ASPHYXIA NEONATORUM*

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Methods and material. My object in this survey was to tour a number of medical schools in the British Isles in order to observe their methods and to discuss their views on

(1) the prediction and prevention of foetal anoxia, (2) the management of labour when foetal distress may be expected, (3) the management of the asphyxiated baby.

I have presented the material in three sections, the first concerns the antenatal events leading to foetal anoxia, the second deals with the management of labour, and the third discusses methods of treatment which may be applicable in domiciliary midwifery.

I have quoted fairly extensively from the National Perinatal Mortality Survey (1961) carried out in 1958 under the auspices of the National Birthday Trust Fund, and I think this is justified because if one excludes congenital abnormalities, the causes of asphyxia neonatorum and perinatal death are very similar, the differences being those of degree. The perinatal mortality is an index of obstetric performance which is in general use now, and comprises the total of stillbirths and deaths in the first week after delivery.

The problem. In general terms the problem of the prevention of asphyxia neonatorum is an exercise in thinking in terms of foetal oxygenation. It is now known that there are many factors operating at all stages during pregnancy and delivery which may interfere with the transfer of oxygen via the placenta to the foetus, and it is necessary to consider these factors systematically at each examination of the patient.

It is true that one will occasionally encounter a case of intrapartum or neonatal death from asphyxia in which there appears to

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be no antecedent cause, and these are perhaps the more important as they represent the unexplored problems in the transfer of oxygen from mother to foetus.

**ANTENATAL CARE**

**The First Visit**

There is a point in practice organization which is worth mentioning here, and that is the importance of an appointment system for antenatal examinations. I realize that not all doctors or practices find it suitable to make appointments for their general work, but I have found by experience that the standard of my antenatal care has improved considerably since I set aside one afternoon a week exclusively to antenatal work. This is particularly important at the patient's first visit, when one can well give her a double appointment. At this time one should elicit and record the significant factors in her personal medical history, and in her past obstetric performance.

**Maternal Medical History**

*Heart disease.* Except when severe congestive cardiac failure is present, maternal heart disease does not appear to cause significant foetal distress. This is perhaps due to the shape of the foetal oxygen dissociation curve, which enables it to abstract oxygen from the mother at low maternal oxygen tensions. The maternal oxygen saturation is decreased in cardiac failure and cyanotic congenital heart disease, and it is important to remember the factors which will aggravate this, such as exercise, chest infections, and breathing hypoxic mixtures such as are present when the Minnitt "gas and air" machine is used.

It is said that the highest maternal cardiac output occurs between the twenty-eighth and thirty-second weeks, and at the end of pregnancy there is considerable cardiorespiratory embarrassment from the size and weight of the abdominal tumour, so that additional rest is indicated at these times. The perinatal mortality rate is a little higher in cases of maternal heart disease than the average, and this seems to be associated mainly with the frequency of premature labour.

*Pulmonary disease.* The same problem of a reduced maternal oxygen saturation is present in severe pulmonary disease such as bronchiectasis, chronic bronchitis, and emphysema as it is with
Photograph of pendant designed by Messrs Gerrard for provosts of faculties
cardiac disease, and the same considerations apply. It is important to treat any superimposed acute chest infection thoroughly.

Anaemia. Maternal anaemia is a common and serious complication of pregnancy. The National Perinatal Mortality Survey (1961), which analyses nearly 8,000 stillbirths, comments on the high incidence of maternal anaemia in these cases, and that remarks in 60 per cent of cases in domiciliary practice no haemoglobin estimations had been made.

The foetus has a tremendous appetite for iron, and at the end of pregnancy the foetal requirements are about 3 mg. of iron per day. The maximum rate of absorption of iron from the intestine is about 1.5 mg. of iron per day, so it is clear that we must raise the maternal haemoglobin level to its maximum value and fill the maternal iron storage reserves in the early months of pregnancy. It is my policy to begin oral iron treatment in all patients as soon as the initial vomiting of pregnancy has ceased, because one is often unable to make good the iron shortage later without recourse to intramuscular injections.

The following case indicates some of the problems.

Mrs. B. J., a well nourished and intelligent primigravid, said she had taken her iron tablets from the beginning of her pregnancy. Her first haemoglobin estimation showed 83 per cent, the next 77 per cent, and then 70 per cent. After four injections of Jectofer (an intramuscular iron compound) it had fallen to 66 per cent. The blood picture was iron deficient and normocytic at all times. Ultimately she was receiving oral iron, intramuscular iron, vitamin C, and folic acid, and the final report just before term showed a haemoglobin level of 83 per cent.

I would not now use Jectofer and oral iron simultaneously as toxic effects may occur.

A megaloblastic anaemia of pregnancy is not uncommon, and in twin pregnancies it is said that there is almost always a folic acid deficiency. In single pregnancies it is probably much more common than was formerly believed.

Weight for weight, green vegetables contain more iron than any other article of diet and, of these, spinach has most. There seems to be little difference between the various iron tablets, but I believe vitamin C helps in the absorption of iron and I prescribe Ferchlor which contains 200 mg. of ferrous sulphate and 50 mg. of vitamin C.

Blood group. Rhesus and ABO blood grouping should be done at the first visit, in case an early blood transfusion should be required, for example following an incomplete abortion. All patients must be
checked for antibodies about six weeks before term as antibody formation to Rhesus positive and ABO factors is known to occur.

_Diabetes._ It is important to diagnose diabetes early in pregnancy since it is known that without strict treatment about one third of babies will be lost. Walker (1959) has shown that the oxygen saturation in the cord blood of these babies is low. They tend to be large babies and undergo long and difficult labours, and these factors predispose to severe asphyxia.

Urine tests for sugar should be done at the first visit. About 30 per cent of pregnant women will show reducing substances in their urine but not all these will be diabetic. Pregnancy causes a lowered renal threshold to glucose due to an increased tubular filtration rate and interference with glucose reabsorption by the tubules. However, all patients showing reducing substances require full investigation. The untreated or inefficiently treated diabetic is at risk all through her pregnancy.

In early pregnancy there is an increased likelihood of moniliasis and pyelonephritis. In mid pregnancy the diabetes becomes unstable and difficult to control. In late pregnancy there is an increased tendency towards toxaemia, hydramnios, premature labour, and intrauterine foetal death from anoxia.

Sir John Peel (1962) has shown that by strict diabetic control he has reduced his stillbirth plus neonatal death rate from 32 per cent in an eight-year period up to 1949 to 13.3 per cent in the three-year period up to 1961.

In the early weeks of pregnancy, weekly blood sugar estimations are arranged at the diabetic clinic. At thirty weeks the patient is admitted to hospital for more strict control. Serial blood sugar estimations are performed once or twice weekly, and the blood sugar is not allowed to rise above 180 mg. per cent, using whatever insulin suits the patient best.

Pregnancy is terminated at 38 weeks by surgical induction or earlier if there is toxaemia, hydramnios, or a big baby. Caesarean section is done if labour has not begun after 24 hours, or if there is vomiting. The baby is intubated at birth if there is any respiratory distress. The pulmonary syndrome (hyaline membrane) is a frequent complication in a baby born of a diabetic mother.

The general practitioner’s task in this problem is to maintain strict surveillance and control of the diabetic patient up to the time of her admittance to hospital. Certain of the patients with glycosuria
due to a low renal threshold will be pre-diabetic, and it is well not to allow any of these patients to go beyond term, particularly if there is any evidence of toxaemia. They will also tend to have large babies.

*Hypertension.* The arbitrary figure for the definition of hypertension which I adopt is a blood pressure reading of above 140/90 mm. Hg. Many recommend lower figures, and it may be that the rise in blood pressure from the level at the beginning of pregnancy is of equal significance to the absolute pressure.

McClure Browne (1959) has shown by a technique of injecting radioactive sodium into the intervillous space and into the uterine muscle that when maternal hypertension was present the half period of clearance of this substance from the intervillous space was 60 seconds compared with 20 seconds in the normotensive patient, and the calculated uterine blood flow by this technique was 200 ml./min. with hypertension compared to a figure of 600 ml./min. in the normal. Thus the oxygen supply to the foetus must be considerably reduced in hypertensive patients who suffer this marked slowing of blood flow to the uterus and placental site.

In the National Perinatal Mortality Survey the cause of death of 451 of the babies was considered to be asphyxia in labour. After mechanical factors, such as accidental haemorrhage, placenta praevia, and prolapsed cord, had been excluded, the two commonest findings were a diastolic pressure above 90 mm. Hg., and prolongation of pregnancy beyond 287 days. A combination of these two factors carries a particularly bad prognosis.

*Pyelonephritis.* I have noticed occasionally in the retrospective histories of patients who have had stillbirths the record of recurrent attacks of pyelonephritis. I do not know if this is due to the associated hypertension or if pyelonephritis has some adverse effect of its own, but I always anticipate trouble when I encounter a story of repeated renal infection.

**Maternal Obstetric History**

*Age and parity.* These may be considered together as there are certain significant associations between the mother's age and parity in the production of foetal asphyxia.

Lewis (1956) states that the incidence of foetal distress and unexplained stillbirth is six times greater in the primigravid than the multigravid after 42 weeks duration of pregnancy. It was a case of this nature which stimulated my interest in this subject.

Mrs. P. E. was aged 24 and primigravid. She had a normal pregnancy except for severe hyperemesis in the early months. She lost two pounds in weight as
she approached term. When fourteen days overdue she went into rather slow labour and produced a severely asphyxiated baby. Spontaneous respiration was established after resuscitation for one hour, but the baby died at 16 hours of age. No abnormality was found at necropsy beyond signs of asphyxia.

Walker (1959) shows that the umbilical vein oxygen saturations at delivery are lower in the primigravid than the multigravid, and that the values for the elderly primigravid are ten per cent lower than those of the young primigravid. McKinley (1959) in her work on cord oxygen values, although she is unable to confirm certain other of Walker’s findings, shows a markedly reduced cord vein oxygen saturation in the elderly post mature primigravid. McClure Brown (1959) also shows that there is a suggestion of slowing of the uteroplacental circulation with age, especially in primigravidae.

The age at which the primigravid begins to encounter increased risk is 25 and over 30 the risk is marked. The National Perinatal Mortality Survey regards the mother over 35 as a special risk, regardless of parity.

With the second and third pregnancies the risks to the foetus become less, but the perinatal mortality begins to rise again in the fourth pregnancy, and the “grand multipara” in her fifth and successive pregnancies is at much greater risk of losing her baby. This is mainly due to the high rate of malpresentations such as breech and transverse lies, and to antepartum haemorrhage. Disproportion can occur in the multipara if her babies are becoming bigger.

Previous obstetric history. Patients tend to reproduce their obstetric pattern. The only exception occurs after a long interval between babies, when they may start again with an entirely new pattern. The patient with the bad obstetric history needs careful observation and, as Ian Donald (1959) points out, the patient who has had a previous intra-uterine death for no apparent reason may suffer from recurrent placental insufficiency and should be closely watched for signs of placental failure as she approaches term. On no account should she be allowed to become overdue.

Date of conception. A careful assessment of the duration of pregnancy when made in the early months is often of considerable value later if the question of postmaturity arises.

At the first visit the patient will have a clearer recollection of her dates, and the size of the uterus and its rate of growth in the early months will give a more accurate indication of the duration of
pregnancy than the fundal height in later months.

Other landmarks will also be present in the early weeks, such as increased frequency of micturition, softening of the cervix, Hegar's sign, and the onset of foetal movements, and each of these can be related to their appropriate time in pregnancy.

Home or Hospital Confinement

This is a more important consideration in Monmouthshire where the hospitals can accept only about 60 per cent of the total number of deliveries, than an area such as Edinburgh where over 90 per cent of the patients are delivered in hospital.

An analysis of 42 completed questionnaires, compiled by the midwives, of the causes of death of stillbirths in domiciliary practice in Monmouthshire illustrates some of the problems.

<table>
<thead>
<tr>
<th>Cause</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prematurity</td>
<td>8</td>
</tr>
<tr>
<td>Congenital lesions</td>
<td>8</td>
</tr>
<tr>
<td>Long labour</td>
<td>4</td>
</tr>
<tr>
<td>Breech delivery</td>
<td>4</td>
</tr>
<tr>
<td>Toxaemia</td>
<td>4</td>
</tr>
<tr>
<td>Twins</td>
<td>3</td>
</tr>
<tr>
<td>Placental separation</td>
<td>2</td>
</tr>
<tr>
<td>Prolapsed cord</td>
<td>1</td>
</tr>
<tr>
<td>Unknown</td>
<td>8</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>42</strong></td>
</tr>
</tbody>
</table>

Twenty one of these patients were having their fourth or more baby, and nine were over 35 years of age. The figure for breech delivery confirms the findings of the National Perinatal Survey, which showed that the mortality of the primigravid breech delivery is six times higher at home compared to hospital. They also found that the mortality of breech delivery is doubled for an unsupervised houseman compared to a consultant or registrar. It is a fallacy to believe that the multiparous breech is a less difficult delivery than the primipara. All the obstetricians with whom I discussed this point were unanimous in their opinion that the multipara can be equally difficult and sometimes more so.

The overall breech mortality is about three and a half times greater than that of vertex delivery, so that antenatal external version at about 34 to 36 weeks is justified, provided that undue force is not used and preferably without a general anaesthetic.

Similar considerations apply to twin deliveries as to the breech
delivery and I do not think that either should intentionally be a home delivery.

It was interesting to note in the previous obstetric histories of these patients that six had had a previous stillbirth, one had two previous stillbirths, and one had a story of stillbirth twins and a stillborn single delivery.

The remaining indications for hospital delivery are generally well known, for example, premature labour, heart disease, diabetes, toxaemia, rhesus antibody formation, and so on.

Subsequent Antenatal Care

Management during pregnancy. After the initial examination and assessment, antenatal examinations are carried out at the usual intervals. During this period a number of conditions can occur which may give rise to an asphyxiated baby during labour. The more important of these are the bleedings of early or late pregnancy, toxaemia, premature labour, and prolonged pregnancy.

Threatened abortion. In any condition in which there is bleeding from the placental site, there is a partial separation of the placenta which heals by scar tissue, leaving a reduced area in contact with the uterine surface. Low cord blood oxygen values are found at term (Walker 1959) and the risks of intrauterine death or neonatal anoxia must always be considered following threatened abortion in early pregnancy.

Mrs I. H. is a case in point. She had two normal pregnancies, and then twins, complicated by hydramnios and toxaemia, who failed to survive after she went into labour at five months. In her next pregnancy she had a central placenta praevia and a live baby following caesarean section. At her first visit in this pregnancy she had a blood pressure of 160/90 mm. Hg. and a urinary infection. At 14 weeks she had a vaginal haemorrhage and was put to bed. At 22 weeks she had a further bleed and spent two more weeks in bed. At 26 weeks she suffered a recurrence of her urinary infection.

When I examined her at 31 weeks I noticed that she was having slight uterine contractions and I put her to bed once more, but despite that she went into labour one week later and produced a severely asphyxiated four pound baby in hospital. The baby survived and is now developing normally.

Patients who have had a threatened abortion in early pregnancy should not be allowed to become postmature.

Antepartum haemorrhage. Bleeding in later pregnancy is of even greater importance, and both Walker and McKinney (1959) find low cord blood oxygen values after antepartum haemorrhage. The National Perinatal Mortality Survey shows that accidental haemorrhage is the most important mechanical factor in producing intra-
partum asphyxia, and in my travels I was impressed by the number of people who commented on the increasing frequency of accidental haemorrhage as a cause of foetal loss.

When placenta praevia is diagnosed the choice lies between securing delivery at the first sign of trouble and accepting the risk of prematurity, or of adopting conservative treatment in the hope of achieving a more mature baby. The general opinion at the moment is towards expectant treatment in hospital whenever possible.

Toxaemia. Abramson (1960) states that the foetal loss from toxae-mia is about 20 per cent. The babies die of intrapartum or post partum asphyxia. McClure Browne (1959) shows that the uterine blood flow in toxaemia is about half the normal value. He shows that exercise reduces the blood flow still further, but rest increases it, which appears to give experimental confirmation to the clinical observation of the value of rest in toxaemia and, indeed, in all conditions in which there is impaired placental function.

On the foetal side there is a corresponding reduction in the umbilical cord oxygen saturation values at term (Walker 1959).

A particularly dangerous combination is the post-mature pre-eclamptic, and Wood and Pinkerton (1961) quote three primigravidae with mild toxaemia who were allowed to continue beyond term. Intrauterine death occurred when they were respectively seven, eight, and twelve days overdue. They consider that the risks of induction after 38 weeks with mild toxaemia may be less than the continuation of pregnancy. Their criteria of toxaemia are a blood pressure of 130/80 mm. Hg., with finger oedema. I also prefer to look for finger oedema than for ankle oedema, as it seems free of some of the fallacies associated with ankle oedema such as varicose veins, gravity, and an abdominal tumour.

Dawkins, Martin and Spector (1961) at University College Hospital made an analysis of 100 consecutive deaths from intrapartum asphyxia. The commonest causes were:

<table>
<thead>
<tr>
<th>Cause</th>
<th>Cases</th>
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</thead>
<tbody>
<tr>
<td>Abnormal labour (premature labour, prolapsed cord, etc.)</td>
<td>24</td>
</tr>
<tr>
<td>Postmaturity</td>
<td>20</td>
</tr>
<tr>
<td>Accidental haemorrhage</td>
<td>17</td>
</tr>
<tr>
<td>Toxaemia</td>
<td>15</td>
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</tbody>
</table>

Fifty of the deaths were regarded as having an avoidable factor, and the most common cause in this group was "undue prolongation of pregnancy in toxaemia" of which there were 21 cases. I think our task in practice is the earlier diagnosis of toxaemia and therefore
the earlier institution of treatment.

MacGillivray (1961) says that toxaemia is more likely to recur in patients who have been toxaemic in their first pregnancy, and that there is a familial tendency, in that sisters of patients who have had toxaemia are more likely to become toxaemic than sisters of patients who have not. He also points out that the likelihood of albuminuria developing is increased when the diastolic blood pressure is 80 mm. Hg. or more at the 20th week; when the rise in diastolic blood pressure between the 20th and 30th weeks is 5 mm. Hg. or more; and when the weekly weight gain in this period is 1½ lbs. or more.

These small points are becoming more important in the diagnosis of toxaemia and one should not need to wait until the gross changes have occurred.

It has been suggested that the fat woman is more likely to become toxaemic, and I have always been suspicious of the buxom young primigravid, who so often develops a sudden severe toxaemia late in pregnancy when one is viewing her rather complacently as having been seen successfully through her pregnancy.

Premature labour. At 33 weeks the baby will weigh about 3 lb. 3 oz. and 33 per cent will survive delivery at this time.

When a weight of 4 lb. 6 oz. is reached one finds that 81 per cent survive, (Report on Prevention of Prematurity, 1959), so it is clear that every week from the 30th week onwards improves the infant’s chance of survival.

Twins are a common cause of prematurity, and it is important to try to rest the mother in bed from the 30th until the 34th weeks and an extra week or two if father is good about the house.

The Monmouthshire County Council give priority in the provision of home helps to these patients, and I think most local authorities are aware of the importance of rest in the prevention of prematurity. Prematurity is high in families in the low social grades, especially if there are already many children. These patients are generally poor attenders at the antenatal clinic and must constantly be chased, and I think the local health authority can offer us a great deal of help with them.

The other important cause is antepartum haemorrhage, either placenta praevia or from a normally situated placenta. Many of these latter are associated with toxaemia or hypertension.

The predominant cause of death in premature babies is asphyxia,
usually with hyaline membrane formation (Donald, 1954). They are a particular problem in resuscitation as their respiratory muscles are barely able to maintain respiration when started. It is particularly important to maintain a clear airway.

Patients in premature labour must be admitted to hospital if time permits, and preferably to one with a premature baby unit, as some of the new work in transfusing babies in metabolic imbalance from respiratory distress is most promising.

_Prolonged pregnancy._ When I discussed the length of a normal pregnancy I was reminded of Omar Khayam, who frequented Doctor and Saint, and heard great argument

About it and about, but evermore

Came out by the same door as in I went.

First one must locate the date of conception as accurately as possible, as described earlier in the article. Without this one cannot begin to talk about postmaturity. Remember the fallacy of the irregular cycle.

Higgins (1956) collected a series of over 9,000 cases of mature babies in which he avoided surgical induction of labour except for the most stringent indications, so that the results may be taken as approaching the natural history of pregnancy. He found that 85 per cent of these babies were born between the 39th and the 42nd weeks.

The stillbirth rates, excluding congenital abnormalities, were:

<table>
<thead>
<tr>
<th>Weeks</th>
<th>Rate</th>
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<tbody>
<tr>
<td>39</td>
<td>15 per 1,000</td>
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<tr>
<td>40</td>
<td>4 &quot;</td>
</tr>
<tr>
<td>41</td>
<td>3 &quot;</td>
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<tr>
<td>42</td>
<td>10 &quot;</td>
</tr>
<tr>
<td>43</td>
<td>11 &quot;</td>
</tr>
<tr>
<td>44</td>
<td>45 &quot;</td>
</tr>
<tr>
<td>over 44</td>
<td>98 &quot;</td>
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The most common factors associated with these stillbirths, apart from the duration of pregnancy, were pre-eclampsia and hypertension. He observes that as pregnancy becomes prolonged the occurrence of disproportion increased, and prolonged labour, difficult forceps delivery, and caesarean section becomes more likely. He feels that we should be critical in accepting the theory of foetal anoxia from an ageing placenta as an adequate explanation of unexplained foetal death after term, and regards obstetric factors such as malpresentation, disproportion, dystocia, and long labour as more important.

Walker (1953, 1958) shows also that the stillbirth rate begins to
rise after the 41st week, and that with prolonged pregnancy and a difficult labour, the incidence of foetal distress and death rises markedly. His cord blood studies show that after the 40th week the oxygen content of the vein and artery begin to fall, and the haemoglobin concentration and oxygen capacity begin to rise, presumably as a compensatory mechanism. His opinion was therefore that “the postmature foetus is more likely to succumb to asphyxial death as the oxygen content of the blood is low despite the high capacity”. He states that an added risk is present with the combination of post-maturity and a patient who is primigravid, elderly, toxæmic, or hypertensive, or who has had an early threatened abortion. The multigravid with a bad obstetric history should not be allowed to go beyond term, and the patient who has a false labour at term and stops should be restarted immediately.

McClure Browne in his uterine blood flow studies shows that the uterine and placental blood flows are reduced towards the lower limits of normal when pregnancy becomes prolonged, and he too emphasizes the increased risk of postmaturity with hypertension.

When we consider the common association of occipitoposterior positions, disproportion, dystocia, and long labours in postmaturity, and if we add to this the probability of reduced placental function and poor foetal oxygenation, it becomes clear that postmaturity is to be regarded with considerable respect.

I think we should regard any pregnancy which is proved to have extended beyond 42 weeks as abnormal. Professor McClure Brown’s observation to me was that any woman who was seven days overdue should have a second opinion and a full obstetric assessment.

**Management of Labour**

*Induction of Labour*

The prevention of asphyxia neonatorum is so often dependent on termination of pregnancy that it is worth while considering this subject in some detail.

The risks involved in inducing labour must always be borne in mind. Maternal catastrophies, such as amniotic embolism, ruptured uterus, and fulminating infection, are rare enough, but one has to save a considerable number of babies by induction to compensate for a maternal death. Foetal risks are those such as infection and prolapse of the cord. The perinatal mortality is increased following induction of labour, though this may be largely related to the cause
of the induction.

The reason for inducing labour is most commonly that the baby is regarded as being in danger of intrauterine death, usually from asphyxia due to placental incompetence. If, therefore, there was a reliable test of early placental failure the induction of labour could be performed with much greater certainty than at present, when the probability of placental failure must be deduced from indirect evidence. Two tests have recently been described, and others are being developed. Vaginal cytology is a technique of examining cells taken from the upper third of the vagina. There is a normal smear of pregnancy which if it becomes abnormal towards the end of pregnancy is thought to indicate diminishing placental function. The other test is the assay of hormonal end-products such as pregnanediol in the urine, which may give an indication of placental activity.

Clinically, we may heed straws in the wind, such as the failure to gain weight in the last few weeks of pregnancy, or even the loss of one or two pounds. A reduction of the circumference of the abdomen at this time also may point to a failing placenta.

Rumbolz et al. (1961) reports on the syndrome of the small, full-term infant and placental insufficiency. This occurs in patients in whom the uterus does not enlarge in the last three months of pregnancy and it is often associated with toxaemia, hypertension, and renal disease. The results are small mature babies, small infarcted placentae, and in his series a 40 per cent foetal loss.

These are the general principles guiding induction. With regard to the detailed indications, labour is generally induced between 38—40 weeks in pre-eclampsia, hypertension, and chronic nephritis. The diabetic is induced from the 36th week onwards, depending on the size of the baby, and the co-incidence of other complications such as toxaemia. The pre-diabetic should not be allowed to go beyond 40 weeks. The patient with Rh antibodies is judged individually at the point where a rising titre is regarded as being more dangerous than prematurity. The history of previously affected babies also weighs heavily in the decision to induce early.

The post-mature patient is generally induced at 41 weeks if she is a primigravid over 30, or a multipara with a bad obstetric history, or if there are the slightest signs of toxaemia or hypertension. If the mother is entirely normal in all respects apart from being overdue, the policy at most hospitals is to induce at 10 to 14 days after
term, much depending on the estimated size of the baby. At Bristol it is the policy to induce at 38 weeks if the patient has had a threatened abortion after the third month.

The main contraindications to induction are maternal mitral stenosis, a transverse or oblique lie, and a patient of less than 36 weeks maturity.

The problem is to estimate the benefits of induction to the baby and balance them against the risks, and then state the indications for induction in writing in the notes. Nothing else clears the mind so effectively.

It is quite feasible to carry out induction at home if the case is well chosen, and this usually means the patient who will go into labour fairly quickly and deliver herself easily. The main danger one must avoid is a prolapsed cord due to a poorly fitting presenting part. At home this would almost certainly mean losing the baby.

For example Mrs S. B.; aged 34, this was her fifth pregnancy. She had one living child, the other three pregnancies having miscarried. She had a past history of thyrotoxicosis and began this pregnancy with a blood pressure of 140/60 mm. Hg. She progressed quite normally except for the raised systolic blood pressure. At 41 weeks she had not gone into labour and her blood pressure was 152/72 mm. Hg. A domiciliary consultation was arranged and at examination it was found that the cervix was ripe and soft. It was agreed that she should have a medical induction, and 24 hours later a fore-water rupture if labour had not begun. Labour did not begin so I performed an A.R.M. at 9.30 a.m. and by 3 p.m. she was fully dilated. She had a spontaneous delivery of an Apgar 9* baby at 3.30 p.m. with the aid of a generous episiotomy.

I regard a second opinion from a consultant colleague as an essential step in the management of induction at home. In the first place the indication for induction is critically reviewed, and in the second place a decision to induce labour is a decision to terminate pregnancy. This occasionally will require a forceps delivery, and about one in ten will require a caesarean section. It is important that the surgeon who will be called upon to do the section should have the opportunity of being consulted at the beginning of events.

The First Stage of Labour

Our main concern in the first stage is to watch closely for signs of foetal distress. These are not as many or as reliable as we would wish, but probably the most important sign is that of foetal bradycardia, particularly if the rate falls below 100.

Mayer (1953) states that bradycardia during the period of dilatation indicates severe anoxia and a poor prognosis. Walker (1959) considers that because we regard foetal heart slowing as a relatively

*See below
dangerous sign we lose very few babies in which this is noticed, but that because we have been unwilling to accept meconium staining and foetal heart rapidity as dangerous signs we lose more of these babies than we should. He stresses the importance of meconium staining associated with accidental haemorrhage and pre-eclampsia. Irregularity of the heart beat is an ominous sign.

We must listen to the foetal heart as frequently as possible during the first stage, but despite this in more than half of the babies dying in utero from anoxia no early change in the foetal heart is detected. Perhaps this situation will be improved by the use of devices such as that described by McRae (1962) who uses a system of continuous heart monitoring by a small abdominal microphone which is relayed to a loudspeaker in the patient’s room or sister’s office. Any change in the foetal heart rate or rhythm should be regarded as a late sign of foetal anoxia.

Our next concern in the first stage is to provide analgesia for the mother without depressing the foetal respiratory centre. Abramson (1960) states that probably all drugs cross the placenta, and the foetus may be four times as sensitive to respiratory depressants as an adult.

Pethidine is used extensively in obstetrics but it has some marked disadvantages. It is a powerful respiratory depressant with a long duration of action, and Corner (1962) points out that it has a marked cumulative effect, so that repeated doses given over a period of 12 to 14 hours before delivery often gives rise to an apnoeic baby. Mushin (1962) says that if pethidine is given to the mother within three hours of birth she should be given levallorphan before delivery. Pethilorphan is probably safer than pethidine but the addition of levallorphan reduces the analgesic effect as well as the depressant effect so that a bigger dose needs to be given. Pethidine seems to be given too much as a routine treatment now and this means that it is sometimes given when perhaps there is strictly no indication. I think it should be used with caution and especially so in those conditions where we expect placental impairment. I carry Lorphan (Roche) 5 mg. in a 5 ml. bottle. It is given as a 1 mg. dose intravenously or intramuscularly. It is effective against all morphine derivatives.

Much of the “pain” in the first stage of labour is really due to tension and apprehension. I have used intramuscular largactil (chlorpromazine) with good results in the first stage, but the hypotensive effect of this drug is perhaps undesirable. I intend to replace
it by sparine (promazine) in future. After these drugs have been used pethidine is often not required.

Hypnosis is probably the best analgesic in labour, but unfortunately not all patients are suitable subjects, and not all doctors can give the required time.

*The Second Stage of Labour*

This is the stage of uterine contraction and retraction, when the placenta is gradually being sheared off the wall of the uterus.

It is thought that the foetus can survive a maximum time of 20 minutes anoxia provided that it is fully oxygenated at the beginning of this period. In any of the conditions in which it is known that foetal oxygenation may be below normal, this period could be much less. If a baby is born asphyxiated we do not know if it has been anoxic for two minutes or 20, nor do we know what its tolerance was at the beginning of labour.

Oxygen given to the mother at 100 per cent concentration or even at pressures above atmospheric will provide a little extra oxygen to the foetus, and should always be given when foetal distress is detected or expected.

The treatment of the second stage should therefore do nothing to aggravate anoxia, it should not be unduly prolonged, and after birth the baby must be fully oxygenated as soon as possible.

The first point brings me to the "gas and air" machine which is in such general use. Nurse staggers in bowed down by the weight of a large wooden box and within a few minutes mother has her finger over the hole and is puffing away. One of my patients told me that she had seen two of me the previous evening when I had been present at her confinement. Very hard on her, I thought! She had been using this apparatus extensively and had been quite hypoxic.

Cole and Nainby-Luxmore (1962) show that at a pulmonary ventilation of 5 litres/minute the oxygen concentration from this machine is 12 per cent, but that at ventilation rates of 40—50 litres/minute it falls to 8 per cent, and these values are not uncommon in the excitement of the second stage.

It seems to me that there is little place for this apparatus in midwifery at the present time. In hospital, where oxygen is freely available, a mixture of 50/50 or 60/40 N₂O/O₂ has excellent analgesic powers and gives an oxygen concentration greater than atmospheric.
These mixtures are now on trial in a number of hospitals in the Lucy Baldwin gas-and-oxygen machine. The control can be locked at the required gas mixture so that lower concentrations of oxygen cannot be given inadvertently.

I called at the British Oxygen Company's medical department to enquire of the possibilities of a portable Lucy Baldwin machine. The snag here is that for equal quantities of nitrous oxide and nitrogen one needs a four times larger cylinder for the oxygen than for the nitrous oxide due to the differing physical properties of the gases, and I cannot see nurses coping with the resultant apparatus. An interesting development is the idea of mixtures of N₂O/O₂ in one cylinder in fixed proportions. Apparently they do not separate out as expected. At the moment it is not certain that the mixtures are stable under all conditions of use.

Trilene seems the best analgesic at present for home use. The inspired oxygen tension is not reduced, and trilene is approved by the Midwives Board under certain conditions. I carry a small, easily portable, cyprane inhaler which can be locked to give a set concentration of trilene.

A long labour is a common cause of asphyxia and foetal death, (N.P.M.S. 1958) and when full dilatation has been achieved delivery should be completed as soon as practical, especially in the cases when asphyxia is expected.

The Ventouse seems an excellent instrument in the hands of those experienced in its use (Chalmers 1962), provided that the application is not extended beyond a period of 40 minutes.

Forceps application in the home had become much less frequently employed in the past years in my practice of four doctors. This is perhaps due to a better selection of cases for hospital delivery or to the increasing realization of the dangers of general anaesthesia at home. It was shown in the Confidential Report on Maternal Deaths (1955) that the inhalation of vomit during general anaesthesia was a common cause of maternal death. There seems now no justification for a general anaesthetic at home, except as a domiciliary visit by a consultant anaesthetist who can intubate the patient and take full control.

However, since the description of pudendal block analgesia, a mid to low forceps extraction can be done with little risk to mother or baby, and perhaps with the "asphyxia risk" baby it should be done more often. Forceps should usually be applied to protect the
head of the premature baby, and for the aftercoming head of the breech.

A generous episiotomy should not be forgotten, as it will often prevent a head bobbing backwards and forwards for ten minutes or more on a tough perineum.

**Resuscitation**

The first requirement on delivering the baby is to make sure he does not inhale whatever may be in his mouth, whether liquor, mucus, or meconium. Karlberg (1960) shows by radiographic studies that the thorax is compressed during its passage through the birth canal, which causes the expulsion of liquor from the upper respiratory tract. This is followed by a sudden expansion as the thorax is born, and now air is drawn into the whole respiratory system.

If the infant's head is kept low during delivery and a little gentle suction is applied to the mouth and pharynx, there is little danger of inhalation of this extruded material.

Butler quotes in the National Perinatal Mortality Survey that of the 451 infants who died of intrapartum asphyxia, one third showed meconium aspiration visible to the naked eye.

The next requirement is to make an accurate assessment of the baby's condition. The system coming into most general use is that of Virginia Apgar (1953). It is a detailed examination of the baby which takes place one minute after delivery. My memory is becoming a little pre-senile so I keep a pasteboard card in my bag with the chart as follows:

<table>
<thead>
<tr>
<th>Score</th>
<th>0</th>
<th>1</th>
<th>2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate</td>
<td>Absent</td>
<td>Below 100</td>
<td>Above 100</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Absent</td>
<td>Irregular</td>
<td>Regular</td>
</tr>
<tr>
<td>Muscle tone</td>
<td>Limp</td>
<td>Moderate</td>
<td>Active movements</td>
</tr>
<tr>
<td>Reflex irritability (pinch foot)</td>
<td>None</td>
<td>Grimace</td>
<td>Cry</td>
</tr>
<tr>
<td>Colour</td>
<td>Blue or white</td>
<td>Body pink. Extremities blue</td>
<td>All pink</td>
</tr>
</tbody>
</table>

Scores are added together one minute after birth
The inferences which may be drawn from the Apgar score are:

- **8—10** Normal baby
- **4—7** Will need a moderate amount of resuscitation
- **0—3** Will need everything available.

A simpler classification is that a baby who is blue, has some muscle tone, and heart rate above 100, should do well, but a baby who is pale, flaccid, and has a heart rate under 100 is in grave danger.

The assessment of the infant's condition is repeated every two or three minutes and a record of improvement or deterioration can be clearly shown. This is where the value of the Apgar score is evident. It is useful to have a large clock on or near the resuscitation table, as at the Postgraduate Medical School, Hammersmith, as time can pass on winged feet.

The equipment one would like to have at hand is as follows:

1. A flat surface to receive baby, such as a small table covered with a napkin. Never try to work in a cot.
2. A mucus catheter with a mucus trap and a soft rubber tube. Donald (1956) is strongly in favour of a mechanical sucker, but I would like to differ with him on this point. My sucker always works.
3. A couple of small infant oropharyngeal airways.
4. A source of oxygen at reduced pressure. The Monmouthshire County Council supply their midwives with a Sparklet apparatus described in *The Lancet* by Waller and Morris (1953). This consists of a small box containing two Sparklet oxygen cylinders of 20 minutes duration, a reducing valve giving a one litre/minute flow, and a rubber bag leading to a rubber funnel. It was initially designed for the administration of intragastric oxygen, though seldom used for that purpose now.
5. An infant laryngoscope. I use the Seward pattern.
6. A neonatal endotracheal tube. William Warne produce a disposable polyvinyl tube which is marketed in a sealed, sterile, transparent envelope. This was described in *The Lancet* by Hamer Hodges and Tunstall (1961).
7. Injection of Lorfan (levallorphan) or Lethidrone (nalorphine).

**Treatment**

*Moderate asphyxia.* Baby is gently sucked out following delivery and carried to the table wrapped in a napkin and held slightly head down, with the head towards the operator. Further oropharyngeal toilet is carried out, and oxygen is gently blown over his face with funnel or tube. His condition is assessed and he is watched. The most difficult thing to do at this time is to leave baby alone. Keep as much of baby covered as is possible as he quickly chills at birth and may become hypothermic.

If respiration is accompanied by sucking in of the chest wall and
there is no improvement in colour, it is probable that the larynx is obstructed, and it is not uncommon to find at post mortem a blob of tenacious, glue-like mucus or meconium. The larynx should be inspected under direct vision and sucked out in these cases.

There is some divergence of opinion on the advisability of general practitioners performing laryngoscopy. Mushin (1962) feels that in unaccustomed hands there is danger of doing more harm to the infant's larynx than the expected benefit warrants. On the other hands, paediatricians (Corner, 1962), obstetricians (McClure Browne, 1962), and other anaesthetists (Davies, 1962) state that a general-practitioner obstetrician should carry a laryngoscope and be able to use it. The latter in fact advocates that its use could be taught to midwives. The correct selection of babies for laryngoscopy seems to be the solution. One such as described above will surely die if its airway is not cleared.

If there are grounds for believing that the baby is suffering from respiratory depression from morphine or pethidine an injection of either 1 mg. Lethidrone or 0.5 mg. Lorfan should be given into the umbilical vein. This injection should be given with great care as Hudson, McCandless and O'Malley (1950) report 20 cases of sciatic paralysis following injections of nikethamide into the cord. The injections were probably given into one of the umbilical arteries and back tracked into the sciatic artery, which is the axial artery to the limb at this stage. I believe any material can cause this trouble, which may lead to loss of the limb. This seems all the more reason to give these drugs to mother before delivery.

Intragastric oxygen is little used now. The oxygen which disappears from the tube is partly metabolized by the intestinal mucosa, and part overflows into the trachea. There are complications such as gastric erosion and rupture of the stomach and there is no provision for removal of CO₂. Acidosis due to accumulation of CO₂ and lactic acid is almost as dangerous as oxygen lack to these babies. After ten minutes the majority will have established spontaneous, regular respiration. Those who are deteriorating will be treated as the severely asphyxiated babies.

**Severe asphyxia.** In addition to the treatment already described, these babies will need artificial ventilation. It will not be enough to get oxygen to the trachea, although this may keep baby pink, because it is equally important to remove carbon dioxide.

An endotracheal tube should be introduced under direct vision
after the larynx has been sucked out. This is now connected to the portable oxygen cylinder. For safety there should be some kind of valve between cylinder and baby, set to blow off at about 20 cms. of water. The initial pressure required to open the alveoli has been measured at between 60—80 cms. of water and one puff may be given at that pressure if baby has not breathed, but only a quarter of that pressure is required subsequently.

It should be emphasized that these babies are completely flaccid and intubation is not unduly difficult, in fact if baby offers any resistance to intubation it may well not need it. Practice in intubation may be obtained on a fresh stillbirth, and I have an arrangement with the obstetric houseman at the local hospital to inform me if one should be available. An alternative method is for the operator to take a mouthful of oxygen from the cylinder with each breath and blow this down the tube.

A method which is regarded by Tizard (1962) as almost as effective as intubation is mouth-to-mouth inflation with a hand on the abdomen to prevent inflation of the stomach. The infant oropharyngeal airway keeps baby’s lips out of the way. The operator uses oxygen from the cylinder as above. One can practise with a manometer to learn the correct strength of puff.

If the heart beat is absent or should fail one can perform external cardiac massage in addition to pulmonary inflation with oxygen. Reilly and Melville (1962) report a successful recovery following open cardiac massage.

Summary

It is difficult to summarize an article such as this. If one had to select the most important points one might choose:

1. Beware of hypertension, postmaturity, increasing maternal age, and primiparity, especially any combination of these factors.

2. The maximum oxygenation and the minimum delay in “asphyxia risk” deliveries.

3. Learn to use an infant laryngoscope.

4. Do not delay the administration of oxygen. Regard a cyanosed baby as you would a cyanosed adult.

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