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GALLOWAY MEMORIAL LECTURE — 1964 *

AMNIOTOMY IN THE TREATMENT OF PLACENTAL INSUFFICIENCY SYNDROME

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PART I — Concept of the Placental Insufficiency Syndrome and Review of Literature.

The Master, Ladies and Gentlemen,

Sir David Galloway can be said to be the pioneer of western medicine in Malaysia, and was one of the founders of this medical school, from which I am privileged to have graduated just over 50 years later. There is yet one other factor in common between Sir David Galloway and the Galloway Memorial Lecturer for 1964. Sir David was a Scotsman by birth, and had his medical education in Scotland; and I too was privileged to have undertaken postgraduate studies and research in obstetrics and gynaecology in Scotland, under the wise guidance of Professor Sir Dugald Baird, Regius Professor of Midwifery, Aberdeen University. Sir Dugald is also a Scotsman by birth, who has dedicated his life to research in obstetrics and gynaecology. Sir Dugald is probably the leading personality in the field of obstetrics and gynaecology in the Commonwealth, if not in the world, at present. Most of what I have to say, this evening, is the reflection of his impact on modern obstetrics.

I am deeply conscious of this honour that has been bestowed upon me by the Singapore Academy of Medicine in inviting me to deliver the Galloway Memorial Lecture for 1964.

Introduction

The last few decades have seen the steady decline of perinatal mortality all over the world, in general. With the reduction of the previous common causes such as prematurity, traumatic births and infections, the placental insufficiency syndrome, with its sequelae of intra-uterine asphyxia, has come to the forefront as the leading cause of perinatal mortality. In the United Kingdom, a national perinatal mortality survey was carried out under the auspices of the National Birthday Trust Fund in 1958. In this survey, during the specified period of one week, 3rd to 9th March 1958, complete clinical data were obtained from over 98 per cent of all the births, and autopsy studies were performed in about 88 per cent of all the perinatal deaths. Claireaux (1961, 1963) reviewed the data from this National Survey, and showed that the placental insufficiency syndrome was the leading cause of perinatal mortality, being responsible, either directly or indirectly, for over 30 per cent of all the perinatal deaths.

Definition

Although our attention has been drawn to this syndrome in current publications, there has been no attempt made to put forth a clear cut concept. The following is my personal concept of the "placental insufficiency syndrome."

"The Placental Insufficiency Syndrome is a state of dysfunction of the placenta in which there is poor overall growth or premature degeneration of the placenta, with resultant reduction in the placental reserve state, to such an extent as to be a danger to the foetus. This danger can manifest itself by retarded intrauterine foetal growth, foetal cachexia, or by a state of intrapartum foetal anoxia, any

* Galloway Memorial Lecture (1964) — Part I, which was delivered to the Academy of Medicine, Singapore, on the 27th August, 1964.

one of which may predispose to perinatal death from asphyxia, intracranial haemorrhage or infection."

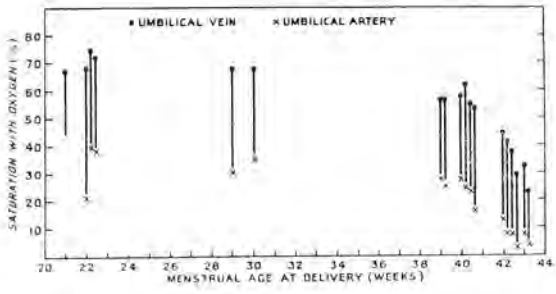
Historical and Experimental Review:

As early as 1934, Sir Joseph Barcroft and his colleagues, working on goat and sheep foetus, had shown that the foetal haemoglobin oxygen saturation deteriorated as pregnancy proceeded towards and beyond, the normal term. Again in 1945, Barcroft and Young showed that in the rabbit foetus, there is evidence of deficient oxygen saturation with special reference to postmaturity. McKiddie (1949) 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, and 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 of gestation. They concluded that the excess of foetal death in prolonged pregnancy could be due to a falling oxygen supply.

Fig. 1

Shows the oxygen saturation of blood in the foetal umbilical vein and artery at different periods of gestation.



(After Walker & Turnbull, (1953) — Lancet Vol. 2, 312).

At about the same time as Walker and Turnbull, Browne and Veal were conducting Uterine (chorio-decidual) blood flow studies in both normal human pregnancy and in cases of pre-eclampsia and chronic hypertension, with the aid of radio-active Na^{24}isotope. They (1953) stated that the average uterine blood flow in normal pregnancy at the 38th week of gestation was about 600ml. per minute, but that in toxaemia and chronic hypertension, the blood flow was reduced to as low as 200ml. per minute before foetal loss occurred. They concluded, therefore, that under normal conditions, the placenta has a considerable functional reserve status.

Pathology of Placental Failure:

TABLE I

Classification of Placental Failure

MATERNAL CAUSES:
1. Defects in Utero-Decidual Circulation.
2. Defects in Chorio-Decidual Circulation.
FOETAL CAUSES:
1. Gross Placental Atrophy or Infarction.
2. Trophoblastic Atrophy in the Chorionic Villi.

The pathology of placental failure can be either of maternal or foetal origin. From the maternal aspect, the failure can be due to defects in the utero-decidual, or in the chorio-decidual circulation, such as occurs in the elderly primigravida, chronic hypertension and uterine scars. From the foetal aspect, the placental failure could be due either to gross placental infarction or to trophoblastic atrophy in the chorionic villi, such as occurs in prolonged pregnancy (postmaturity), toxaemia of pregnancy, and diabetes mellitus. However, there is a close inter-relationship between the maternal and foetal aspects of placental failure.

Scott Russell (1963) has pointed out that attempts to correlate placental histology and the clinical picture have proved very disappointing; and that a foetus can die despite reasonably normal histology in the placenta,

or live despite serious faults. He pointed out that the outstanding contribution of morbid histology to placental function has been the clear demonstration of the changing placental structure as pregnancy advances, and the consequent inference that the placental function also changes.

Aetiology:

Clinically, the aetiology of the placental insufficiency syndrome can be broadly divided into two groups — the first group represents those conditions where there is little doubt as to the occurrence of placental insufficiency, and where the state of placental insufficiency is usually of a major degree. The second group represents those conditions where the extent of placental insufficiency is variable, and often of a minor degree.

TABLE II

Aetiology of Major Degrees of Placental Insufficiency Syndrome.

1. Postmaturity Syndrome (Prolonged Pregnancy).
2. Pre-Eclampsia/Eclampsia Syndrome.
3. Chronic Hypertensive Vascular Disease.
4. Pyelonephritis.
5. Diabetes Mellitus.
6. Elderly Primigravida.

(a) **Major Aetiological Factors:**

(i) **Postmaturity Syndrome:**

There is little doubt in the mind of the practical obstetrician that the postmaturity syndrome or prolonged pregnancy does exist, and is still a major contributor to perinatal mortality and morbidity. Barcroft and his colleagues (1934, 1945) have displayed this problem in the goat, sheep and rabbit foetus. McKiddie (1949), Walker and his colleagues (1953, 1958), and Browne (1961, 1963) have in a masterly way surveyed the extent of this problem in the human pregnancy.

Walker (1958) had categorically enunciated that the three cardinal hazards of the postmaturity syndrome are:—

(1) a rise in the perinatal mortality,

(2) an increasing incidence of foetal distress, and

(3) an increasing incidence of difficult and operative delivery.

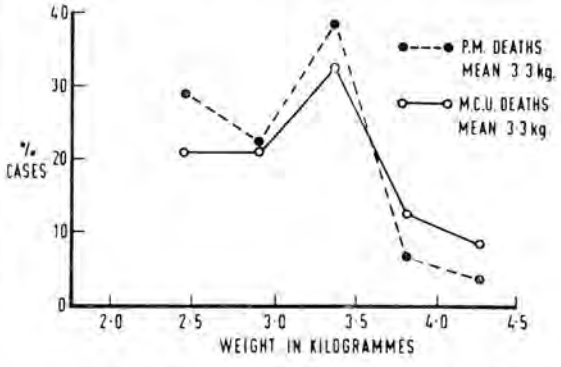
He had further stressed that it was extremely important to realize that each of these three factors was an integral part of the syndrome, since they were closely interwoven clinically, and in many aspects interdependent.

Browne (1963) in his Joseph-Price Oration, four years later, came to similar conclusions, and his results are summarised in the following graphs:

Fig. 2

Relationship of Birth-Weight to Foetal Maturity

The figure shows the average weights of perinatal deaths in postmature and mature full-term pregnancies.

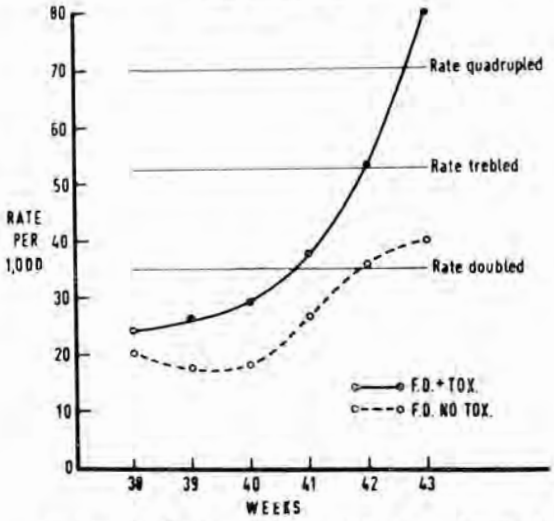


(After J. C. M. Browne (1963) — Amer. J. Obstet. & Gynae., **85**, 573. With the courtesy of the publishers — The C. V. Mosby Company).

The above figure shows that there is little difference in the birth-weights of perinatal deaths in mature and postmature pregnancies. Hence, it is fallacious to infer that because the dead foetus is of average size, it could not be a postmature death. In fact, a postmature foetal death may, in some instances, be of low birth-weight.

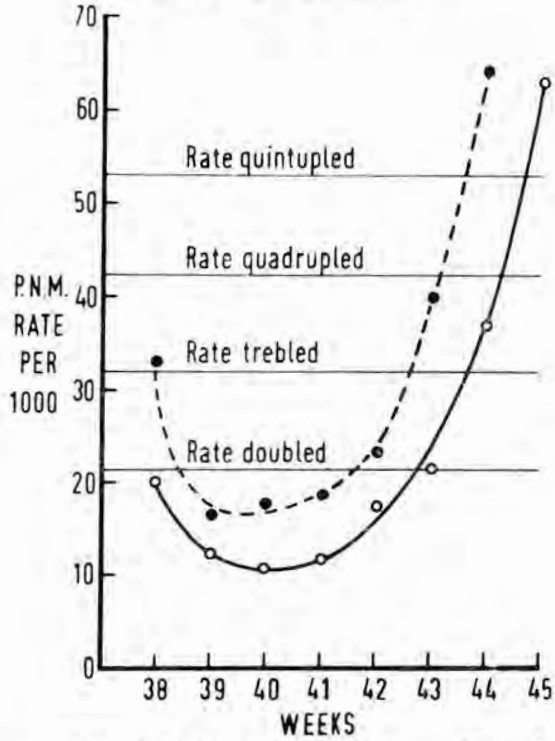
Figure 3. Shows that the incidence of foetal distress rises very sharply in the postmature pregnancy, and this is especially marked, if there is co-existing toxaemia of pregnancy.

Fig. 3
Relationship of Foetal Distress to Foetal Maturity and Toxaemia.



(After J. C. M. Browne (1963) — Amer. J. Obstet. & Gynae., **85**, 573. With the courtesy of the publishers — The C. V. Mosby Company).

Fig. 4
Relationship of Perinatal Mortality to Foetal Maturity and Toxaemia.



(After J. C. M. Browne (1963) — Amer. J. Obstet. & Gynae., **85**, 573. With the courtesy of the publishers — The C. V. Mosby Company).

Figure 4. Shows that there is a steep increase in perinatal mortality in pregnancies prolonged beyond the 42nd week of gestation, and this feature is more marked when there is co-existing toxaemia of pregnancy.

(ii) **Pre-Eclampsia/Eclampsia Syndrome:**

The above combination represents the true toxaemia of pregnancy, and there is little doubt that toxaemia of pregnancy predisposes to the placental insufficiency syndrome. Gross placental degenerative changes is a constant feature of moderate and severe toxaemia of pregnancy. Figures 3 and 4 from Browne's (1963) paper clearly indicate that toxaemia of pregnancy predisposes to a higher incidence of foetal distress and perinatal mortality, and this is more so, when there is co-existent postmaturity.

(iii) **Chronic Hypertensive Vascular Disease:**

Chronic hypertension, in the pregnant mother, predisposes to the severe forms of placental insufficiency syndrome, either directly by precipitating placental infarctions, or indirectly by predisposing to toxaemia of pregnancy. Townsend (1963), and Bourne and Williams (1962) have drawn our attention to this problem.

(iv) **Pyelonephritis:**

Chronic pyelonephritis is often associated with secondary hypertension, which can predispose to placental insufficiency. The high perinatal mortality rates that co-exist with chronic pyelonephritis is only partly due to placental insufficiency.

(v) **Diabetes Mellitus:**

There is some degree of controversy as to whether placental insufficiency is a feature of diabetic pregnancies. Scott Russell (1963), Robertson et al (1963), and Eddie (1963) have found that, in the pregnant diabetic patient, the estimation of urinary oestriol and pregnanediol excretion is seldom of any guide in the detection or management of placental insufficiency state. Russell (1963) categorically stated that in the diabetic pregnancy, the

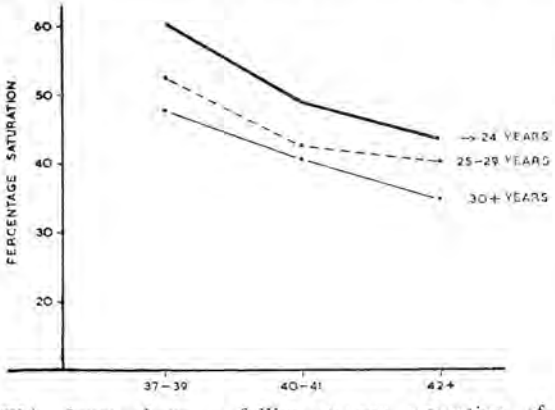
placenta, although often large, was inadequate to meet foetal requirements of oxygen and nutrition. He further stated that presumably the risk to the foetus in maternal diabetes was metabolic, and not hormonal, and was hence not reflected in altered hormonal excretions.

(vi) Elderly Primigravida:

Turnbull and Baird (1957) conducted controlled scientific studies in the human pregnancy and found that the average oxygen saturation of foetal haemoglobin became less as maternal age and the length of gestation increased, and was sometimes dangerously low, especially in primiparae aged 30 or more, delivered after the 41st week of gestation. They then postulated that the relatively high rate of perinatal mortality in the elderly primigravidae — often clinically unexplained and accompanied by postmortem evidence of foetal asphyxia — is due in part to inadequate foetal oxygenation after term.

Fig. 5

Relationship of oxygen saturation in Foetal Haemoglobin to maternal age and duration of gestation.



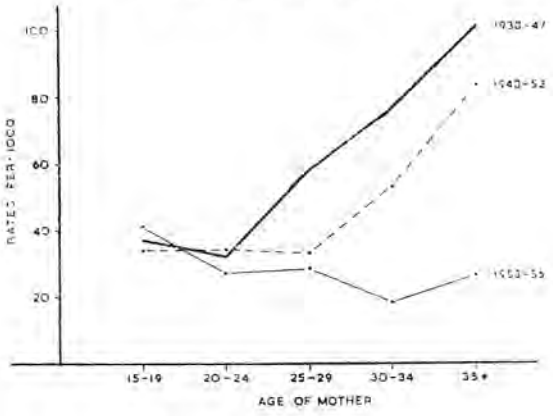
This figure shows a falling oxygen saturation of Foetal Haemoglobin with increasing age of patient and maturity of pregnancy.

(After Turnbull and Baird (1957) — Brit. Med. J., **2**, 1021).

In the light of these findings, Baird, in 1953, implemented a policy at the Aberdeen Maternity Hospital, whereby labour has been induced routinely by artificial rupture of the membranes in primigravidae aged 25 or more and who are undelivered before the end of the 41st week of gestation.

Fig. 6

Falling Perinatal Mortality Rates with Policy of Surgical Induction.



(After Turnbull and Baird (1957) — Brit. Med. J., **2**, 1021).

The above figure shows that as a result of the above policy, there was a steep fall in the perinatal mortality rates in the elderly primigravidae, during the years 1953-6, to such an extent as to eliminate the excess of perinatal deaths.

TABLE III

Aetiology of Minor Degrees of Placental Insufficiency Syndrome

1. Unexplained Past Perinatal Death.
2. Habitual Abortion (Unexplained).
3. Prolonged Involuntary Infertility.
4. Threatened Abortion/Antepartum Haemorrhage.
5. Multiple Pregnancy.
6. Previous Uterine Scar { L.S.C.S. / C.C.S. / Myomectomy }

(b) Minor Aetiological Factors:

(i) Unexplained Past Perinatal Death:

It is an accepted belief that certain types of women are poor obstetric performers, and that patients who have had a past perinatal death of unexplained aetiology, are more susceptible to a recurrence of this status. In such instances it is probable that placental insufficiency,

of unexplained basis, may be the underlying factor.

(ii) **Unexplained Habitual Abortions:**

Those authorities, who are advocates of the placental insufficiency syndrome, believe that patients with a history of unexplained habitual abortions, have a greater tendency to develop a state of placental insufficiency in their subsequent pregnancies. It is possible that minor aberrations in the genes (sperm or ovum), which could lead to such cases of unexplained habitual abortions, could also be responsible for the production of defects in the state of placental structure and function. However, placental insufficiency status does not co-exist with every case of habitual abortion.

(iii) **Prolonged Involuntary Infertility:**

Those women, who have had prolonged involuntary infertility, are said to have a higher tendency to develop the placental insufficiency syndrome. Statistically there is evidence that such patients have a higher incidence of unexplained foetal distress and asphyxial perinatal deaths.

(iv) **Threatened Abortions/Antepartum Haemorrhage:**

It is a well accepted fact that patients with episodes of placental haemorrhage in any period of their pregnancy do tend to sustain varying degrees of placental damage, even though the pregnancy proceeds to term. But whether any such particular case does sustain that degree of placental damage, so as to produce a state of placental insufficiency, is often difficult to ascertain.

(v) **Multiple Pregnancy:**

Most authorities, subscribing to the concept of placental insufficiency, do accept the fact that patients, with multiple pregnancy, tend to run a higher risk of developing placental insufficiency. In fact, McClure Browne (1962) categorically states that the optimal duration for twin pregnancy is 38 weeks and not 40 weeks. He even goes so far as to advocate termination of uncomplicated twin pregnancies, soon after the 38th week of gestation, based upon his strong suspicions, which he has yet not been able to substantiate. However, most other authorities are prepared to await the 40th week of gestation, before considering the termination of uncomplicated twin pregnancies.

(vi) **Previous Uterine Scar:**

The ardent advocates of the placental insufficiency syndrome postulate that patients who have had a scar in the uterus, be it lower segment caesarean section, classical caesarean section, or myomectomy, do run a higher risk of developing the placental insufficiency syndrome, in their subsequent pregnancies. There is some reason to believe that this may be so in those cases where the placenta becomes implanted over the site of uterine scar, and hence become impaired in blood supply. But each case should be assessed on its individual merit, as to the state of placental function.

DIAGNOSIS:

Clinical Aids to Diagnosis:

TABLE IV

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.

(i) **Clinical History:**

Just as in other fields of medical practice, the clinical history serves as an 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 Tables II and

III should make the obstetrician aware of the possibility of the state of placental insufficiency. These clinical conditions have been described in some detail, and hence nothing further need be said.

(ii) **Weight Changes in Pregnancy:**

Weight changes in normal pregnancy have been exhaustively studied in the various centres of the United Kingdom. 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 last 4 weeks of pregnancy, weight is gained at the rate of about 1 pound per week (Browne 1962). 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.). However, in Malaysia, the women and newborn babies tend to be smaller and my guess is that the probable average total weight-gain would be about 18 pounds ± 4 pounds. Hauck (1963) had estimated the average total weight-gain in the Nigerian pregnant woman to be around 12 pounds.

It is stated that in those cases of placental insufficiency 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 near term.

When the above weight pattern prevails, and there is no other explanation, such as vomiting, 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 weight-loss may indicate the necessity for early delivery.

(iii) **Alterations in the Uterine Size:**

Browne (1962) 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. This measurement can be taken as an index of uterine size, although due allowance must be made 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 soon.

(iv) **Alterations in the Volume of Liquor Amnii:**

Wrigley (1945) 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, and may indicate the necessity for delivery. Further, if at amniotomy, there is observed to be very scanty or no liquor, it is an ill-omen; and very strict vigilance during labour, or even an urgent caesarean section may be called for, to salvage the foetus.

(v) **Unexplained Evidence of Foetal Distress in Labour:**

The presence of unexplained signs of foetal distress in early labour should make the obstetrician suspect the presence of placental insufficiency syndrome, especially so if there is a co-existing clinical history as outlined earlier. The foetal distress may manifest itself either by meconium stained liquor before labour pains ensue, or by the slowing or irregularity of the foetal heart sounds in the first stage of labour. 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 V

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.

(i) Oestriol Studies in Blood/Urine:

Klopper et al (1961), Coyle et al (1962), Coyle and Brown (1963), Banerjea (1962), and Kellar et al (1959) have all shown that placental function can be evaluated by conducting urinary oestriol studies. Similarly, Roy et al (1963) 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 isolated urinary/blood oestriol studies are of no value in prognosticating placental function, whereas serial studies in any particular patient can be useful to evaluate the state of placental function.

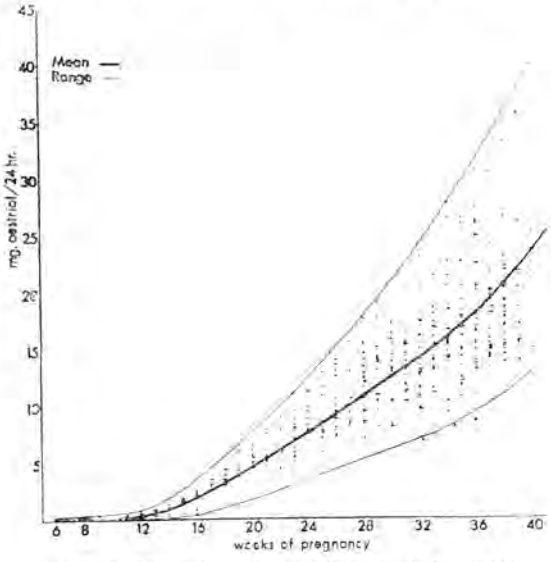
Figure 7 shows the usual pattern of urinary oestriol excretions in 36 normal pregnancies. This table is taken from the excellent publication of Coyle and Brown (1963).

Figure 8. 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 9¼ 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 accidental haemorrhage, and foetal death ensued.

Fig. 7

Normal Urinary Oestriol Levels.

This figure shows a steady increase in Urinary Oestriol levels with the advance of pregnancy.

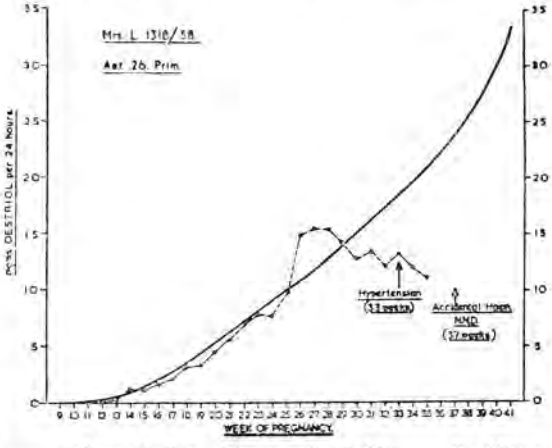


(After Coyle, M. G. and Brown, J. B. (1963) — J. Obstet. & Gynae. Brit. Cwlth., **70**, 225).

Fig. 8

Abnormal Urinary Oestriol Pattern

This figure shows a falling urinary oestriol pattern with Placental Insufficiency State.



(After Kellar, R. et al (1959) — J. Obstet. Gynae. Brit. Emp., **66**, 804).

(ii) Progesterone/Pregnanediol Studies in Blood/Urine:

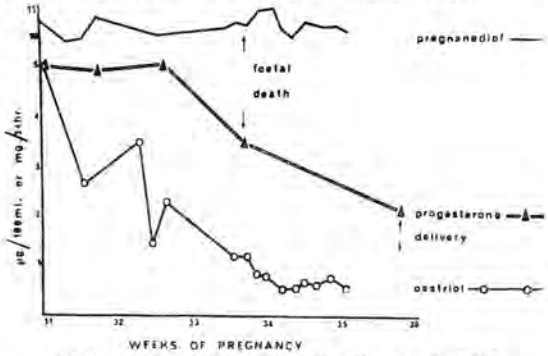
Klopper (1963), Klopper et al (1955), Coyle et al (1956), Greig et al (1962) and Russell et al (1960) have shown that the

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 Figure 9.

Fig. 9

Abnormal Progesterone/Pregnanediol Levels.

This figure shows a falling blood Progesterone/ Urinary Pregnanediol levels in a case of Foetal Death associated with Placental Insufficiency.



(After Greig, M., Coyle, M. G., et al (1962) — J. Obstet. & Gynae. Brit., Cwlth., **69,** 772).

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

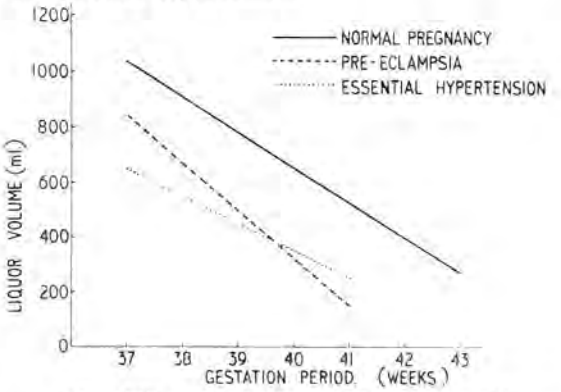
(iii) Liquor Amnii Volume Studies:

Elliott 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 amnii. In the normal pregnancies it was found that the volume of liquor reached a peak at 38 weeks, when a mean volume of 1,100ml. was obtained. Thereafter, the volume fell progressively to a level of below 300ml. at 43 weeks. In pre-eclamptic and hypertensive patients the liquor volume was not only less than normal but fell progressively from levels around 700ml. at 37 weeks. These findings are well displayed in the above figure (Figure 10). 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, were consistent with the above findings of low liquor amnii volumes. They state that volumes under 300ml. suggest that the foetus is in grave danger from placental insufficiency state.

Fig. 10

Alterations in Liquor Amnii Volume.

Patterns of Liquor Amnii Volume at various gestation period in normal pregnancy, Pre-Eclampsia, and Essential Hypertension.



(After Elliott, P. and Inman, W. H. W. (1961) — Lancet Vol. **2,** 835).

(iv) Vaginal Cytological Studies:

Wood et al (1961) have stated that, with the aid of vaginal endocrine cytology, they were able to prognosticate impaired placental function in patients with pre-eclamptic toxaemia or chronic hypertension. Their assessment of the vaginal smear was made from the following 4 features:—

(1) the amount of cell desquamation;
(2) the size of the cells;
(3) the pattern of the cells, and
(4) the cornification index.

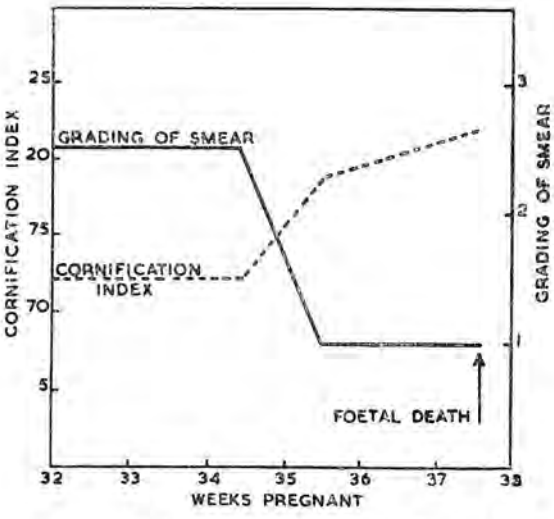
Good placental function is characterised by —

(1) intensive cell desquamation;
(2) large cells;
(3) equal proportion of basal and navicular types of cells, and
(4) cornification index of less than 10.

Fig. 11

Vaginal Cytology in Placental Dysfunction.

This figure shows an abnormal pattern of vaginal cytology in a case of Placental Dysfunction associated with Chronic Hypertension.



(After Wood, C., Osmond Clarke E. and Murray, M. (1961) — J. Obstet. & Gynae. Brit. Cwlth., **68**, 778).

Poor placental function, on the other hand, is characterised by —

(1) poor and scanty cell desquamation;

(2) small cells;

(3) poor crop of basal and navicular cells, and

(4) high cornification index.

These latter features are displayed in the above figure which is representative of placental insufficiency syndrome in a case of chronic hypertension.

(v) Iso-Citric Dehdrogenase Enzyme Levels in Blood:

Dawkins, MacGregor and MacLean (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. Morris and Jeacock (1962) also found that there were abnormal patterns of the enzyme levels in toxaemia of pregnancy and accidental haemorrhage. However, this test is still in an experimental state.

Summing up this section on the diagnostic aspects of the placental insufficiency syndrome, it is apparent that there is no single test for the detection of placental insufficiency. The diagnosis is essentially an inference, after a careful assessment of the overall clinical picture; and in some cases, ancilliary investigations, such as oestriol/pregnanediol levels in the urine or blood, may be helpful to reach a decision.

Summary:

1. A concept of the "Placental Insufficiency Syndrome" has been put forth.

2. The pathology of placental failure has been tabulated.

3. The aetiology of the placental insufficiency syndrome has been discussed, as viewed from the obstetrician's angle.

4. Aids to the diagnosis of this syndrome, both clinical and ancilliary, have been reviewed.

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(i) Figure 1 — Lancet, (1953), Vol. II — Walker, J. & Turnbull, E.P.N.

(ii) Figure 2 — Amer.J.Obstet.Gynae., (1963), Vol. 85 — Browne, J.C.M.

(iii) Figure 3 — Amer.J.Obstet.Gynae., (1963), Vol. 85 — Browne, J.C.M.

(iv) Figure 4 — Amer.J.Obstet.Gynae., (1963), Vol. 85 — Browne, J.C.M.

(v) Figure 5 — Brit.Med.J. (1957), Vol. II — Turnbull, E.P.N. and Baird, D.

(vi) Figure 6 — Brit.Med.J. (1957), Vol. II — Turnbull, E.P.N. and Baird, D.

(vii) Figure 7 — J.Obstet.Gynaec.Brit. Commonw., (1963), Vol. 70 — Coyle, M.G. & Brown, J.B.

(viii) Figure 8 — J.Obstet.Gynaec.Brit.Emp., (1959), Vol. 66. — Kellar, R. et al.

(ix) Figure 9 — J.Obstet.Gynaec.Brit. Commow., (1962), Vol. 69 — Greig, M., Coyle, M.G. et al.

(x) Figure 10 — Lancet (1961), Vol. II — Elliott, P. and Inman, W.H.W.

(xi) Figure 11 — J.Obstet.Gynaec.Brit. Commonw., (1961) — Wood, C., Osmond-Clarke, E. and Murray, M.

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