INDUCTION OF LABOUR

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'The first account of any artificial method of bringing on premature labour was given to me by Dr. C. Kelly. He informed me that about the year 1756 there was a consultation of the most eminent men in London at that time, to consider of the moral rectitude of, and advantages which may be expected from, this practice, which met with their general approbation. The first case in which it was deemed necessary and proper fell under the care of the late Dr. Macaulay, and it terminated successfully. (The patient was the wife of a linen-draper in the Strand.) The thing has often been the subject of conversation, and proposed by writers, but some have doubted the morality of the practice; and the circumstances which may render the operation needful and proper have not been stated with any degree of precision' (T. Denman, 1801).

It is appropriate that induction of labour should be the obstetric subject chosen for this symposium from Bristol. The catheter for puncture of the membranes designed by Drew-Smythe (1931), then Professor of Obstetrics at Bristol, achieved world-wide recognition and use. James Hamilton, of Edinburgh, 100 years before, had described a method of rupturing the membranes at some distance from the external os by means of a catheter and stylet. Indeed, even before that, in 1810, he had suggested digital separation of the membranes from the lower segment. Even today all the methods used are indeed artificial, and will remain so until a satisfactory physiological explanation, capable of imitation, of the causes of the onset of labour has been found.

The Onset of Labour

The discovery of hormones led us to anticipate that this physiological explanation of the causes of the onset of labour would not be long in coming. It has been known for some time that progesterone desensitizes the uterine muscle to the action of the posterior pituitary oxytocic factor, and that oestrogen sensitizes. However, the giving of large doses of oestrogen to women in late pregnancy does not initiate labour, even when followed by oxytocic administration. It is true that in some cases where the foetus is already dead this method will achieve success, but certainly not when the foetus is alive. The problem, therefore, is a complicated one.

Recently Csapo (1961), as a result of his experiments at the Laboratory of the Physiology of Reproduction, the Rockefeller Institute, New York, has propounded the progesterone block theory. He has shown that while the contractile capacity of the myometrium increases during pregnancy the contractile response cannot spread from one cell to the next. It is blocked by progesterone in the presence of intact placental function, and it is believed to be a direct effect of placental progesterone in the muscle cells in contact with the placental site. There is thus a progesterone concentration gradient in the myometrium dependent on distance from the placental site. As a result of this Csapo has been able to produce selective delivery of the fetuses from the two horns of a rabbit's uterus. He suggests that 'the progesterone block offers only a relative defence which is inadequate if the uterine volume becomes greater than the defence mechanism can balance', and he concludes 'that effective uterine activity is controlled by the ratio \( V/Pm \) where \( V = \) volume and \( Pm = \) metrial progesterone concentration. If this ratio is small, pregnancy is maintained, and if it is large pregnancy is terminated. . . Thus twin pregnancy, hydramnios, and toxemia would predispose to premature labour, for the first two increase \( V \) while the third is thought to reduce \( Pm \). This theory does not have to consider the state of the cervix, and indeed, Wood, Booth and Pinkerton (1962) found that, in cases of fetal death in utero or fetal conditions incompatible with life, the intra-amniotic injection of 50% glucose successfully induced labour even when the cervix was not 'ripe' (taken up and ready to dilate).

It would appear, therefore, from what has been said above that 'if we are to imitate this regulating device we must develop a local progesterone therapy' (Csapo, 1961). In the meantime we are left with the other methods of inducing labour as follows:

Methods of Induction of Labour

A. Medical

Oxytocic drip ± previous oestrogen priming.
B. Surgical
   (a) Intra-amniotic injection.
   (b) Sweeping of membranes.
   (c) Rupture of forewaters.
   (d) Caesarean section.

C. Combined Medical and Surgical
   (a) Rupture of forewaters followed by oxytocic drip.
   (b) Oxytocic drip followed by rupture of forewaters.
   (c) Caesarean section after a or b.

A bath and enema will be the necessary preparation of the patient before any of the above methods. I have not permitted the use of castor oil for many years. It is unpleasant to take, unpleasant in its effects, in particular causing painful hemorrhoids in the early days of the puerperium, it is too easily prescribed a second or third time by those who do not have to take it, and its action on the uterus is questionable.

Previous Estrogen Priming

This is only in cases of intra-uterine death. The pregnant woman tolerates large doses of oestrogen, and is prescribed stilboesterol, 50 mg. four-hourly, for 48 hours.

Oxytocic Drip

This should only be done in hospital where the patient must be carefully observed. Ideally, the oxytocin-sensitivity test (Nixon and Smyth, 1959) should be carried out as a preliminary, but few hospitals have the apparatus for doing it.

First make sure that: (1) There is a longitudinal lie; (2) there is no cephalopelvic disproportion; (3) there are no factors predisposing to ruptured uterus; and (4) that chloroform is available.

Procedure

Use 5% dextrose and not saline in all patients. Put 1 Unit of Syntocinon into the first bottle (550 ml.). Start at 12 drops per minute and increase the rate at half hourly intervals to 24, 40 and 60 drops per minute or until satisfactory contractions are established without embarrassing the foetus. Put 5 Units in the second bottle, start again at 12 drops per minute (i.e. the same dose as 60 per minute of the first bottle) and work up gradually as before. Five Units per bottle is the maximum concentration used.

The Syntocinon drip should be kept going until the end of the 3rd stage. There is a tendency for it to stop late in the 1st stage when the patient becomes restive.

The drip may be set up in the patient’s room and provided there is no alteration in the foetal heart in the first half-hour there should be no difficulty. If there is alteration in the foetal heart, stop the drip.

Dangers

(1) Tonic uterine contraction, which is relieved by stopping the drip and rapidly inducing anaesthesia with chloroform.
(2) Foetal distress or death because of the strong uterine contractions.
(3) Ruptured uterus.

The patient must, therefore, be watched very carefully and quarter hourly recordings made of pulse, foetal heart-rate, uterine contractions (both frequency and their duration), and drip-rate. The blood pressure is taken half hourly.

No vaginal examination is to be done except by a doctor in the early stages of ‘oxytocic drip labour’.

Cyclopropane anaesthesia after an oxytocic drip is very dangerous as it may cause ventricular and cardiac arrest. If the patient—possibly because of foetal distress—comes to a Caesarean section, the anaesthetist must be made aware of this danger.

Rupture of the Forewaters

By artificial rupture of the membranes is meant rupture of the forewaters with Kocher’s forceps preceded by a membrane sweep. Dangers to be avoided are:

(1) Infection—Swabbing the vagina and cervix carefully in addition to the vulva with Hibitane cream before doing the vaginal examination is most important. This necessitates passing a speculum.
(2) Prolapsed cord—There is a risk of cord prolapse when artificial rupture of the membranes is carried out on patients with a presenting part which does not fit the lower segment closely, e.g. hydramnios, twins, occipitoposterior position, breech, and in multiparous patients with a floating foetal head above the brim. One can avoid this risk by guiding down the presenting part bimanually and allowing the liquor to escape slowly. Membranes should not be ruptured if there is any suspicion of cord presentation.
(3) Detaching a low-lying placenta.
(4) Injury to the genital passage.
(5) Injury to the foetus.

If the patient is not in labour 24 hours after rupture of the forewaters:

(1) Send a catheter specimen of urine, throat swab and high vaginal swab to the laboratory for culture and sensitivity.
(2) Prescribe chloramphenicol, 500 mg. stat. and 250 mg. six hourly for 5 days. (To
minimize risk of intra-uterine fetal pneumonia and genital infection.)

(3) Proceed to an oxytocic drip in pre-eclampsia cases: in postmaturity cases this is done after 48 hours.

Hind-water puncture of the membranes by a Drew-Smythe catheter has been abandoned. It is not without risk, for example, hitting the placental site and causing separation and bleeding, and the method is not so certain in bringing on labour as forewater rupture (Nixon and Smyth, 1959).

Since the majority of cases will have rupture of the forewaters it is well to describe the procedure in detail as follows:

The blood pressure should be recorded. The abdomen is carefully palpated and the findings noted. The fetal heart rate is counted. After vulval and vaginal cleansing in the lithotomy position vaginal examination is done. The presenting part is confirmed, pelvic assessment is repeated, and placenta previa excluded. The length and dilatation of the cervix is stated and the index finger is inserted into it. Once again the presenting part is felt. It may be necessary for an assistant to push on the fundus of the uterus at this stage. The membranes are swept off the internal os as far as possible. Then the long Kocher’s forceps (or Smyth’s amnionotomy forceps) are inserted alongside the finger and the membranes seized. No liquor may be seen but a hair from the baby’s head will have been included in the tip of the forceps, thus confirming rupture of the membranes. Occasionally a sacral position of the cervix is encountered and difficulty is experienced in getting the finger into the cervix. In such a case it will be necessary to expose the cervix with a speculum and to place a swab-holding forceps on the anterior lip of the cervix. Having done this the speculum is withdrawn and the finger is inserted into the cervix using the forceps on the cervix to pull it downwards towards the finger. If liquor escapes in a rush the finger should be kept in the cervix to allow slow release and to feel the presenting part descending. The fetal heart rate is counted again and at quarter-hour intervals for the following hour. If the liquor is meconium-stained the fetal heart rate should be continuously observed and a decision reached as to whether Cesarean section is indicated.

**Indications for Induction of Labour**

**A Maternal**

- Pre-eclampsia.
- Eclampsia.
- Essential hypertension.
- Renal Disease.

**B Fetal**

- Diabetes Mellitus.
- Intra-uterine death of the foetus.

**Pre-Eclampsia and Hypertension in Pregnancy**

The indications for induction of labour in such cases are:

1. To cure eclampsia after control of fits.
2. To cure accidental hæmorrhage.
3. To cure pre-eclampsia (to prevent eclampsia and (or) accidental hæmorrhage).

**Acute case**

(a) Rising blood pressure despite treatment.
(b) Increasing albuminuria.

**Acute-on-chronic case**

(a) Rising blood pressure.
(b) Accidental hæmorrhage.

**Chronic case**

(a) Maintenance of blood pressure at toxaemic level (140/90 mm. Hg or above) under treatment and gestation at least of 36 weeks’ duration.
(b) Continuing slight or recurrent slight albuminuria, not exceeding 14 days’ duration.
(4) To avoid placental insufficiency: Cured pre-eclampsia however mild or recurrent; gestation at least of 36 weeks’ duration.

Despite these indications there is a tendency amongst doctors to delay induction further because the baby is too small. The size of the baby in utero (or the early stage of gestation) plays no part in the assessment of the case for induction.

Postmaturity

Walker (1954) has shown that 'The percentage saturation of the blood in the umbilical vein in the latter half of normal pregnancy is 70 at or about the 30th week, 60 at 39-40 weeks, and falls to under 30 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 30-40% 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-43rd week the supply is dangerously low in almost all fetuses'. He says:

'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 fetal 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, Cesarean section is seriously considered on the indication of “fetal distress”. With the already anoxic fetus, Cesarean section is often preferred if forceps delivery would be difficult, and might put an undue strain on the fetus.

'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 Cesarean section is performed.

The combination of pre-eclampsia or eclampsia and postmaturity is lethal to the fetus.

The larger postmature child can cause cephalopelvic disproportion requiring Cesarean section or difficult forceps delivery. It can cause difficulty with the delivery of the shoulders, and gross lacerations of the vagina, perineum, and rectum. Postmaturity can cause maternal and fetal damage.

Theobald (1959), discussing the choice between death from postmaturity or prolapsed cord and life from induction of labour, states: 'The number of healthy mature babies that can be saved by inducing labour is of the order of 1.2% of all hospital deliveries, of which 0.6% are saved from death from postmaturity. Many “unnecessary” inductions will of course be done, an occasional cord will prolapse and a few women will fail to go into labour in a reasonable time. . . . The surgical induction of labour is not meddlesome midwifery but the imitation of Nature. It is associated with a perinatal mortality of the order of 0.5%, and deserves a wider use'.

Lennon (1957), in a series of 987 surgical inductions, combined when necessary with the oxytocin drip, achieved a corrected perinatal mortality rate of 1.6%, and a Cesarean section rate in the series of 1.94%; 78% of the patients were delivered within 48 hours of induction. The author in the years since has found no reason to alter his procedure which follows that of Walker detailed above. The figures quoted remain much the same.

Prevention of Stillbirth in Rh Haemolytic Disease

Of mothers who have not previously carried an affected baby, 25-30% show a critically high titre of Rh antibodies, and in such cases the risk that the pregnancy will end in a dead baby is approximately 30%. Out of every three stillbirths in these cases, two occur after the 37th week of the pregnancy, and it is, therefore, recommended that first affected babies be delivered 21 days before term if the mother has a critically high titre of Rh antibodies determined by the indirect antiglobulin technique.

When the mother has already carried an affected baby, the antibody titre during the pregnancy, considered in conjunction with the severity of haemolytic disease in the previous child, is of value in determining whether and at what time of gestation premature delivery is indicated (Tovey and Valæs, 1959).

Cephalopelvic Disproportion

There is little argument against induction of labour for this reason in the multipara. On the other hand, trial of labour in the primipara has been basic practice in this country since Holland (1920) emphasized cranial stress in the fetus during labour and the effects of excessive stress on the intracranial contents, namely torn tentorium cerebelli and subdural cerebral haemorrhage. But there has been in the meantime a tremendous improvement in the diet of the community, especially preventing rickets, so that contracted pelvis is not nearly so common. Further, sepsis is no longer the scourge that it was. Consequently, there has been recourse to induction of labour in cephalopelvic disproportion in the primigravida...
(MacLennan, 1954; Black, 1960). MacLennan, indeed, found that the results in cases of trial of labour when labour occurred spontaneously were not at all good, and he advocated in cases of apparently slight disproportion that induction of labour should be carried out between the 36th and 38th weeks, and thereafter followed by a trial of labour. Black assesses his cases of disproportion under general anaesthesia at the 38th week, and the forewaters are ruptured if:

(a) There is no disproportion or only a mild degree of it.
(b) The child is of reasonable size.
(c) The vertex is presenting.
(d) The mother is not elderly and she has shown herself to be of reasonable fertility.
(e) If not more than one previous Caesarean section has been carried out.

With regard to the secundipara the author would emphasize one point. Some doctors tend to accept the birth of one baby as proof that all is well with the mother's pelvis. But so many primigravidae have a small baby due to pre-eclampsia that there may be quite gross disproportion due to a bigger baby in the second pregnancy, and unless pelvic assessment is carried out carefully in such cases trouble may ensue if labour is allowed to start at term or beyond. Thus, beware the woman who has had one and only one small baby.

Results

In Bristol in the university department over a five-year period 2,770 inductions of labour by rupture of the forewaters have been carried out. These inductions were done for the following reasons:

<table>
<thead>
<tr>
<th>Induction Type</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-eclampsia and other forms of hypertension</td>
<td>47.8</td>
</tr>
<tr>
<td>Postmaturity</td>
<td>33.4</td>
</tr>
<tr>
<td>All other indications</td>
<td>18.8</td>
</tr>
</tbody>
</table>

(Total deliveries over 5 year period 6,881).

The dangers to the mother, as far as we could see, were as follows:

<table>
<thead>
<tr>
<th>Induction Type</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prolonged induction-delivery interval (over 48 hours)</td>
<td>20.0</td>
</tr>
<tr>
<td>Infection</td>
<td>1.4</td>
</tr>
<tr>
<td>Prolapse of cord</td>
<td>0.4</td>
</tr>
<tr>
<td>Prolonged 1st stage of labour (over 24 hours)</td>
<td>3.5</td>
</tr>
</tbody>
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The results so far as the foetus is concerned have been as follows:

<table>
<thead>
<tr>
<th>Induction Type</th>
<th>%</th>
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</thead>
<tbody>
<tr>
<td>Total number of inductions</td>
<td>2,770</td>
</tr>
<tr>
<td>Stillbirths</td>
<td>61</td>
</tr>
<tr>
<td>Neonatal deaths</td>
<td>18</td>
</tr>
</tbody>
</table>

This gives an uncorrected rate for stillbirths and neonatal deaths of 2.85%. Out of 79 babies lost, 24 were grossly abnormal and of the 55 normal babies, 18 weighed under 4 lb. Of the deaths 41.8% were after induction for pre-eclampsia or hypertension; 8.9% were after induction for post-maturity.

The oxytocic drip method has been criticized, but our results, supporting Theobald (1959), are as follows:

- Total cases in which oxytocic drip was used: 318
- Cases ending in loss of the baby: 24
- Gross congenital defect: 10
- Gestation under 28 weeks: 2
- Babies under 4 lb. weight (including two under 28 weeks): 9

Deaths related to indications for induction:

- Pre-eclampsia, hypertension, and one eclampsia: 9
- Postmaturity: 2
- Fetal abnormality: 10
- Accidental haemorrhage: 3
- Overall Caesarean section rate: 2.4%
- Forceps-rate (including prophylactic forceps delivery in pre-eclampsia and fetal distress): 13.5%

Comment

It is obvious that many needless inductions of labour are carried out, but it is very difficult to select the proper cases for induction—that is, those who would get fetal distress during labour and/or still birth or cerebral anoxia. Our induction figures have been comprehensive rather than particular. There is no doubt that this programme of induction of labour by rupture of the forewaters and the release of liquor amnii has led to quicker labours, by shortening the first stage. This does not mean an increase in the Caesarean section rate because of those cases that failed to go into labour. In 11.5% of cases an oxytocic drip was used in conjunction with rupture of the forewaters, and then labour ensued. One of the interesting points is that there have been few cases of really difficult forceps delivery—uterine action seems to have improved and consequently so has the mechanism of labour.

Until an easy method is devised for estimating the degree of placental insufficiency, particularly in cases of pre-eclampsia and postmaturity, induction of labour by rupture of the forewaters ± oxytocic drip offers in our hands a method of delivery which is safe for the mother and safer for the baby than the hazards of placental insufficiency due to pre-eclampsia, hypertension and postmaturity. 'Leaving it to Nature' does not produce comparably good figures. But leaving statistics aside, the good results recorded above
have been achieved easily and without the worries of prolonged labour and difficult forceps delivery.
The same results can be achieved in two ways—the hard way with ‘blood, and sweat, and toil’ or the easy way described here.
A great deal remains to be done on this subject. A little has been done in retrospect by the paediatrician, but the obstetrician is in the position to undertake more research on the protection of the nervous system during pregnancy and labour. For example, a more reliable method is needed for estimating placental insufficiency. Radioactive isotopes have been used for this but have their own dangers and have not given the results expected. The obstetrician should pay particular attention to the carbon-dioxide tension and the effects of maternal sedation on the baby during the administration of analgesic drugs and anaesthesia.

Whether the practice described above, of increasing the number of inductions of premature labour, is as good as it seems requires further investigation, especially whether there are more spastic children from such cases than when labour has not been accompanied by the release of liquor amnii. The causes of prematurity and the damage done to the nervous system by early release from the uterus should also be investigated.
A cure for pre-eclampsia, the cause of which still remains obscure, must be found. The subject cries out for research, and the co-operation of paediatrician, anaesthetist and obstetrician should be close. Indeed, the programme from this point of view should be co-ordinated.
It is hoped that research will soon find an easy and certain method, preferably medical, for induction of labour at all stages of gestation.

REFERENCES
Tovey, G. H., and Valaes, T. (1959): Prevention of Stillbirth in Rh Haemolytic Disease, Ibid., ii, 521.