>21.0</td>
<td>14.5</td>
<td>35.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1948-52</td>
<td>56</td>
<td>12.5</td>
<td>5.4</td>
<td>17.9</td>
</tr>
<tr>
<td>Stephens</td>
<td>1956</td>
<td>1951-55</td>
<td>50</td>
<td>4.0</td>
<td>4.0</td>
<td>8.0</td>
</tr>
<tr>
<td>Clayton</td>
<td>1956</td>
<td>1949-56</td>
<td>201</td>
<td>11.5</td>
<td>17.3</td>
<td>28.8</td>
</tr>
<tr>
<td>Boughton and Perkins</td>
<td>1956</td>
<td>1940-56</td>
<td>51</td>
<td>19.7</td>
<td>9.7</td>
<td>29.4</td>
</tr>
<tr>
<td>White: All cases</td>
<td>1956</td>
<td>1936-56</td>
<td>760</td>
<td>3.5</td>
<td>9.8</td>
<td>13.3</td>
</tr>
<tr>
<td>Hormone Treated Cases</td>
<td>1956</td>
<td>1936-56</td>
<td>298</td>
<td>3.5</td>
<td>9.8</td>
<td>13.3</td>
</tr>
<tr>
<td>Pedersen and Brandstrup</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Long Term Series</td>
<td>1956</td>
<td>1946-55</td>
<td>130</td>
<td></td>
<td></td>
<td>10.0</td>
</tr>
<tr>
<td>Short Term Series</td>
<td>1956</td>
<td>1946-55</td>
<td>135</td>
<td></td>
<td></td>
<td>33.0</td>
</tr>
<tr>
<td>Hagbard: Early Series</td>
<td>1956</td>
<td>1948-51</td>
<td>217</td>
<td>33.6</td>
<td>10.9</td>
<td>44.5</td>
</tr>
<tr>
<td>Recent Series</td>
<td>1956</td>
<td>1951-54</td>
<td>25</td>
<td>14.1</td>
<td>11.9</td>
<td>26.2</td>
</tr>
<tr>
<td>Black and Miller</td>
<td>1958</td>
<td>1940-49</td>
<td>68</td>
<td>13.3</td>
<td>10.2</td>
<td>23.3</td>
</tr>
<tr>
<td></td>
<td>1958</td>
<td>1950-57</td>
<td>118</td>
<td>12.7</td>
<td>3.4</td>
<td>16.1</td>
</tr>
</tbody>
</table>

### Table 2

<table>
<thead>
<tr>
<th>Viable pregnancies</th>
<th>Stillbirths %</th>
<th>Neonatal deaths %</th>
<th>Total foetal loss %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peel and Oakley: collected series (mainly no termination)</td>
<td>458</td>
<td>29.4</td>
<td>10.8</td>
</tr>
<tr>
<td>K.C.H. series (termination)</td>
<td>141</td>
<td>11.3</td>
<td>14.2</td>
</tr>
<tr>
<td>American clinics (no termination)</td>
<td>226</td>
<td>16.2</td>
<td>6.4</td>
</tr>
<tr>
<td>American clinics (termination)</td>
<td>584</td>
<td>8.8</td>
<td>8.7</td>
</tr>
<tr>
<td>Hagbard (no termination)</td>
<td>217</td>
<td>33.6</td>
<td>10.9</td>
</tr>
</tbody>
</table>
Table 3 (After Peel and Oakley)

<table>
<thead>
<tr>
<th>Insulin</th>
<th>No. of pregnancies</th>
<th>Live</th>
<th>Stillborn</th>
<th>Neonatal death</th>
<th>Total foetal loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>395</td>
<td>236</td>
<td>114</td>
<td>45</td>
<td>159 (40%)</td>
<td></td>
</tr>
<tr>
<td>Non-Insulin</td>
<td>42</td>
<td>28</td>
<td>10</td>
<td>4</td>
<td>14 (33%)</td>
</tr>
</tbody>
</table>

Results are not possible but it is unfortunately true that foetal death can occur without evidence of diabetic instability. Good chemical control of the diabetes does not, alas, guarantee foetal survival. There must therefore be some factor which accounts for the foetal deaths which occur in the well controlled patients. Per contra, there must also be a factor which operates to protect the foetus in the majority of well controlled cases. At the present time it is uncertain what this factor may be.

Following on from the hypothesis that diabetic control is important it is logical to enquire into the relationship between foetal loss and the severity of the maternal diabetes. 'Severity of diabetes' may be defined in terms of the time which elapses between the cessation of insulin and the onset of ketosis. From this point of view and from the point of view of insulin requirement, no direct relationship exists (Oakley, 1953; White et al., 1956). It has furthermore been shown by Peel and Oakley (1950), that foetal mortality is as high in non-insulin users as in those cases requiring insulin. This finding has been confirmed by other writers (Given et al., 1950; Miller et al., 1944).

White has introduced a classification of severity of diabetes in pregnancy based on the age of onset, duration of the disease and the presence of vascular complications. She claims that those which fall into the more severe categories show a higher foetal loss than those in the less severe categories. Oakley (1953) and Pedersen (1954) on the other hand state that there is no relation between the age of onset and duration of the disease and the foetal mortality. Pedersen and Brandstrup (1956) agree with White in finding a low foetal mortality in group A and a high foetal mortality in group F. They were unable to show any difference in groups B, C and D. This is borne out by the figures quoted by Nelson et al. (1953). One is left with the impression that White’s classification is not entirely satisfactory and does not completely relate the ‘severity’ of the maternal disease to the foetal mortality. Nevertheless, there appears to be a group of mothers with vascular degenerative disease in whom there is a high perinatal mortality but there is no evidence that this is due to the duration of the diabetes alone. It seems possible that the presence of maternal vascular disease resulting from the diabetes and adversely affecting placental blood flow may in the end provide a link but here the further difficulty arises in how to assess accurately the degree of vascular degenerative changes. Radiological studies to demonstrate arterial calcification can at best only provide a rough guide and give no indication of the functional impairment of the vascular tree. Berglund and Zetterstrom (1954) have suggested on the basis of some preliminary observations on the oxygen saturation of cord blood of infants delivered of diabetic mothers, that severe hypoxia may in fact occur in utero.

Toxaemia

Early writers were impressed with the frequency with which toxaemia develops in the last trimester of diabetic pregnancy. They also noted that cases developing toxaemia showed a much higher foetal loss than those that did not. The reported incidence varies from 3 per cent. (Nelson et al., 1953) to 50 per cent. (Barns and Morgan, 1949; Koller, 1953). The reason for the wide variation is partly due to differences in the management of the patients. Those series in which routine termination at or before the end of the 36th week is practised show a lower incidence than those in which the pregnancy is allowed to proceed longer. Peel and Oakley (1950) showed an incidence of toxaemia of 10.7 per cent. in the former as opposed to 18.9 per cent. in the latter. The high incidence after 37 weeks is a further point in favour of termination at or before this date bearing in mind that the foetal loss will rise pari passu with the increasing incidence of toxaemia.

Hydramnios

The incidence of hydramnios in diabetic pregnancy like the incidence of toxaemia is variously reported. This is almost certainly due
to the difficulty in estimating minor degrees of the condition clinically. Peel and Oakley (1950) believe that every diabetic pregnancy has some degree of hydramnios and that it reaches a severe degree in 30 per cent. They relate hydramnios to a raised incidence of toxaemia, larger babies, and a higher foetal mortality. They were, however, unable to correlate its incidence with a raised average blood sugar during the pregnancy. Pedersen (1954) confirmed this point but showed that the incidence was less in those patients with well controlled diabetes, as judged by physiological levels of blood sugar during the pregnancy. From the therapeutic point of view, rest appears to have a beneficial effect in diminishing hydramnios slightly but no other measures are of proven value. It is well to remember when treating these patients that major congenital defects, responsible for death of the foetus, occur much more commonly than in diabetic patients without hydramnios. Amniotomy should only be resorted to in severe cases since it is frequently followed by the onset of labour.

Congenital Abnormalities

An increased incidence of major congenital abnormalities is quoted by many authors on this subject (Peel, White, Warren and Lecompte, Miller and others). There have been a number of series in which no increase has been reported (Potter and Adair, Mengert et al., Reis et al., Hall and Tillman, Moss and Mulholland). The evidence for the increased incidence of congenital abnormalities is unsatisfactory according to Cardell (1953) but Reid (Quoted by Miller, 1956) states that there seems to be a statistically significant increase in the incidence of lethal abnormalities. Nevertheless it should be noted that the majority of foetal abnormalities in infants born of diabetic mothers are non-fatal. Pedersen (1956) comments that the occurrence of 3 per cent. of major congenital abnormalities will prevent the foetal mortality in diabetic pregnancies from falling to the levels found in non-diabetics.

Difficulties in Delivery

Apart from the complications of toxaemia and hydramnios, practical obstetrical difficulties arise when the diabetic patient is to be delivered. The large size of the babies has contributed to the adoption of Caesarean section as the method of delivery of choice in the majority of cases. Caesarean section affords a means of terminating the pregnancy at an elective time and avoids the potential trauma of vaginal delivery as well as the inertia and unbalanced diabetic state which not infrequently occurs in the induced labour. Peel and Oakley (1950) showed that one pregnancy in six resulted in stillbirth occurring during labour and vaginal delivery, whereas the 'intrapartum' loss was only one in 70 when Caesarean section was employed. The neonatal death rate is about the same whether the infant is delivered by the vaginal route or by section. As is commonly the case in this controversial subject a proponent for the opposite point of view is easily found. Pedersen and Brandstrup (1956) only performed Caesarean section where it was indicated on obstetrical grounds and induced the remainder (89 per cent.) at the beginning of the 38th week. The low foetal mortality (10 per cent.) was the same as that in a similar number of patients in whom inductions were only performed in 30 per cent., the remainder being delivered by Caesarean section. It would seem therefore that a definite place exists for the induction of labour where an easy vaginal delivery may be expected to take place. This is especially true of multigravida with a history of a previous normal delivery or in primagravida when the head is deeply engaged in the pelvis and the cervix is favourable. It must, however, be borne in mind that a failed induction followed by a Caesarean section carries an increased risk of neonatal infection.

The Role of Hormone Therapy

The value of hormone therapy in diabetic pregnancy was first investigated following the work of Smith and Smith (1934) who postulated that an imbalance of female sex hormones, low levels of oestrogen in the blood and progesterone in the urine (measured as urinary pregnandiol) and high levels of serum chorionic gonadotrophin existed in diabetic pregnancies. It was further claimed that the balance could be corrected by the administration of oestrogen and progesterone in increasing dosage during the pregnancy, and that as a result of such therapy the foetal mortality was lowered. The claim, backed as it was by the good results obtained by White and her colleagues in a large series of cases, was hard to refute, but with the publication of the report of the Medical Research Council's controlled trial and the recent series of cases comparable in severity with those of White which have shown equally good results without hormone therapy (Long et al., 1954; Pedersen and Brandstrup, 1956; Stephens, 1956; Black and Miller, 1958), it is no longer generally believed that hormone therapy is of any value. In a recent paper White (1956) points out that the hormone therapy she employs represents only a small part of the management of the pregnant diabetic. The excellent results obtained by White are most likely due to the close supervision which her patients receive during their pregnancies.
Management of the Newborn Infant

The services of an experienced Paediatrician are essential. He should form an integral part of the diabetic pregnancy team, indeed it may be that the reductions in perinatal mortality during the past 20 years in part at least may reflect the general improvement in perinatal mortality for all infants during this period (Miller, 1956). The detailed care of these infants is beyond the scope of the present paper.

A Scheme of Management for a Diabetic Pregnancy

The following scheme of management is largely based on that at present in use at King's College Hospital with certain additions culled from the literature quoted above.

The management of the pregnancy is undertaken by a team consisting of an obstetrician, and a physician experienced in the treatment of diabetes and a paediatrician experienced in the care of infants born of diabetic mothers. It goes without saying that a high degree of nursing skill and nurses familiar with the difficulties of the situation are essential if the best results are to be obtained.

Ante-Natal Care

First Visit

A routine ante-natal examination is made and the diabetic condition fully assessed. The insulin dosage is adjusted as necessary, two doses of soluble insulin being almost invariably preferred. The diet is fixed as regards carbohydrate intake. Unlimited fat and protein is allowed unless the patient is overweight when some restriction of the former is advisable.

Subsequent Visits

The patient should be seen in the ante-natal and diabetic clinics every fortnight. In the latter clinic a complete and careful review of the patient's diabetic state is carried out. The insulin dosage is adjusted in relation to urine and blood sugar tests and to the presence or absence of ketonuria. A preprandial blood sugar level of not more than 170 mg. is the aim. The patient is admitted for stabilization at the earliest signs of any relapse. In addition, hospital admission is often necessary in the case of minor intercurrent illnesses (influenza, pyelitis, etc.). The whole of the ante-natal care should be managed personally by one obstetrician and one physician only.

Ante-natal Admission

The patient should be admitted at 32 weeks. Control of the diabetes is now placed on a day-to-day basis with five specimens of urine tested for sugar and acetone daily. Blood sugar estimations are carried out twice weekly or more often as indicated. A small dose (4 to 12 units) of protamine zinc insulin is added to the evening dose of soluble insulin and the combined injection given before tea in those patients who show sugar and acetone in the first morning specimen of urine. The carbohydrate allowance is increased by 60 g. daily to reduce the occurrence of ketosis. If ketosis persists in association with a normal or low blood sugar additional carbohydrate is given in the form of sugar taken with the main meals.

In general the patient should be delivered about the end of the 36th week unless toxaemia or hydramnios make earlier delivery advisable. Caesarean section is performed on all primigravida. In the case of the multigravida patient induction is reserved for those patients who have had a previous successful vaginal delivery. Hypoglycaemia and ketosis are avoided on the day of operation or labour by a reduced insulin dosage and adequate carbohydrate intake e.g. in the case of the patient undergoing Caesarean section at 9 a.m. half the normal morning dose of soluble insulin is given and 30 g. of glucose are given intravenously (60 c.c. of a 50 per cent. glucose solution) with the premedication. General anaesthesia (thiopentone, suxamethonium, pethidine, nitrous oxide and oxygen) is preferred to spinal epidural or local analgesia. Control of the diabetes in the immediate post-operative period or during labour where induction has been performed is based on an 'emergency routine' of four-hourly urine testing supplemented by blood sugar estimations as necessary. The baby is handed

<table>
<thead>
<tr>
<th>Years</th>
<th>No. of cases</th>
<th>Toxaemia</th>
<th>Hydramnios</th>
<th>Caesarean Section</th>
<th>Induction of labour</th>
<th>Spontaneous onset of Labour</th>
<th>Stillbirths</th>
<th>Neonatal deaths</th>
<th>Congenital abnormalities</th>
<th>Total foetal loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>1954 Mid-1956</td>
<td>74</td>
<td>23</td>
<td>32</td>
<td>37</td>
<td>27</td>
<td>13</td>
<td>8</td>
<td>5</td>
<td>6.8%</td>
<td>28.4%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>32.2%</td>
<td>43.5%</td>
<td>50.0%</td>
<td>36.3%</td>
<td>13.7%</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mid-1956</td>
<td></td>
<td>7</td>
<td>9.5%</td>
<td>23</td>
<td>49</td>
<td>15</td>
<td>10</td>
<td>7</td>
<td>9.5%</td>
<td>14</td>
</tr>
<tr>
<td>Oct. 1956</td>
<td></td>
<td>4.5%</td>
<td>32.2%</td>
<td>66.2%</td>
<td>20.3%</td>
<td>13.7%</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

NOTE.—Only those pregnancies which went past 28 weeks are included. Two cases have been excluded from the 1957 figures because they were not admitted at 32 weeks.
over to the expert paediatric care and nursing immediately after delivery.

Appendix
A scheme of management substantially the same as that described above has been in operation at King's College Hospital since mid-1956. The number of cases is not yet very large but the results obtained so far are encouraging. The writer has collected the figures up to October, 1958 and they are reproduced by kind permission of Mr. J. H. Peel and Dr. W. G. Oakley. For comparison a similar consecutive series from 1954 to mid-1956 is also given. The main difference in the management of the two series lies in the fact that since mid-1956 the patients have been admitted for rest and stabilization at 32 weeks.

It will be seen that an improvement in the foetal loss has occurred and in particular the stillbirth rate has fallen. There has also been a decrease in the incidence of toxæmia and of hydramnios.

Acknowledgments
I am grateful to Mr. J. H. Peel and Dr. W. G. Oakley for their advice and assistance in preparing this paper.

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J. M. Brudenell

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