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AMNIOTOMY IN THE TREATMENT OF PLACENTAL INSUFFICIENCY SYNDROME

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PART II — Management of the Placental Insufficiency Syndrome and the Results of Study.

INTRODUCTION:

The crux of the problem in the management of the placental insufficiency syndrome is to be able to secure the safe delivery of a viable sized foetus, which could possess a reasonable chance of survival outside the maternal environment. Due to a state of placental dysfunction, this maternal environment has become inadequate to meet the oxygen and nutrient demands of the growing foetus.

Regime of Management:

TABLE I

Management of Placental Insufficiency Syndrome

1. Bed Rest.
2. Sedation.
3. Specific Therapy of the Underlying Cause.
4. Induction of Labour.
5. Caesarean Section.

(i) Bed Rest:

When placental insufficiency is suspected and the foetus is still premature, it is advisable to keep the patient at rest in bed, so as to improve the state of utero-decidual blood flow, and thereby to enhance placental circulation and perfusion. The chances of survival of an infant born prematurely depend on the duration of gestation rather than on its birth weight, which in any case will not increase, and may even decrease, if the placenta cannot keep pace with the demands of the foetus. For example, a 5-pound mature infant, born to a mother with moderate pre-eclampsia at the 38th week of gestation, has a better chance of survival than a similar sized infant born to a mother with mild pre-eclampsia but more prematurely at the 34th week of gestation.

Decision as to the best time to effect delivery demands a balanced obstetric judgment, weighing the risks of prematurity on the one hand, and the risks of placental insufficiency on the other hand. Taking all these into account, the time comes when delivery seems imperative if a live child is to be secured, but till then, bed rest can be of great help to improve the state of placental circulation.

(ii) Sedation:

Anxious and restless patients will benefit from barbiturates or tranquilisers to alleviate their symptoms, and thereby render bed rest therapy more effective. Sodium Amytal or Luminal are the popular barbiturates prescribed. Sparine (promethazine hydrochloride) is probably the most popular tranquiliser in obstetric practice, at present. Patients with severe abdominal cramps or backache, and who need to be in bed rest, will benefit from some form of analgesic.

(iii) Specific Therapy of the Underlying Cause:

It is stated that, as a rule, it is best to avoid the use of diuretics to treat the oedema in toxaemia of pregnancy, because their use

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may result in loss of body weight in the patient. This weight loss may mimic weight changes due to placental deterioration, and so lead the obstetrician to effect delivery before it is really necessary, (Browne, 1962). In most instances, the oedema co-existing with pre-eclampsia or chronic hypertension, will be found to resolve itself by rest in bed.

The use of hypotensive agents in the treatment of placental insufficiency syndrome is again debatable. Whereas hypotensive drugs have a place in the therapy of patients with moderate or severe chronic hypertensive vascular disease, their use in the treatment of pre-eclamptic toxaemia, or even mild essential hypertension is not universally accepted, and is stated to be unnecessary by most authorities.

Effective prevention and treatment of anaemia in the pregnant patient does go a long way to decrease the deleterious effects of placental insufficiency state on the foetus, whatever may be the predisposing cause of placental failure.

In those patients with chronic pyelonephritis, proper vigilance to prevent reinfection of the renal tract, and effective therapy of the renal infection, when it does occur, can go a long way to decrease the adverse effects on the state of placental function.

Similarly, in those patients with diabetes mellitus, the effective and constant vigilance, and control of the maternal diabetic status can in turn contribute to considerable improvement in the state of placental function, and hence to a higher foetal salvage rate. Good team-work is very essential in the care of the pregnant diabetic patient, who should be under the joint responsibility of both physician and obstetrician throughout her pregnancy. In addition, the paediatrician and in some cases the anaesthetist, should be brought into the picture for the care of the patient during the labour and delivery. Decision as to when to effect delivery needs balanced judgment, if perinatal death from either foetal immaturity or placental insufficiency is to be avoided.

(iv) Induction of Labour:

In those cases of placental insufficiency syndrome where the foetus is of viable size and maturity, termination of pregnancy by the induction of labour will be indicated. In most hospitals, and in most instances, the method of choice 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 cervical canal and theraby allow for subsequent surgical induction by amniotomy.

Surgical Induction of Labour is performed by the artificial rupture of the amniotic membranes. In most instances, the bag of forewaters are punctured with a Kocker's artery forceps, or a special amniotomy forceps. In a very few instances with a high and unengaged presenting part, the hind-waters of the amniotic sac may have to be punctured with a Drew-Smythe catheter, to prevent the prolapse of the umbilical cord.

Browne (1962) states that a careful note should be made of the volume, consistency and appearance of the liquor amnii at the time of induction. 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 contractions of labour themselves impair placental function, and may be the last straw for a foetus already embarrased by placental insufficiency from some other cause, such as toxaemia of pregnancy or post-maturity.

Although the concept of placental insufficiency syndrome and its concomitant hazards have been brought to the attention of the medical profession only in the last decade, pioneer research work on this important facet of obstetric practice had been undertaken by Professor Sir Dugald Baird and his co-workers in Aberdeen, more than 15 years ago, viz, as early as 1949 by Walker and Turnbull. Baird (1960) sugests that there is much to be said for induction of labour to avoid the risks of placental insufficiency syndrome, and for a

more liberal use of Caesarean Section to avoid the undue stress to the baby in labour. In 1953, following the findings of Walker and Turnbull (1953), Professor Sir Dugald Baird implemented the policy at the Aberdeen Maternity Hospital, whereby routine surgical induction of labour was performed on those patients, suspected of having placental insufficiency syndrome. The results of his Study were presented in his Ingleby Memorial Oration, entitled "The Evolution of Modern Obstetrics", (Baird, 1960). He showed that there was an obvious fall in perinatal mortality from toxaemia of pregnancy and postmaturity in the Aberdeen primigravid women of all age groups, during the second 5-year period of 1953 to 1957, as compared to the first 5-year period of 1948-52, and that the fall was most marked in primigravid women aged 30 years or more.

In his oration, Baird (1960) concluded that in order to achieve maximal foetal salvage from placental insufficiency, it was necessary to perform numerous routine inductions of labour, on the basis of epidemiological inferences, and to use Caesarean Section freely in the interests of the baby. He further states that the policy of surgical induction of labour is justified in the absence of any more selective method of prevention of the placental insufficiency state, and by the very results in foetal salvage that have been attained.

Unfortunately surgical induction of labour by amniotomy is not without its attendant risks of intra-amniotic infection with danger to both foetus and mother. Infection is especially liable to occur when labour does not quickly follow the induction. Occasionally a Caesarean Section has to be done because labour fails to start despite every effort.

Surgical induction of labour, therefore has its pros and cons, and it is the responsibility of every obstetrician to select carefully those cases that are to be subjected to this therapy. I have attempted to summarise the salient advantages and distadvantages of surgical induction of labour in the following table. (Table II):

TABLE II

Advantages/Disadvantages of Surgical Induction of Labour.

ADVANTAGES:
1. High Success Rate.
2. Short Induction-Delivery Interval.
3. Allows for Early Detection of Foetal Distress.
4. Physiologically Sound.
DISADVANTAGES:
1. Failure to go into Labour.
2. Intra-Amniotic Infection.
3. Prolapse of the Umbilical Cord.

(v) Caesarean Section:

Caesarean Section, as a therapeutic procedure, in the management of the placental insufficiency syndrome, may be indicated as an elective procedure or as an emergency measure. Elective Caesarean Section may be indicated in those cases, where labour pains can be a risk to the foetus in utero. Such is the case in the elderly primigravida with a bad past obstetric history of prolonged involuntary infertility or habitual abortions. The primigravid pregnant diabetic patient is also best treated by elective Caesarean Section.

Emergency Caesarean Section, in the management of the placental insufficiency syndrome, is indicated either for foetal distress during the first stage of labour, or where labour pains fail to ensue following surgical induction and where the use of the pitocin drip is contraindicated, such is the case in the multigravid diabetic patient. An emergency Caesarean Section may also be indicated, following upon amniotomy, when severe intra-amniotic infection sets in, thus making delay in awaiting vaginal delivery, hazardous to both infant and mother. It is also indicated when the patient stubbornly fails to go into labour, despite surgical induction and intensive intravenous oxytocin medication. The last two groups of cases should be few indeed, if due care is paid to the strict selection of cases, that are to be subjected to this regime of therapy.

Similar views have been expressed by Professor Sir Dugald Baird (1960) who states that occasionally a Caesarean Section has to

be done because labour fails to start despite every effort to effect delivery, following upon surgical induction of labour by amniotomy. In this context, he further concluded that the increased use of surgical induction and Caesarean Section in the treatment of placental insufficiency syndrome has undoubtedly saved many infants, and fulfils the criteria of sound obstetrical practice. Hence this regime of management has come to stay, unless it can be shown that there is an increased risk to the mother from this therapy, or if a more effective alternative way in the treatment of placental insufficiency syndrome is forth-coming.

Regime of Study:

Over the past 15 months, from May 1963 to July 1964, following upon my return from the United Kingdom, I had conducted a personal study project to evaluate the efficacy of *Selective Amniotomy* in the treatment of the placental insufficiency syndrome, in both Government Units of Kandang Kerbau Hospital. This study project had the approval of the Hospital Postgraduate Committee, and in particular the blessings of the Clinical Heads of both the Government Units, Mr. T. H. Lean of the 'A' Unit and Dr. S. M. Goon of the 'B' Unit. I wish to express my sincere gratitude to both of them, and to the numerous doctors and members of the nursing staff in both the Government Units, for having rendered invaluable assistance, without which this project could not have been successfully undertaken.

The essence of success in this study is dependent entirely upon the SELECTIVENESS of those cases that are to be subjected to the surgical induction of labour, by amniotomy. In the absence of facilities in Singapore, to perform the elaborate ancillary laboratory investigations to detect placental insufficiency syndrome, my criteria of *Selection* is based upon the following two features: -

(a) **Clinical Picture of the Case.**

By this I mean, that the clinical history reveals the presence of any one of the causes of placental insufficiency syndrome, either major or minor, as detailed in Table II and III, earlier on (in Part I of this Lecture). The co-existence of other stigmata of placental insufficiency syndrome, such as abnormal weight patterns, blood pressure readings and urinary albumin, may serve as additional parameters which may point to the necessity of induction.

(b) **The State of the Cervix.**

This is my second criteria for selective induction. With very few exceptions, a genuine state of placental insufficiency syndrome is associated with favourable state of the cervix, also referred to as the "ripeness" of the cervix, which in turn renders surgical induction of labour to be technically easy. In my view, a "ripe" cervix is that state of the cervix which fulfils one or more of the following criteria viz.: -

(i) Soft velvety state of the cervix — contrast to the firm fibrous cervix, when unripe.

(ii) A partially dilated cervical canal, accommodating easily 1 finger in primigravida, and 1½ to 2 fingers in a multigravida

(iii) An easily distensible and elastic state of the cervical canal to digital palpation.

I have been of the firm opinion that there is a direct correlation between placental dysfunction and the "ripening" of the cervix, a view which has been fully substantiated by my observations in the present investigations. I have observed that in those cases, where there is a premature degeneration of the placenta before term, such as would occur in moderate and severe pre-eclamptic toxaemia, there is invariably an associated premature "ripening" of the cervix, as described above. In other words, it is not unusual to find the state of the cervix in a patient with moderate pre-eclampsia at the 37th week of gestation to be very similar to the cervix of a genuinely post-mature patient, and this state of "ripeness" of the cervix in these two instances will be a sharp contrast to the "unripe" cervix in a patient with an uneventful normal pregnancy just before term.

It is apparent in my mind, that there seems to be a direct inter-relationship between degeneration of the placenta, and the readiness of the uterus to go into labour. What the

nature of the inter-relationship is, and how this is mediated, be it humoral or enzymatic, is still a puzzle to the research obstetrician. The solution to this problem awaits the enquiring and adventurous research worker.

Based upon the above criteria of case-selection, the patients for this study project are picked up from the ante-natal clinics and the ante-natal wards of both the Government Units of Kandang Kerbau Hospital. All cases in this study project have their final screening by myself, as to their suitability for surgical induction of labour. All selected patients are admitted to the labour wards of the Kandang Kerbau Hospital.

After a routine soap and water enema, a vaginal examination is carried out by me, and if the state of the cervix is favourable, then induction of labour is performed by artificial rupture of the amniotic membranes (fore-waters). At the time of induction, an observation is made of the quality and quantity of liquor amnii. The foetal heart is also auscultated before and after the induction procedure. All patients, after amniotomy, are put on an hourly foetal heart/maternal pulse, and an 8-hourly temperature chart. In addition, they are placed on a 4-hourly blood pressure chart in those cases of pre-eclamptic toxaemia. A vulval pad is applied and the patient is allowed to rest in bed.

Every case so treated is personally followed up, and a great majority of these cases will have become established in labour within 18 to 24 hours of the induction. Once labour has been established, the subsequent management would be no different from cases with spontaneous onset of labour.

Of those cases that failed to become established in labour within 18 to 24 hours of amniotomy, an intravenous oxytocin drip was administered to stimulate the onset of labour. If an oxytocin drip was commenced, it was usually continued until the child had been delivered, and labour completed. In very few instances, where the delivery was not effected with an intravenous oxytocin drip on the first day after amniotomy, the drip had to be administered on the subsequent day again. The oxytocin drip was commenced usually with 2 units of oxytocin per pint of 5% dextrose at 20 drops per minute. The rate and concentration of the oxytocin drip were gradually increased until uterine contractions simulating normal labour pains ensued; thereafter no further increase of the drip rate was made, and the drip was maintained until delivery had been effected. Hence the final concentration of oxytocin that has to be administered would vary with the individual uterine sensitivity to the oxytocin. In a certain percentage of cases, oxytocin drip had to be given to hasten and complete the labour that had ensued after amniotomy per se.

Caesarean Sections were resorted to in those cases, where there were the usual maternal or foetal indications in the first stage of labour; and also in those cases which had failed to become established in labour after the above regime of induction.

RESULTS OF STUDY:

TABLE III

Pattern of Study Project:

CASE – PATTERN	NO. OF CASES	%
Cases considered for amniotomy based upon the Clinical History/Ante-Natal Record	1022	100%
Cases rejected on unfavourable state of cervix	22	2.2%
Cases subjected to amniotomy regime	1000	97.8%
No. of mothers in Study Project	1000	—
No. of infants in Study Project ... ... (Includes 10 twin pregnancies)	1010	—
Gross maternal mortality	0	—
Gross perinatal mortality	13	12.9/1,000 Births

TABLE IV

Indications for Induction (1,000 cases)

MAJOR INDICATIONS			MINOR INDICATIONS		
Postmaturity Syndrome	=	61.6%	Unexplained Past Perinatal Death	=	0%
Postmaturity Syndrome + PET	=	4.6%	Habitual Abortions	=	0.1%
Pre-Eclampsia/Eclampsia	=	30.3%	Involuntary Infertility	=	0.2%
Chronic Hypertension	=	0%	APH/Threatened Abortions	=	1%
Diabetes Mellitus	=	0.6%	Previous L.S.C.S.	=	0.5%
Elderly Primigravida	=	0.6%	Twins	=	0.2%
Chronic Pyelonephritis	=	0.3%			
TOTAL	=	98%	TOTAL	=	2%

Table III shows the pattern of this study project. In all, 1022 cases were considered for surgical induction of labour, on the basis of the clininical history and the ante-natal record. However, 22 cases of postmaturity were rejected, because of the unfavourable state of the cervix. In most of these 22 cases, the postmaturity status was not confirmed, on radiological assessment. In some of them, vaginal reassessment, one to two weeks later, revealed a favourable cervix, and amniotomy was duly performed. In none of these 22 cases, were the babies lost from deferment of the induction.

In this Study, 1000 out of the 1022 cases (97.8%) were subjected to amniotomy, and the results that will be reviewed, represent the study of these 1000 consecutive cases. There were no maternal deaths, and the gross perinatal mortality was 12.9 per 1,000 births.

The indications for the induction of labour in the 1,000 cases have been summarised in Table IV. It is apparent that 98% of the inductions have been undertaken for major causes of placental insufficiency, as outlined in Part I of this Study. The postmaturity syndrome has been the indication for induction in two-thirds (66.2%) of the cases. In one-third (34.9%), toxaemia of pregnancy is the indication. The co-existence of pre-eclamptic toxaemia with postmaturity is usually lethal to the foetus, and this combination was the indication in 4.6% of the cases.

Table V shows that one-third of cases (31.8%) studied were primigravida, and a further quarter (27.2%) of them were grand multiparae.

TABLE V

Parity Distribution (1,000 cases)

PARITY PATTERN	%
Para 0	31.8%
Para 1	13.0%
Para 2	11.4%
Para 3	8.8%
Para 4	7.8%
Para 5 and over	27.2%

TABLE VI

Induction — Delivery Interval

Induction — Delivery Interval (I.D.I.)	%
Under 6 hours	23.8%
Between 6 to 12 hours	30.3%
Between 12 to 24 hours	29.3%
Between 24 to 36 hours	12.2%
Between 36 to 48 hours	2.4%
Bewteen 48 to 60 hours	2.0%

The Induction — Delivery Interval, or the I.D.I., is the interval of time between the artificial rupture of the amniotic membranes and the attainment of delivery. This interval phase will include the time-lag between amniotomy and the onset of labour pains, and also the total duration of the first and second stages of labour.

The above table (Table VI) indicates that more than half (54.1%) of all the cases induced, were delivered within 12 hours of the surgical induction of labour, and in none of these cases, was intravenous oxytocin therapy utilised. Just under one-third (23.3%) of all cases were delivered between 12 to 24 hours of amniotomy, and most of these cases also required no oxytocin therapy. In the remaining 16.6% of cases, the induction — delivery interval was prolonged beyond 24 hours. In almost all these cases, with the exception of those few cases with prolonged hypertonic dysfunctional labour, intravenous oxytocin had to be utilised either to induce the onset of labour pains, or to hasten the labour, that had commenced after amniotomy. In all, 19.1% of cases in this Study, required intravenous oxytocin therapy at some stage of their induction — delivery phase, (Table VII).

Table VII shows that 4 out of the 1,000 cases (0.4%) required oxytocin therapy to effect dilatation of the cervical canal before amniotomy could be performed. In 2 of these 4 cases, moderate pre-eclamptic toxaemia, before the 38th week of gestation was the indication, and in the other 2 instances, the post-maturity syndrome, confirmed by clinical and radiological assessment of the foetal maturity, was the indication. In all 4 cases, amniotomy was performed between 4-8 hours after the commencement of the oxytocin drip, when vaginal reassessment revealed the cervical canal to be dilated sufficiently, for amniotomy to be feasible.

Another 9.8% of the cases required intravenous oxytocin therapy to induce the onset of labour pains after amniotomy. Hence, in this Study, only 10.2% of the 1,000 cases required intravenous oxytocin to induce the onset of labour pains; the remaining 89.8% became established in labour following amniotomy. This observation, which had also been made by other authorities, reaffirms the author's belief that there is a close inter-relationship between placental degeneration and the onset of labour following amniotomy in placental insufficiency. It is probable that placental degeneration provides the chemical stimulus, be it hormonal or enzymatic, which primes the uterus for its readiness to go into labour; and under these circumstances, amniotomy will result in a state of uterine decompression, which may be the final mechanical trigger for labour to commence. This is put forward as a possible hypothesis.

In this Study, a further 8.9% of cases required intravenous oxytocin therapy to hasten and complete the sluggish labour, that had ensued after amniotomy. Thus, in all, 19.1% of the 1,000 cases had required oxytocin therapy to effect delivery. Caesarean Section was performed for "*Failed Amniotomy/Oxytocin Induction*" in 0.8% of the 1,000 cases.

TABLE VII

Pattern of Intravenous Oxytocin Therapy

PATTERN OF CASES STUDIED	NO. OF CASES	%
Incidence of i/v oxytocin drip used to dilate cervix (before ARM)	4	0.4%
Incidence of i/v oxytocin drip to induce labour (after ARM)	98	9.8%
Total incidence of cases requiring oxytocin to induce labour	102	10.2%
Incidence of i/v oxytocin drip used to hasten and complete labour	89	8.9%
Gross incidence of i/v oxytocin drip used to effect delivery	191	19.1%
Incidence of L.S.C.S. done for failed amniotomy/Oxytocin induction	8	0.8%

In placental insufficiency syndrome, the routine use of intravenous oxytocin therapy, along with amniotomy, is unnecessary. That this is so, shown by the fact that 80.9% of all cases subjected to amniotomy alone attained delivery, without oxytocin therapy; and that in 8.9% of cases oxytocin therapy was only required to complete the labour that had commenced after amniotomy. Similar views have been expressed by Baird (1960) and others, who advocate amniotomy in the treatment placental insufficiency syndrome.

In fact, the routine use of intravenous oxytocin therapy, can be lethal to the foetus, in some instances. Firstly, the use of oxytocin therapy, without amniotomy, may not allow for the early detection of foetal distress—namely meconium stained liquor amnii. Secondly, the oxytocin therapy if administered to those cases, whose uterine contractions are destined to be of normal quality, duration and frequency, is likely to result in a state of abnormal hypertonic uterine activity. This may be the last straw to be foetus in utero, already embarrassed by placental dysfunction.

The diagnostic criteria of infection in this Study was taken as a single or multiple rise of temperature above 100.4°F, after the induction and until the patient's discharge from hospital, which varied between 2 to 14 days post-partum. Based upon this criteria, the gross infection rate 3.9%.

An analysis of these 39 cases revealed that in 1.4% (14) of cases, the procedure of amniotomy was not directly responsible. However, in 25 cases there was clinical evidence of intra-amniotic infection, giving an infection rate of 2.5%. In all these patients, the infection responded to the usual routine antibiotic therapy of penicillin and streptomycin. None of the infants, in this Study, were lost from intra-amniotic infection.

TABLE VIII

Infection Pattern following Induction

Gross Infection Rate (39 cases)	3.9%
Aetiology of Infection (T° = 100.4°F or above):	
Intra-amniotic Infection (25 cases)	2.5%
Urinary Infection (10 cases)	1.0%
Uterine Sepsis, following MRP (2 cases)	0.2%
Abdominal Wound Sepsis (1 case)	0.1%
Perineal Wound Sepsis (1 case)	0.1%

TABLE IX

Prematurity Pattern in Study

PATTERN OF PREMATURITY IN STUDY:	No. of INFANTS	%
TOTAL NO. OF INFANTS IN STUDY	1010	100%
Infants with birth-weight below 5½ pounds	82	8.1%
Infants with birth-weight below 5 pounds	29	2.9%
Distribution of the 29 infants under 5 pounds birth-weight:-		
Postmaturity Syndrome	8	0.8%
PET/Eclampsia Syndrome	15	1.5%
Diabetes Mellitus with PET	1	0.1%
Twin Pregnancy	1	0.1%
Ante-Partum Haemorrhage	4	0.4%

It must be emphasized that intra-amniotic infection following induction is one of the hazards of the procedure. Every obstetrician who practises induction of labour by amniotomy, is conscious of this danger to the mother and neonate. Such risks can be reduced to the minimum, if the principle of surgical asepsis is strictly observed during amniotomy, and if the induction delivery interval is kept short.

In 1935, the International Medical Committee of the League of Nations had advocated the acceptance of the definition of a premature infant as one that had a birth-weight of $5\frac{1}{2}$ pounds (2,500 gms.) or less, regardless of the period of gestation. On this basis, the prematurity rate in this Study was 8.1% (82 infants). However, the newborn infants in Malaysia are, on the average, smaller by about $\frac{1}{2}$ pound, in comparison to their Western counterparts. Hence, it is conventional to regard the 5-pound birth-weight, as the upper limit of prematurity in this country, (Wong, 1964). On this basis, the incidence of prematurity was 2.9%.

An analysis of these 29 infants reveals that in 21, the indications for induction were toxaemia of pregnancy (15), diabetes mellitus with PET (1), twins (4), and ante-partum haemorrhage (1); and in all these four groups, a higher prematurity rate is to be expected. In 8 of these 29 cases, the postmaturity syndrome had been the indication. Whilst, the author is prepared to accept the possibility that in some of these cases, the dates may have been misleading, and genuine postmaturity non-existent, this statement cannot be applied en bloc to all the 8 cases. For, it is an accepted fact that postmature infants may fall into the prematurity group, due to their low birth-weight, and that such infants do run a higher risk of perinatal deaths.

Table X reveals that 96.6% of the 1,000 consecutive cases, subjected to induction of labour, came to vaginal delivery; in 89.3% of cases, spontaneous delivery occurred, and in 7.3% of cases, assisted vaginal delivery was undertaken for the usual maternal or foetal indications.

Caesarean Section was performed in 3.4% of cases, but of these only 8 (0.8%) cases required Caesarean Section for "failed induction of labour". In one case (0.1%), destructive vaginal delivery had to be undertaken.

Table XI shows that, in this Study, the gross Caesarean Section rate was 3.4%. In all these 34 cases amniotomy was performed for clear-cut major indications of placental insufficiency syndrome. (15 cases of postmaturity, 17 cases of pre-eclamptic toxaemia, and 2 cases of postmaturity with toxaemia).

The indications for the 34 Caesarean Sections have been tabulated, and it reveals that in over one-third (13 cases) of these cases, foetal distress was the indication. In fact, in all these 13 cases, amniotomy allowed for the early detection of foetal distress, and it is probable that most of these infants would have succumbed to intra-uterine asphyxia from placental insufficiency, if not for their early detection following amniotomy. In fact, the last section of Table XI reveals that in 2 instances, the infants (Cases 1 and 3) still succumbed to intra-uterine asphyxia, despite attempts to salvage them by Caesarean Section. In both these instances, the avoidable factor was the delay in the induction of labour. In Case 1, the induction ideally should have been performed soon after the 42nd week of gesta-

TABLE X

Delivery Pattern

Mode of Delivery (1,000 cases):	%
Spontaneous Vaginal Delivery	89.3%
Assisted Vaginal Delivery (Forceps/Ventouse)	7.3%
Caesarean Section Delivery	3.4%
Caesarean Section for Failed Induction	0.8%
Foetal Craniotomy and Extraction	0.1%

tion, and in Case 3, just before or at term, rather than allow the patient to proceed into the 42nd week of gestation with toxaemia of pregnancy.

A review of the indications for the Caesarean Sections reveals that in 26 out of the 34 cases, Caesarean Section delivery would have been indicated, irrespective of whether amniotomy had been undertaken. In the remaining 8 cases, Caesarean Section had to be done because labour failed to ensue following amniotomy, and subsequent oxytocin therapy. This, *Failed Induction — Caesarean Section Rate of 0.8%* is indeed a very low price to pay for the salvage of several hundreds of babies from the hazards of the placental insufficiency syndrome.

Three of the 13 perinatal deaths in this Study, followed delivery by Caesarean Section, as detailed in the last section of Table XI. In two instances, the placental insufficiency syndrome was directly responsible for the deaths. In the third instance (Case 2), the faulty technique, in the delivery of the infant at Caesarean Section, had resulted in the inhalation of liquor amnii during the delivery, with the resultant perinatal death from aspiration pneumonia.

The next table (Table XII) is self-explanatory. The gross perinatal mortality in in this Study was 12.9 deaths per 1,000 births. The placental insufficiency syndrome, with its sequelae of intra-uterine asphyxia, was directly responsible for 10 out of the 13 (76.9%) perinatal deaths and these are the first 10 cases summarised in the Table. In 7 of these 10 deaths, the postmaturity syndrome was responsible for the deaths, whilst in the 8th and 9th cases, the co-existence of pre-eclamptic toxaemia with prolonged pregnancy proved lethal to the foetus. In all these 9 unbooked cases, lack of ante-natal care was the principal

TABLE XI

Caesarean Section Pattern

Total No. of cases in Amniotomy Study	=	1,000 cases
Total No. of Caesarean Sections performed	=	34
Caesarean Section Rate	=	3.4%

Indications for Induction in the 34 cases:		3.4%
Postmaturity Syndrome	15 cases	1.5%
Postmaturity Syndrome + PET	2 cases	0.2%
Pre-Eclamptic Toxaemia	17 cases	1.7%
Indications for Caesarean Section in the 34 cases:		3.4%
Foetal Distress	13 cases	1.3%
Cephalo-Pelvic Disproportion	5 cases	0.5%
Major Hypertonic Uterine Dysfunction	6 cases	0.6%
Fulminating Toxaemia of Pregnancy	1 case	0.1%
Elderly Primigravida (37 years) Maternal Distress	1 case	0.1%
Failed Induction of Labour	8 cases	0.8%

Perinatal Deaths following Caesarean Section = 3 Infants:

Aetiology	L.S.C.S. Indications	Cause of Death
CASE 1 — Postmaturity (44/52)	— Foetal Distress	Asphyxia
CASE 2 — Postmaturity (44/52)	— Major Ut. Dysfunction	Aspiration Pneumonia
CASE 3 — PET at 42/52	— Foetal Distress	Asphyxia

TABLE XII
PERINATAL MORTALITY PATTERN

Total No. of Infants in Study Project = 1010 (10 Twins)
Total No. of Foetal (Perinatal) Deaths = 13
Gross Perinatal Mortality Rate = 12.9/1,000 births

Case No.	Hospital Reg. No.	Induction-Indication	Mode of Delivery	B.Wt.	S.B./N.N.D.	Cause of Death (Autopsy/Clinical)
1	21597/63	Postmaturity (44/52)	Caesarean Section	5- 4	N.N.D. (1 day)	Asphyxia (autopsy)
2	23503/63	Postmaturity (44/52)	Vacuum Extraction/ Forceps Delivery	6- 8	S. B.	Asphyxia (autopsy)
3	40148/63	Postmaturity (44/52)	Spontaneous	4- 8	S. B.	Asphyxia (autopsy)
4	41637/63	Postmaturity (43/52)	Spontaneous	4-12	N.N.D. (1 day)	Asphyxia (clinical)
5	6056/64	Postmaturity (45/52)	Spontaneous	5- 2	N.N.D. (1 day)	Asphyxia (clinical)
6	8643/64	Postmaturity (45/52)	Spontaneous	5-10	N.N.D. (1 day)	Asphyxia (clinical)
7	15580/64	Postmaturity (46/52) (Diabetes)	Spontaneous	9-12	N.N.D. (1 day)	Asphyxia (autopsy)
8	6207/64	PET (42/52)	Spontaneous	5- 8	N.N.D.(2 days)	Asphyxia (autopsy)
9	14552/64	PET (42/52)	Caesarean Section	7- 0	S. B.	Asphyxia (autopsy)
10	2752/64	Diabetes Mellitus (39/52) (+ PET)	Spontaneous	4- 4	N.N.D. (1 day)	Asphyxia (clinical)
11	17742/64	PET (39/52) (Diabetes)	Shoulder Dystocia/ Extraction	11- 4	S. B.	Birth Trauma (clinical)
12	35278/63	Postmaturity (44/52)	Caesarean Section	6-14	N.N.D. (1 day)	Aspiration Pneumonia (autopsy)
13	1347/64	PET (42/52)	Spontaneous	4- 3	N.N.D.(6 days)	Multiple Foetal Abnormalities

Summary of causes of Perinatal Deaths:

Placental Insufficiency Syndrome (10 cases)	76.9% of Perinatal Deaths
Birth Trauma (Shoulder Dystocia) (1 case)	7.7% of Perinatal Deaths
Caesarean Section Technique (1 case)	7.7% of Perinatal Deaths
Multiple Foetal Abnormalities (1 case)	7.7% of Perinatal Deaths
Perinatal Deaths in Caesarean Sections	3 deaths (Table XI)

avoidable factor. Theoretically most of these 9 deaths could have been avoided, if induction had been performed earlier — before the end of the 43rd week of gestation in uncomplicated prolonged pregnancies, and at or before term in those pregnancies complicated by pre-eclamptic toxaemia.

In Case No. 10, the past obstetric history was atrocious — 10 out of her 13 previous pregnancies had terminated in stillbirths from diabetes mellitus and placental insufficiency. In this present pregnancy, she was kept in the ante-natal ward from the 35th week of gestation for control of her diabetic status. Although her diabetes remained well under control, there was clinical evidence of poor foetal growth in utero. She also developed pre-eclampsia, and induction had to be performed at the 39th week of gestation, despite the small foetal size. The foetus, at birth, weighed only 4 pounds 4 ounces; it showed evidence of severe placental dysfunction, and succumbed to asphyxia 22 hours later. The placental insufficiency in this case, was obviously of prolonged duration and of severe degree.

Birth Trauma from severe shoulder dystocia (Case No. 11) contributed to 7.7% of the perinatal deaths (one case). The mother had gross hydramnios, and unfortunately the foetal macrosomia of 11 pounds 4 ounces was not diagnosed, until after birth. In both cases, No. 7 and No. 11, the maternal diabetic status was not diagnosed in the ante-natal period, and only became apparent when the glucose tolerance test was done post-partum, in view of the foetal macrosomia.

As stated earlier, aspiration pneumonia during Caesarean Section was the cause of one perinatal death (Case No. 12), contributing to 7.7% of the perinatal deaths. The last perinatal death (Case No. 13) was due to gross foetal abnormalities of hydrocephalus and spina bifida, which were incompatible with life.

CONCLUSIONS:

The results of this Study have substantiated the efficacy and safety of amniotomy in the treatment of the placental insufficiency syndrome.

Surgical induction of labour (amniotomy) is a great boon in this era of modern obstetrics. Let us use it wisely, discriminately and justly. Let it not fall into disrepute by becoming the first resort of the impetuous obstetrician, the routine of the ignorant obstetrician, or the tool of social convenience of the impatient obstetrician. Surgical induction of labour has come to stay, but let it be an Elective and Selective procedure.

SUMMARY:

1. The various aspects in the management of the placental insufficiency syndrome have been tabulated and discussed.
2. The place of amniotomy in the treatment of the placental insufficiency syndrome has been evaluated in the author's series of 1,000 consecutive cases.
3. The results of this Study reaffirm the efficacy and safety of selective amniotomy as a therapeutic procedure in this condition.
4. The results indicate that there is a direct correlation between placental degeneration and the readiness of the uterus to go into labour.
5. Selective amniotomy has come to be accepted as an invaluable tool to the present-day obstetrician.

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REFERENCES

Baird, D. (1960): Lancet, **2**, 557.

Baird, D. (1960): Lancet, **2**, 609.

Browne, J. C. M. (1962): Postgrad. Med. J., **38**, 225.

International Medical Committee of the League of Nations: as quoted in "British Obstetric Practice" (1959): 2nd Ed., Heinemann, London, p. 559.

Turnbull, E. P. N. and Baird, D. (1957): Brit. Med., J., **2**, 1021.

Walker, J. and Turnbull, E. P. N. (1953): Lancet, **2**, 312.

Wong, H. B. (1964): Personal Communication.