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The Placental Insufficiency Syndrome - II

by

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PART II
Diagnosis and Management

Diagnosis:

Clinical Aids to Diagnosis

TABLE I

Clinical Aids in the Diagnosis of the Placental Insufficiency Syndrome.

1. Clinical History.
2. Weight Changes in Pregnancy.
3. Alterations in the Uterine Size.
4. Alterations in the Volume of Liquor Amnii.
5. Unexplained Evidence of Foetal Distress in Labour.

1. Clinical History:

Just as in other fields of medical practice, the clinical history serves as an important and invaluable aid to the diagnosis of a possible state of placental insufficiency in any particular pregnancy. From the clinical point of view the presence of any one of the clinical conditions listed in the Table III or IX in part I of this paper should make the obstetrician aware of the possibility of the state of placental insufficiency occurring in that patient's present pregnancy. These clinical conditions, which predispose to the placental insufficiency syndrome, have been described in some detail, and have been further summarised in Tables III and IX. Hence, nothing further need be said on this.

2. Weight Changes in Pregnancy:

Weight changes in normal pregnancy have been exhaustivey studied in the various centres of the United Kingdom and the United States. It is stated that the average weight gain throughout pregnancy in an average sized British woman is of the order of 24 pounds, and that in the latst 4 weeks of pregnancy, weight is gained at the rate of about 1 pound per week (Browne 1962). However, Thomson and Billewicz (1957) from Aberdeen have stated that the mean normal weight-gain for the whole of pregnancy in Scottish women was 27.6 pounds (12.5 kgm). The range of normal weight gain in a British pregnant woman can be taken to vary $\pm$ 4 pounds of the 24 pound mark (20 to 28 pounds). However, in Malaysia, the women and newborn babies in general are of smaller size, and my guess is that the average total weight gain in pregnancy would be about 18 pounds $\pm$ 4 lbs. Hauck (1963) had estimated the average weight gain in the Nigerian pregnant woman to be around 12 pounds.

It is stated that in placental insuffiiciency syndrome, the normal weight gain pattern is not maintained (Browne 1962), and that one or more of the following abnormal patterns may be observed:

a) Overall weight gain in the entire pregnancy may be much less than normal.

b) Weight gain ceases, and the weight becomes static.

c) There may even be an actual loss of weight of 1 or 2 pounds.

When the above weight pattern prevails, and there is no other explanation, such as vomitting, diarrhoea, or diet restriction, it is

stated that foetal death is likely to occur within the next 10 days or so (Browne 1962). Hence, a cessation of weight gain or a sustained weight loss may indicate the necessity for early delivery of the child by whatever means that is appropriate.

3. Alterations in the Uterine Size:

Browne (1962) has stated that the girth of the abdomen at term is on the average 40 inches, and at 36 weeks it is 36 inches, when measured at the umbilicus level. This measurement can be taken as an index of uterine size, although due allowance must be made of course for any obesity. If the abdominal girth, which has been increasing steadily, begins to diminish, this again is an indication that placental insufficiency is impending and that the child should be delivered within a week or so.

4. Alterations in the Volume of Liquor Amnii:

Wrigley (1946) in discussing postmaturity pointed out that the same observer palpating the uterus daily may detect a diminution in the amount of liquor amnii within the uterine cavity. A decrease in the volume of liquor amnii, and a relative increase in the ratio of foetal volume to liquor amnii volume within the uterus is an accepted feature of impending placental insufficiency, in particular post-mat urity. This may indicate the necessity for delivery in those cases where placental insufficiency has been suspected. Further, if at the time of amniotomy, there is observed to be very scanty or no liquor, this feature should be often regarded as ill-omen in those cases, where placental insufficiency has been suspected. Very strict vigilance during labour or, even an urgent Caesarean Section may be called for in such cases to salvage the foetus.

5. Unexplained Evidence of Foetal Distress in Labour:

The presence of unexplained clinical signs of foetal distress in patients in early labour should make the obstetrician suspect the presence of placental insufficiency syndrome, especially so if there is a co-existing clinical history of any one of the factors that have been tabulated in Tables III and IX (Part I) The foetal distress may manifest itself either by meconium stained liquor soon after the rupture of the membranes, and well before strong labour pains ensue, or by the slowing or irregularity of the foetal heart sounds in the first stage of labour. Professor Scott Russell (1962) had stated that under normal circumstances, there should be no alterations to the foetal heart-sounds during the uterine contractions of the first stage of labour, and that if slowing was observed then this pointed to abnormality.

Ancilliary Aids to Diagnosis:

TABLE II

Ancilliary Laboratory Aids in the Diagnosis of the Placental Insufficiency Syndrome

1. Oestriol Studies in Blood/Urine
2. Progesterone/Pregnanediol Studies in Blood/Urine.
3. Liquor Amnii Volume Studies.
4. Vaginal Cytological Studies.
5. Iso—Citric Dehydrogenase Enzyme Studies.

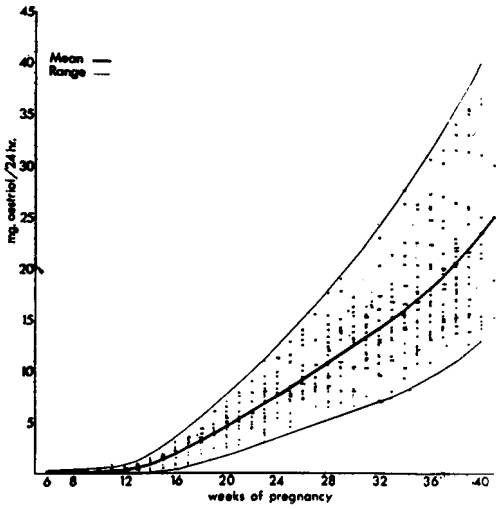
1. Oestriol Studies in Blood/Urine:

Klopper, MacNaughton and Michie (1961), Coyle, Greig and Walker (1962), Coyle and Browne (1963), and Kellar et al (1959), have all at different times shown that placental function can be evaluated by conducting urinary oestriol studies. Similarly, Roy, Harkness and Kerr (1963, a and b) have shown that placental function can also be evaluated by conducting blood oestriol studies. However, all authorities in this sphere of research have emphasized that isloated urinary/blood oestriol studies are of no value in prognosticating placental function, whereas serial studies in any particular patient can be of great help in prognosticating the state of placental function.

Table III, below shows the usual pattern of urinary oestriol excretions in 36 normal pregnancies. This table is taken from the excellent publication of Coyle and Brown (1963)—Fig. 1, J. Obstet. Gynaec. Brit. Commonw., Vol. 70, p. 226.

TABLE III

Normal Urinary Oestriol Levels



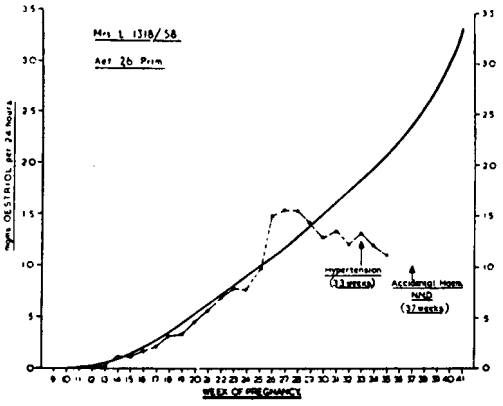
Oestriol excretion during 36 normal pregnancies (Baby weights over 6½ lbs) showing fitted mean, Maximum and minimum values.

(After Coyle and Browne (1963) — J. Obstet. Gynaec. Brit. Common., Vol. 70. P. 226).

TABLE IV, below, shows the abnormal pattern of urinary oestriol excretions, with falling levels in a patient who developed pre-eclampsia at the 33rd week of gestation. She then developed an accidental haemorrhage and delivered herself at the 37th week of a devitalised 5 pounds 9 ounces baby which died within a few hours of birth. It is interesting to note that there was clear cut evidence of falling urinary oestriol levels about 4 to 6 weeks before the clinical state of accidental haemorrhage and foetal death ensued.

TABLE IV

Abnormal Urinary Oestriol Pattern.



Urinary Oestriol excretion levels in a patient who developed pre-eclampsia and accidental heamorrhage.

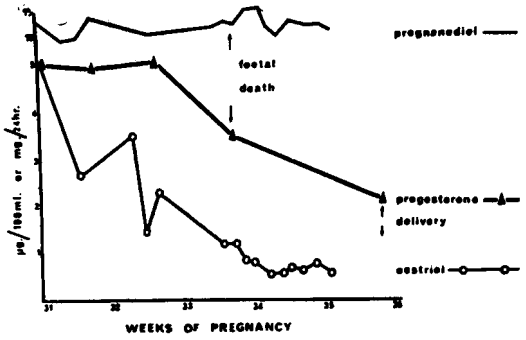
(After Kellar, R. et al. (1959 — J. Obstet. Gynaec. Brit. Emp., Vol. 66 P. 804).

2. Progesterone/Pregnanediol Studies in Blood/Urine:

Klopper (1963), Klopper et al (1955), Coyle et al (1956), Greig et al (1962) and Russell et al (1960) and many other research workers have all shown that the serial assays of blood progesterone or urinary pregnanediol during the pregnancy can be a useful measure in ascertaining the state of placental function. Placental insufficiency syndrome is stated to be characterised by a falling blood progesterone, or urinary pregnanediol levels. Hence the information obtained is very similar to urinary oestriol studies, as is shown in Table V below:

TABLE V

Abnormal Progesterone/Pregnanediol Levels



A case of severe Toxaemia which terminated in foetal death.

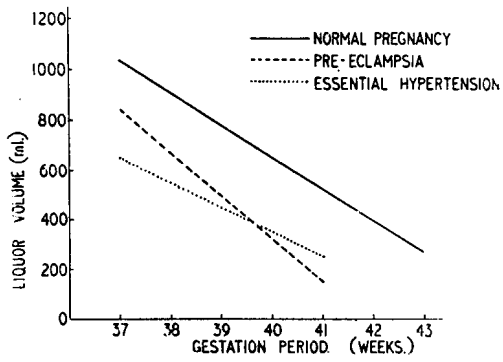
(After Greig., M., Coyle, M.G. et al (1962 — J. Obstet, Gynaec. Brit. Commonw. Vol. 69. P. 772)

However, Russell (1963) claims that pregnanediol assays are far cheaper than oestriol studies, and also quicker to perform. Whilest the above endocrine assay studies are available in most of the Teaching Hospitals in the United Kingdom, it is much regretted that such facili ties are unavailable in Malaysia at the present.

3. Liquor Amnii Volume Studies:

TABLE VI

Alterations in Liquor Amnii Volume.



Computed mean liquor volumes.

(After Elliott, P. and Inman, W.H.W. (1961) — Lancet, Vol. II, P. 85).

Elliot and Inman (1961) have used the volume of liquor amnii as a measure of placental function in late pregnancy. They studied patients with normal pregnancies, as well as cases of pre-eclampsia and chronic hypertension. A dye dilution technique was used to measure the volume of liquor as follows: After obtaining a sample of liquor, a measured quantity (7 to 14 mgm) of "Coomassie Blue" was injected and allowed to mix with the remaining liquor. A sample of the dye-stained liquor was withdrawn 8-25 minutes after the injection, and the volume of liquor amnii calculated from a measurement of the degree of dye-dilution that had occurred.

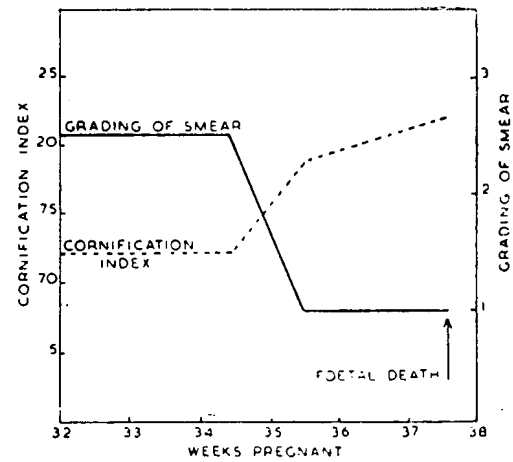
In the normal pregnancies it was found that the volume of liquor reached a peak at 38 weeks, when a mean volume of 1,100 ml. was obtained. Thereafter the volume fell progressively to a level of below 300 ml. at 43 weeks. In pre-eclamptic and hypertensive patients the liquor volume was not only less than normal but fell progressively from levels to around 700 ml. at 37 weeks. These findings are well displayed in the above table (Table VI).

The authors concluded from their study that the clinical findings of maternal loss of weight, and diminution of the girth in late pregnancy in those cases of placental insufficiency syndrome, was consistent with the above findings of low liquor amnii volume. They state that volumes under 300 ml. suggest that the foetus is in grave danger from placental insufficiency state.

4. Vaginal Cytological Studies:

TABLE VII

Vaginal Cytology in Placental Dysfunction.



Vaginal Cytology in a patient with Essential Hypertension — Foetal Death from Intra-Partum Anoxia. Grading of smear diminished and cornification index rose two weeks prior to foetal death.

(After Wood, C., Osmond — Clarke, F. and Murray, M.—J. Obstet. Gynaec. Brit. Commonw., Vol. 68 P. 778).

Wood, Osmond-Clarke and Murray (1961) have gone so far as to state that with the aid of Vaginal endocrine cytological studies, they were able to prognosticate any evidence of impaired placental function in pregnant patients with pre-eclamptic toxaemia or chronic hypertension. Their assessment of the vaginal smear was made from the following 4 features:

i) the amount of cell desquamation.

ii) the size of the cells.

iii) the pattern of the cells.

iv) the cornification index.

Good placental function is characterised by:-

I. intensive cell desquamation.

II. large cells.

III. equal proportion of basal and navicular types of cells.

IV. cornification index of less than 10.

Poor placental function, on the other hand, is characterised by:-

I. poor and scanty cell desquamation.

II. small cells.

III. poor crop of basal and navicular cells.

IV. high cornification index.

These latter features are displayed in the above table (Table XVII), which is representative of placental insufficiency syndrome in a case of Chronic hypertension.

5. Iso-Citric Dehydrogenase Enzyme Levels in Blood:

Dawkins, MacGregor and McLean (1959) suggested that the estimation of the placental enzyme—iso-citric dehydrogenase—in the maternal serum could be used as an index of placental function. Working on the same theme, Morris and Jeacock (1962), also showed that there were abnormal patterns of the enzyme levels in the maternal serum in some cases of toxaemia of pregnancy and accidental haemorrhage.

However, the above tests, like those of Vaginal endocrinal cytology, and liquor amnii volume studies, are highly specialised and still in an experimental state and hence cannot be used for the routine investigation of placental insufficiency in any particular pregnancy.

Summing up this section on the diagnosis of placental insufficiency syndrome, it must be quite apparent to the reader that there is no single faultless test for the detection of placental insufficiency. The diagnosis is essentially an inference, on the part of the obstetrician, after a careful assessment of the overall clinical features; and in some cases, ancilliary investigations, such oestriol/pregnanediol levels in the urine, may be of help to reach a decision.

Regime of Management:

TABLE VIII

Management of Placental Insufficiency Syndrome

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

1. 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 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 if the placenta cannot keep pace with foetal demands. For exa-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 patient with borderline toxaemia but prematurely at 34 weeks gestation. Decision as to the best time to effect delivery demands a nice obstetric judgement, weighing on the one hand the risks of prematurity and on the other hand the risks of placental insufficiency. 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.

2. Sedation:

Barbiturates, tranquilisers and analgesics can be used in certain cases with placental insufficiency to achieve a state of physical and mental rest. Anxious and restless types of patients will certainly benefit from barbiturates or tranquilisers to alleviate their symptoms, and hence render bed rest therapy more effective. Patients with severe abdominal cramps or backache and who need to be in bed rest, will benefit from some form of analgesics. Sodium Amytal or Luminal are the popular barbiturates prescribed. Sparine (promethazine hydrochloride) is probably the most popular tranquilizer in obstetric practice. Pethidine, Omnopon or Morphia could be used as effective analgesics when indicated.

3. 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 symptomatically, because their use may mimic weight changes due to placental deterioration, and so lead the obstetrician to effect delivery

before it is really necessary. Excessive oedema, co-existing with pre-clampsia or chronic hypertension, will be found to resolve itself in most instances by bed rest therapy per se.

The use of hypotensive agents in the treatment of placental insufficiency syndrome is again debatable. Whereas hypotensives 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 in utero, whatever 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, can go a long way to decrease the adverse effects on the placental function.

Similarly, in those patients with diabetes mellitus, the effective and constant vigilance, and control of the maternal diabetic status can contribute considerably to improved state of placental function, and hence to a higher foetal salvage rate. The care of a diabetic pregnant mother should be the joint responsibility of obstetrician, physician, paediatrician and in some cases anaesthetist too.

4. Induction of Labour:

In those cases of placental insufficiency syndrome where the foetus is of viable size and maturity, termination of pregnancy will be called for. In most hospitals and in most cases, the method of choice for the termination of the pregnancy is by *Surgical Induction of Labour*, in the first instance. This is performed by the artificial rupture of the amniotic membranes, and in most cases the fore-waters are punctured with a Kocher's forceps, or a special amniotomy forceps. In a very few cases, with high and unengaged presenting part, the hindwaters may have to be punctured with a Drew-Smythe Catheter, to prevent cord prolapse. At the time of induction, a careful note should be made of the volume, consistency and appearance of the liquor amnii. If the liquor escapes freely and colourless, and there is plenty of it, placental insufficiency is not likely, and delivery can be awaited calmly. On the other hand, if the liquor is scanty, thick and stained with meconium, the foetus is already at risk because of 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 embrassed by placental insufficiency from some other cause such as toxaemia of pregnancy or postmaturity.

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

TABLE IX

Advantages/Disadvantages of Surgical Induction of Labour

ADVANTAGES:
1. High Sucess 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.

5. Caesarean Section:

Caesarean Section, as a therapeutic procedure, in the management of the placental insufficiency syndrome may be performed as an *Elective* procedure or as an Emergency procedure. Elective Caesarean Section may be called in certain very high risk cases, such as an elderly primigravida with a borderline pelvis, or an elderly patient with a bad past ob-

stetric history such as prolonged involuntary infertility or habitual abortions. Primigravid pregnant diabetic patients are also best treated by elective caesarean section. Emergency Caesarean Section, in the management of placental insufficiency syndrome is indicated either for foetal distress at any time in the first stage of labour; or where the patient fails to go into labour following surgical induction but where a "pitocin drip" is contraindicated, *e.g.* multiparous diabetic pregnancies; or where severe intra-uterine infection sets in, thus making delay for a possible vaginal delivery, hazardous to both infant and mother; or where the patient stubbornly fails to go into labour, despite surgical induction and intensive I/V Oxytocin medication by the continuous drip method. The last group of failures should be few and

TABLE X

Results of Surgical Induction of Labour.

1. **No. of Cases Studied**		500 cases
2. **Pattern of Cases:**	**No. of Cases**	%
Postmaturity (Prolonged pregnancy)	304	60.8
Pre-Eclamptic Toxaemia	182	36.4
Miscellaneous (APH, PREVIOUS LSCS etc.)	14	2.8
3. **Parity Distribution:**		
Primigravida	176	35.2
Multiparae	324	64.8
4. **Induction—Delivery Interval:**		
Under 12 hours	331	66.2
Between 12 to 24 hours	122	24.4
Between 24 to 36 hours	35	7.0
Over 36 hours	12	2.4
5. **Mode of Delivery:**		
Vaginal Delivery	447	95.4
Lower Segment Caesarean Section	23	4.6
6. **Indictations for LSCS: (23 cases):**		
Foetal Indications (Distress)	9	1.8
Maternal Indications (CPD, Severe PET, Previous LSCS, Hypertonic Dysfunctional Labour)	7	1.4
Failed Induction of Labour	7	1.4
7. **Oxytocin Drip Rate**	56	11.2
8. **Infection Rate** (Intra-amniotic)	21	4.2

far between by strict selection of cases for the induction therapy.

Results of Personal Study:

Over the past one year, following my return from the United Kingdom in May 1963, I have been conducting a personal study programme to evaluate the efficacy of *Surgical Induction of Labour (ARM)* in the therapy of placental insufficiency syndrome. The above study has the approval of the Kandang Kerbau Hospital Postgraduate Committee, and in particular the approval of the Clinical Heads of the Government Units, Mr. T. H. Lean and Dr. S. M. Goon.

Scheme of Study:

All the cases in this study programme 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. A vaginal examination is carried out, and if the state of the cervix is favourable, then induction of labour is performed by artificial rupture of the amniotic membranes (fore-waters). Every case so treated is personally followed up and a great majority of these cases have been found to be in established labour within 18 hours of induction.

Of those cases, that failed to become established in labour within 24 hours of surgical induction, an intravenous oxytocin drip was administered to stimulate labour, and thereby attain delivery. Caesarean Section was resorted to in those cases where there were the usual maternal or foetal indications in the first stage of labour,

TABLE XI

PERINATAL MORTALITY IN STUDY

Perinatal Deaths in the above series of 500 cases=4 cases.
Perinatal Death Rate=8 per 1,000 cases (0.8%).

Case 1: (NND) **Postmaturity**—44 weeks gestation. Dates certain. Labour induced by ARM, followed 20 hours later by I/V Orasthin Drip. Labour ensued 24 hours after the induction, and after 25 hours in dysfunctional type of labour, there was failure of progress in the late first stage of labour. Delivery by Vacuum Extractor failed, and LSCS performed. Infant, was male, birthweight=5 lbs. 4 ozs., and length=19 inches. Infant was severely asphyxiated and had peeling of skin over both feet. Died after 4 hours. Autopsy revealed *Asphyxial Death*, consistent with the *postmaturity syndrome*.

Case 2: (S.B.) **Postmaturity**—44 weeks gestation. Dates certain. Labour ensued 4 hours after ARM, but the foetal heart suddenly stopped after 18 hours in labour, and about 12 hours later, a stillborn male foetus, weighing 6 lbs. 8 ozs. and length=19 inches, delivered by vacuum extractor. Autopsy revealed *Asphyxial Death* consistent with *postmaturity syndrome*.

Case 3: (NND) **Postmaturity**—44 weeks gestation. Dates certain. Labour ensued within 10 hours of ARM, but the labour was hypertonic dysfunctional type and prolonged to over 45 hours, when foetal distress set in. LSCS performed. Infant was male, birthweight=6 lbs. 14 ozs. and length=20 inches. Infant was severely asphyxiated and lived only for 5.1/3 hours. Autopsy revealed *Aspiration Pneumonia and Asphyxia*, consistent with *postmaturity syndrome*.

Case 4: (NND) **Mild PET/Hydramnios at 42 weeks gestation.** About 4 pints of liquor drained at ARM. Labour ensued within an hour of induction and after 6 hours labour, she had a spontaneous vaginal delivery of a living female infant, birthweight=4 lbs. 3 ozs. and length=15 inches. Infant had multiple congenital malformations of *Hydrocephalus, Spina Bifida and Talipes*, and succumbed from these malformations on the 6th day of life.

and in those cases, which had failed to go into labour after the above regime of induction.

The results of my study have been tabulated in Tables X and XI Five hundred consecutive induction cases were reviewed in all, and of these, over 60% were done for prolonged pregnancy, and over 35% for toxaemia of pregnancy. Minor causes contribute to less than 3% of all the inductions. There is a relatively high proportion primigravida inductions (35%), and majority of these were for toxaemia of pregnancy.

A glance at the Induction-Delivery Interval revealed that just over 90% of all cases studied were delivered within 24 hours of surgical induction, and a very high proportion of this (over 80%) did not require any oxytocin stimulation.

It is apparent that only 4.6% (23 cases) of this study required caesarean section. Reviewing the indications for the Caesarean section, it is apparent that in 3.2% (16 of the 23) of the cases, the Caesarean Section was done for other than "Failed Induction of Labour", and these sections would most likely have taken place, irrespective of the Surgical induction. In only 1.4% (7 cases) of cases studied was Caesarean Section done for "Failed Induction of Labour". This low "failed induction rate" is achieved partly by the rigid personal selection of the cases, and by personal supervision of such cases until delivery is attained.

It is apparent that intravenous oxytocin drip was only utilised in 11.2% of the cases studied, and it represents those cases that failed to become established in good labour, after about 18 to 24 hours of amniotomy lapse. Here again, rigid selection has contributed to a low oxytocin drip rate.

Table XI summarises the case-histories of the 4 perinatal deaths, that occurred in the above series. Case 4, with lethal congenital malformations is incompatible with life. The other three cases, however, are avoidable deaths, theoretically at least. All three were unbooked cases, presenting at the clinic for the first time at 44th week of gestation. Their asphyxial deaths from postmaturity, might have been avoided if induction had been performed earlier—at the 43rd week of gestation.

Conclusion:

"*Surgical Induction of Labour*" *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, for the present time, but let it be an elective and selective procedure.

Acknowledgements

I wish to make official acknowledgments to the Editors of the undermentioned Journals, and the authors of their respective publications for the use of the following Tables from their publications, in this paper:-

TABLE	JOURNAL	AUTHORS
Table III	J. Obstet. Gynae. Brit. Commonwealth, (1963). Vol. **70**	Coyle, M.G. & Browne, J.B.
Table IV	J. Obstet. Gynae. Brit. Emp., (1959). Vol. 66.	Kellar, R.et al.
Table V	J. Obstet. Gynae. Brit. Commonw. (1962), Vol. **69**	Greig., M., Coyle, M.G. et al.
Table VI	Lancet (1961), Vol. II	Elliott, P. and Inman W.H.W.
Table VII	J. Obstet. Gynae. Brit. Commonw., (1961), Vol. **68.**	Wood, C., Osmond—Clarke, E. and Murray, M.

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