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Prolonged pregnancy hazards and management

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Introduction

The occurrence of prolonged pregnancy in both the human and animal species has for long been accepted. There exists doubts in the minds of some obstetricians as to the possible hazards of this syndrome, and the need for active interference in its management. However, majority opinion, at present, is in agreement that the prolonged pregnancy syndrome, if allowed to be untreated, does contribute directly to increasing perinatal mortality, and indirectly to both foetal and maternal morbidity.

Review of Experimental and Clinical Research

As early as 1934, Sir Joseph Barcroft and his colleagues had shown that in the foetuses of goats and sheep there was deterioration of the oxygen supply as pregnancy proceeded towards, and beyond, the normal term. Still later, Barcroft and Maureen Young (1945) found that there was deficient oxygen saturation in the blood of the cerebral venous sinuses in postmature rabbit foetuses. Barcroft (1946) hinted that the postmature human foetus might be in a similar position as regards oxygen supply, and this possibility was also emphasised by Traut (1946) and Eastman (1954).

Interests in the possible hazards of the prolonged pregnancy syndrome in the human pregnancy was revived by McKiddie (1949) who suggested specifically that a falling oxygen supply might explain some of the special features seen in his cases of prolonged pregnancy syndrome. In 1949, guided by the work of Barcroft, and stimulated by the clinical findings of McKiddie, Walker and Turnbull in Aberdeen conducted extensive studies on the oxygen saturation in the cord blood of the human foetus. In 1953, they published their findings, which showed that the average oxygen saturation of foetal haemoglobin was about 70 per cent at the 30th week of gestation, and with the advance of gestation the oxygen saturation steadily fell to reach 60 per cent at the 40th week, but thereafter the fall was very steep—the oxygen saturation being only 30 per cent at the 43rd week. They concluded that the excess of foetal deaths in prolonged pregnancy could be due to a falling oxygen supply.

Clifford (1954) states that the prolonged pregnancy syndrome is a problem only in the primigravidae. Most authorities, who subscribe to the hazards of prolonged pregnancy, consider it to be more so if the patient is older. In this context, Turnbull and Baird (1957) have shown that the prolonged pregnancy syndrome constituted a special hazard in the primigravidae aged 25 years and over.

More recently, Browne (1963) in his Joseph-Price Oration entitled "Postmaturity", reviewed the clinical and autopsy data, arising out of the National Perinatal Mortality Survey conducted in England and Wales in March 1958. From his study, he concluded that the prolonged pregnancy syndrome constituted a definite practical problem, in view of the concomitant hazards that may arise. To quote his own words: "placental insufficiency appears to be a reality in prolonged pregnancy, or at least in some cases of it. It is probably reasonable to say that every pregnancy, if it goes on long enough without delivery, will end in antepartum death, but that some placentas become inadequate sooner than others, earlier failure

of the placenta being encouraged by the presence of toxaemia, increased maternal age, and possibly by some familial factor not yet determined. On the other hand, some placentas do not age nearly so soon, and in these cases the baby will, of course, continue to grow. In these cases disproportion may well arise, and the association of disproportion and disordered uterine action with labour in prolonged pregnancy is well known." So much then, for the protagonists' viewpoint on prolonged pregnancy.

However, there are several authorities, who hold diametrically opposite viewpoints. Bancroft-Livingston and Neill (1957) from Belfast were unable to confirm the experimental findings of Walker and Turnbull (1953) on deficient oxygen saturation of foetal haemoglobin in their cases of prolonged pregnancy. Hence the Belfast School (Bancroft-Livingston and Neill, 1957; Macafee and Bancroft-Livingston, 1958) holds the view that the problems of placental insufficiency, intrapartum asphyxia and foetal dysmaturity are no extra hazards of the prolonged pregnancy syndrome! Gibberd (19 58), although accepting the problem of prolonged pregnancy, states that more babies are lost as a result of induction for postmaturity than are saved by it!.

Hazards of the Prolonged Pregnancy Syndrome

In the light of our present-day knowledge, the increasing foetal and maternal morbidity, and foetal mortality, associated with untreated prolonged pregnancy, may be the sequelae of any one or more of the three hazardous syndromes that can be a co-existent feature of this condition.

TABLE I

Hazardous Syndromes of Prolonged Pregnancy

1. Placental Insufficiency Syndrome
2. Uterine Dysfunction Syndrome
3. Disproportion Syndrome

1. Placental Insufficiency Syndrome:

The occurrence of the syndrome of placental insufficiency in patients with prolonged pregnancy is now an accepted fact. There is a mass of clinical and laboratory experimental evidence in support of this fact, and some of these evidences have been cited in the preceding section of this paper; but of outstanding significance is the epidemiological data arising out of the 1958 National Perinatal Mortality Survey in the United Kingdom (Browne (1963); Claireaux (1961) and (1963); Butler and Bonham (1963)).

Placental insufficiency will, in its turn, contribute to a state of intra-uterine foetal hypoxia, which, if severe enough, can precipitate foetal death. Such foetal deaths are of asphyxial origin, and may occur during labour, soon after birth, or less commonly even before the onset of labour itself. Impaired placental function may also contribute to a higher incidence of foetal distress in labour, which can lead to an increasing incidence of operative delivery, be it caesarean section, forceps or ventouse delivery, so as to salvage the asphyxiated infant from its hostile environment.

2. Uterine Dysfunction Syndrome:

That the uterus in labour functions less efficiently in those cases of prolonged pregnancy, has been pointed out by many authorities (Masters and Clayton (1940); Walker (19 58); Lindren, Norman and Viberg (1958); and Browne (1963)). This state of inefficient uterine action results in a higher incidence of the uterine dysfunction syndrome, which in its turn will contribute to a higher incidence of prolonged and difficult labour. Such labour may precipitate foetal or maternal distress, and both of these will be indications for an increase in the rate of operative and difficult delivery, be it caesarean section, forceps or ventouse. The foetal distress, if severe and not promptly dealt with, may predispose to a higher perinatal mortality rate.

3. Disproportion Syndrome:

In those cases of prolonged pregnancy syndrome, where placental failure does not occur or is minimal, the foetus will continue to

grow as the pregnancy becomes prolonged. In such instances, the foetal skull also grows, not only in the direction of skull enlargement but more important in the direction of hardening of the foetal skull. This state of enlargement and hardening of the foetal skull, on the one hand, will contribute to an increasing incidence of cephalo-pelvic disproportion, which will necessitate a rise in the operative delivery rate, both of caesarean section and forceps delivery. On the other hand, even in the absence of major disproportion, the labour in these cases are likely to be prolonged with subsequent development of both foetal and maternal distress. Such distress will again predispose to a higher incidence of operative delivery, and perinatal mortality.

It thus becomes apparent that three hazardous syndromes of untreated prolonged pregnancy will all ultimately contribute to any one or more of the following sequelae *viz.*:—

(a) An increasing incidence of foetal distress.
(b) A rise in the perinatal mortality rate.
(c) An increasing incidence of difficult and operative delivery.

These were, in fact, the conclusions reached by Walker (1958) from his study.

The results of Walker's (1958) study are summarised in Table II, and they clearly indicate that both the incidence of foetal distress and perinatal mortality are higher in the group of prolonged pregnancy cases as compared to the control group.

TABLE II

Results of Walker's Study in Aberdeen

A. FOETAL DISTRESS:	
1. Slowing of Foetal Heart	**Incidence**
Babies delivered between 39-40th week - -	7%
Babies delivered after 42nd week - - -	12%
2. **Meconium Stained Liquor Amnii:**	
(i) **Before the onset of labour:**	
Cases induced 2 weeks before term - -	4%
Cases induced after the end of 40th week -	10%
(ii) **In Labour (where initial liquor at ARM was clear):**	
Cases induced 2 weeks before term - -	4.5%
Cases induced after the end of 40th week -	9%
B. PERINATAL MORTALITY (1948-52):	
Perinatal Mortality for deliveries in 40th and 41st week of gestation -	1.2%
Perinatal Mortality for Deliveries after the 42nd week of gestation -	2.8%

Management of Prolonged Pregnancy:

Before treatment can be instituted in any case of prolonged pregnancy, it is essential for the obstetrician to ascertain for himself the diagnosis with particular reference to the following cardinal features *viz.*:—

(a) Certainty of Dates
(b) Viable size of Foetus
(c) Impaired state of Placental Function
(d) Presence of Disproportion

As my predecessor, Mr. T. H. Lean, has already covered these diagnostic aspects, I shall not elaborate on these features.

TABLE III

Management of Prolonged Pregnancy

1. Induction of Labour:
(i) Medical—Oxytocin Medication
(ii) Surgical—Amniotomy
2. Caesarean Section:
(i) Elective
(ii) Emergency
3. Assisted Vaginal Delivery:
(i) Forceps Delivery
(ii) Ventouse Delivery
(iii) Breech Extraction

In Table III (above), the salient aspects in the management of the prolonged pregnancy syndrome have been outlined.

1. Induction of Labour:

In the management of prolonged pregnancy syndrome, induction of labour still remains the chief weapon in the armamentorium of the obstetrician. In the light of our present-day knowledge, and in particular the findings of the 1958 National Perinatal Mortality Survey in the United Kingdom (Claireaux (1963); Butler and Bonham (1963)), it is considered unwise to allow cases of uncomplicated prolonged pregnancy to go untreated far beyond the 42nd (T+14 days) week of gestation. Active interference to terminate such pregnancies is considered justifiable to reduce perinatal mortality, and both maternal and foetal morbidity. However, when cases of prolonged pregnancy have other pathological stigmata coexisting, then the termination of pregnancy before the end of the 42nd week of gestation is not only justified but also becomes imperative. In particular, the presence of such stigmata as toxaemia of pregnancy, chronic hypertension, chronic pyelonephritis, elderly primigravida and multiple pregnancies, may prove lethal to the foetus, if such pregnancies are not terminated well before the end of the 42nd week of gestation.

Induction of labour can be effected by either medical or surgical methods, or by a combination of both methods. In the treatment of placental insufficiency syndrome, surgical induction of labour has been shown to be a safe and effective procedure (Baird, 1960; Sinnathuray, 1963 (a), (b), (c), (d)). Hence in most centres, and in most instances, the method of choice in the treatment of prolonged pregnancy is *Surgical Induction of Labour* by amniotomy. In very few cases, where the state of the cervix renders surgical induction to be technically difficult, then an intravenous oxytocin drip may be administered to produce effacement and dilatation of the cervical canal, and thereby allow for subsequent surgical induction by amniotomy.

At the time of induction, a careful note should be made of the volume, consistency and appearance of the liquor amnii. If the liquor is plentiful, escapes freely, and is colourless, then placental insufficiency is unlikely, and delivery can be awaited calmly. On the other hand, if the liquor is scanty, thick and stained with meconium, then there is impending risk to the foetus owing to placental insufficiency, and a special watch should be maintained on the foetal heart until the child is safely delivered. It should be remembered that the uterine contractions of labour themselves impair placental function, and may be the last straw for a foetus already embarrassed by placental insufficiency.

Oxytocin medication is usually reserved for those cases, which have failed to become established in good labour, after a variable interval of 18-24 hours, following upon amniotomy. By adopting such a procedure, not only are the number of cases requiring oxytocin medication considerably reduced (as will be seen from this study), but also one avoids the use of oxytocin on those cases of prolonged pregnancy, whose uterine activity, following upon amniotomy, are destined to be of the normal labour type. If oxytocin therapy is inadvertently used in this latter group of cases, there arises the risk of inducing a state of abnormal uterine activity, which may predispose to foetal death by aggravating the already impaired state of placental function, or by inducing a precipitate delivery; this abnormal state of uterine activity may also predispose to hypertonic dysfunctional type of labour, or even to the remote risk of uterine rupture.

2. Caesarean Section:

In the treatment of prolonged pregnancy syndrome, caesarean section may be indicated for foetal distress in the first stage of labour, for cephalo-pelvic disproportion, and in those cases of major hypertonic uterine dysfunction, where the labour is prolonged and there is failure of progress in the first stage. Often there is co-existence of minor disproportion with the uterine dysfunction. Finally emergency caesarean section may be indicated, following upon amniotomy, when severe intra-amniotic infection sets in, thus making the delay in awaiting vaginal delivery, hazardous to both infant and mother, or when the patient stubbornly fails to go into labour despite surgical induction and intensive oxytocin medication. These last two

groups of cases comprise the "*Failed Induction of Labour*" category, and they should be few indeed, if due care is paid to the strict selection of cases for the induction therapy. Very occasionally the umbilical cord may prolapse, at amniotomy, and this will necessitate an immediate caesarean section to salvage the foetus.

3. Assisted Vaginal Delivery:

Assisted vaginal delivery, be it by forceps, ventouse or breech extraction is beneficial in those cases of prolonged pregnancy syndrome, where there is evidence of placental insufficiency or uterine dysfunction. The latter condition will manifest itself by prolonged labour, especially in the second stage, whereas placental insufficiency may manifest itself by scanty meconium-stained liquor at amniotomy, or by signs of foetal distress in labour. The indication for assisted vaginal delivery becomes more imperative in the presence of the other stigmata of placental insufficiency syndrome such as toxaemia of pregnancy, chronic hypertension and elderly primigravidae.

Results of Study

Over the period of 15 months, from May 1963 to July 1964, the author had conducted a personal study programme to evaluate the efficacy of *Surgical Induction of Labour (ARM)* in the treatment of the placental insufficiency syndrome. The results of the study of 1,000 consecutive inductions have been reviewed by the author elsewhere (Sinnathuray 1964), (c) and (d)). Out of these 1,000 consecutive inductions done for placental insufficiency syndrome, 662 cases were induced for the prolonged pregnancy syndrome. The results that will be reviewed represent the analysis of these 662 consecutive cases of the prolonged pregnancy syndrome.

Scheme of Study

All cases in this study project had their final screening by the author, as to their suitability for surgical induction of labour. The selected patients are admitted to the labour wards of the Kandang Kerbau Hospital. The dates of LMP and EDD, in each of these patients are checked twice at least, once in the ante-natal clinic and again after admission to the labour wards.

After a routine soap and water enema, a vaginal examination is carried out, and if the cervix is favourable, then surgical induction of labour by amniotomy is performed. The details of this procedure have been described elsewhere (Sinnathuray (1964) (d)). In those few cases where the cervix did not seem to be favourable, the foetal maturity was reassessed radiologically, and if this confirms the foetus to be of viable size and maturity, then surgical induction of labour was still proceeded with.

Every case so treated is personally followed up, and a great majority of these cases will have become established in labour within 18 hours of induction. Of those cases that failed to become established in labour within 18 to 24 hours of the surgical induction, an intravenous oxytocin drip was administered to stimulate labour, and thereby attain delivery (Sinnathuray (1964) (d)). Caesarean section was undertaken for the usual foetal or maternal indications in the first stage of labour, and in those cases of "failed induction of labour".

Results

As far as the author could ascertain, in all these 662 cases that were induced for the prolonged pregnancy syndrome, the pregnancy was prolonged beyond the 42nd week of gestation. In 93.1% of these 662 cases, the prolonged pregnancy was uncomplicated, whereas in 6.9% of the cases there was co-existent toxaemia of pregnancy. Just over a quarter (27.3%) of the cases were primigravidae in this study.

A glance at the Induction-Delivery Interval revealed that just over three-quarters (76.1%) of all cases studied were delivered within 24 hours of surgical induction, and a very high proportion of this group did not require any oxytocin stimulation. It is apparent that intravenous oxytocin drip was only utilised in 18.3% of all the cases; in about half of this number (9.7%), the drip was utilised to stimulate the onset of labour pains, whereas in the other half (8.6%) it was utilised to hasten and complete the sluggish labour that had commenced after amniotomy. Rigid selection of cases had contributed to the low oxytocin drip rate in this study. A possible hypothesis for this phenomena has been put forth elsewhere (Sinnathuray (1964), (d)).

TABLE IV

Results of Surgical Induction of Labour in the Treatment of Prolonged Pregnancy Syndrome

1. **Pattern of Study:** Out of 1,000 consecutive inductions done for Placental Insufficiency Syndrome, **662 cases** were induced for the **Prolonged Pregnancy Syndrome,** (pregnancy prolonged beyond the 42nd week of gestation).

Pattern of Cases	**No. of cases**	%	
Prolonged Pregnancy (Uncomplicated)	616	93.1%	
Prolonged Pregnancy with PET	46	6.9%	
2. Parity Distribution:			
Primigravida	181	27.3%	
Multiparae	481	72.7%	
3. Induction-Delivery Interval:			
Under 12 hours	348	52.6%	
Between 12 to 24 hours	156	23.5%	
Between 24 to 36 hours	128	19.4%	
Over 36 hours	30	4.5%	
4. Oxytocin Drip Rate:			
Incidence of i/v oxytocin to induce labour	64	9.7%	
Incidence of i/v oxytocin to hasten labour	57	8.6%	
Gross incidence of i/v oxytocin drip	121	18.3%	
5. Mode of Delivery:			
Spontaneous Vaginal Delivery	589	88.9%	
Assisted Vaginal Delivery	56	8.5%	(1.5%)*
Lower Segment Caesarean Section	17	2.6%	(2.4%)*
	*K.K. Hospital rate		
6. Indications for LSCS (17 cases):			
Foetal Distress	5	0.75%	
Major Uterine Dysfunction	5	0.75%	
Cephalo-Pelvic Disproportion	3	0.45%	
Failed Induction of Labour	4	0.65%	
7. Gross Infection Rate:	24	3.6%	
8. Prematurity Rate:			
Infants under 5½ lbs.	40	6.0%	
Infants under 5 lbs.	8	1.2%	
9. Perinatal Mortality Rate:			
Gross Perinatal Mortality	8	12/1,000	births
Autopsy/Clinical Causes of Death (8 cases):			
Intra-Uterine Asphyxia with Stillbirth	2	3/1,000	
Intra-Uterine Asphyxia with N.N.D.	5	7.5/1,000	
Liquor Amnii Aspiration Pneumonia (LSCS) N.N.D.	1	1.5/1,000	
10. Gross Maternal Mortality:	Nil		

In this study, 88.9% of the cases attained spontaneous vaginal delivery. In 8.5%, assisted vaginal delivery had to be undertaken for the usual indications; and this rate of 8.5% was more than five times as high (8.5%. vs. 1.5%) as that for the whole Hospital during the the year 1963. In this study, the caesarean section rate was only 2.6% (2.4% for the whole Hospital). Reviewing the indications for the caesarean section, it is apparent that in three-quarters (13 out of 17 cases) of all the sections, the operation was done for the complications of prolonged pregnancy syndrome, namely placental dysfunction and foetal distress (5 cases), major uterine dysfunction (5 cases), and disproportion (3 cases), and all these sections would have taken place irrespective of the surgical induction of labour. In fact, in those cases of foetal distress, amniotomy allowed for earlier detection of the placental dysfunction state. In only 0.65% (4 cases) of cases studied was caesarean section undertaken for "*Failed Induction of Labour*". This very low failed induction rate was achieved partly by the rigid personal selection of the cases, and by stringent supervision of such cases until delivery was attained.

The gross infection rate was 3.6%, and the majority of these cases were due to intra-amniotic infection—one of the hazards of the induction procedure. In all these 24 cases, the infection responded to routine antibiotic therapy with penicillin and streptomycin.

The prematurity rate in this study was 6% if the 5½ lbs. birth-weight was taken as the diagnostic index. However, the newborn babies in Malaysia are smaller by about ½ pound than their Western counterparts (Wong, 1964), and hence if the 5 lbs. index is used, the gross prematurity rate in this study was only 1.2% (8 cases). It is quite probable that in some of these 8 cases, the gestational dates may have been wrong, and hence genuine postmaturiy non-existent.

In this study, there were no maternal deaths, and the gross perinatal mortality rate was 12 per 1,000 births (8 cases). Of these 8 cases, 2 were asphyxial stillbirths and 5 were asphyxial neonatal deaths, and in all these 7 cases, severe placental dysfunction secondary to post-maturity syndrome was the underlying cause of death. In the 8th case, faulty technique at caesarean section had resulted in the aspiration of liquor amnii with resultant neonatal death. In 7 out of the 8 perinatal deaths in this study, the labour was induced in the 44th week of gestation, and it is theoretically possible that many of these deaths could have been avoided, if induction had been performed earlier—at the 43rd week of gestation—when placental function would have been better.

Conclusions

It is now well accepted that the prolonged pregnancy syndrome, if untreated, predisposes to states of placental insufficiency, uterine dysfunction and cephalo-pelvic disproportion, which in their turn may contribute to a higher rate of perinatal mortality, and to both foetal and maternal morbidity. Selective induction of labour by amniotomy has come to be accepted as an invaluable therapeutic procedure in the management of this condition.

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