# CORRELATION OF FOETAL OUTCOME WITH PLACENTAL CHANGES IN POSTDATED PREGNANCY*

by

M. SANGHMEETA,**
J. MIRCHANDANI,***
and
M. REYS,****

The increased perinatal loss and evidence of dysmaturity in the newborn in postdated pregnancy suggests impaired placental function. There are progressive morphological changes with ageing of the placenta. There is increased fibrin and calcium deposition with age, but the degree of these deposits is variable. The white infarcts are common in term and in postterm placentae, but these do not cause diffuse atrophy of the placental tissue or foetal death. With age, the size of the villus decreases, stromal fibrosis increases, there is thinning of the syncitial layer with increased aggregation of syncitial nuclei and there is diminution of the number of cytotrophoblastic cells.

*Material and Methods*

For the present study 50 placentae from normal cases with 37 to 41 week gestation and 50 placentae of more than 41 weeks or postterm gestation were studied. After the initial inspection for subchorionic flakes, it was weighed with membranes trimmed at the edge. One cubic centimeter of tissue from each of the four quadrants of maternal aspect of the placenta, avoiding any abnormal area, was cut and fixed in Zenker's fluid. The rest of the placenta was fixed in 10% formalin. The formalin fixed placenta was cut in strips of 0.5 cm. thickness in two planes at right angles and the extent of infarcted area and degree of calcification was noted. Four slices were selected from the central area and sections from maternal aspect of each was cut, fixed in alcohol and embedded in paraffin. One hundred terminal villi were counted in each of the four haemotoxylin and eosin stained sections, the number of villi with the syncitial knots were expressed as 'percentage' of these four hundred.

Four sections were taken from the tissue fixed in Zenker's fluid and stained with Heidenhains iron haemotoxylin stain (Wigglesworth 1962). The number of villi in which Langhans cells could be identified, were counted in 100 villi in each of the four sections and expressed as percentage.

*Observations and Comments*

*Placental Weight and Placental Coefficient*

The average weight of the postterm placenta was more than that of the term

*Part of thesis, M.D. Delhi University.
**Post-graduate Student in Obst. & Gynae., L.H.M.C. & H., New Delhi.
***Asstt. Prof. in Obstetrics & Gynaecology, Lady Hardinge Medical College & Hospital, New Delhi.
****Associate Professor of Pathology, Lady Hardinge Medical College & Hospital, New Delhi.
Received for publication on 14-11-73.

placenta and placenta co-efficient, i.e. the ratio of weight of the baby to placenta also increased from 0.17 at term to 0.19 in postdated (Table Ia).

TABLE I (A)

*Average Weight of Placenta, Baby and Placental Co-efficient in Postdated Pregnancy*

| | Average weight of placenta | Average weight of baby | Placental co-efficient |
|---|---|---|---|
| Control | 479.9 G | 2884.4 | 0.17 |
| Post dated | 600.0 | 3160.8 | 0.19 |
| 41 Week | 616.0 | 3153.0 | 0.195 |
| 42 „ | 605.5 | 3231.0 | 0.187 |
| 43 „ | 518.0 | 3048 | 0.191 |

While no placenta in the control group weighed more than 600 G., 20% of postdated placentae weighed over 600 grammes. Garrow and Hawes (1971) suggested that the increased weight of placenta is due to increased blood trapping as the weight of the blood free postterm placenta was not increased.

Potter (1952) considered that except with twins there is little evidence that foetal death is ever a result of insufficient placental size. The dysmaturity syndrome is found to be more frequent when placental co-efficient is increased above 0.17 in postdated group, while foetal distress, stillbirth and neonatal deaths are least with placental co-efficient between 0.17 to 0.19 (Table Ib).

*Fibrin Deposits*

The deposition of fibrin in intervillous space occurs as a result of placenta aging (Nesbitt 1958; Wilkin 1961; Marias 1962). Fox (1963) considered deposition of fibrin to be due to slowing of the circulation. It is found even at term though to a greater degree in postterm placenta. The frequency of the subchorionic fibrin deposition in an area more than 1 sq. cm. is 3 times more, i.e. in 48% of postterm placenta as compared to 16% of term placenta, as seen in Table II. However, foetal loss does not seem to be related to the extent of fibrin. In postterm group there were 2 stillbirths with fibrin deposit of 1 sq. cm. area. There was no foetal loss at term even when fibrin deposit in placenta was more than 5 sq. cm.

*Placental Calcification*

Calcification is a comon finding in mature placenta. Radiologically 20.83 per cent of mature placenta were shown to be heavily calcified by Master and Clayton (1940). Wentworth (1965) did not agree with Fox (1963) that heavy calci-

TABLE I(b)

*Correlation of Foetal Outcome With Placental Co-efficient in Postdated Pregnancy*

| Placental Co-ef. | 0.16 or less (7) | | 0.17 to 0.19 (27) | | 0.2 or above (16) | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| Foetal distress | 1 | 14.28 | 3 | 11.1 | 3 | 18.75 |
| Still birth | 2 | 28.56 | 1 | 3.7 | 3 | 18.75 |
| Neonatal death | 1 | 14.28 | 1 | 3.7 | 3 | 18.75 |
| Dysmaturity | 1 | 14.28 | 9 | 33.3 | 6 | 37.5 |

TABLE II

*Area of Subchorionic Fibrin in 50 Control and 50 Postdated Placentae*

| Subchorionic fibrin plaque | Control | | Postdated | |
|---|---|---|---|---|
| | No. | % | No. | % |
| Nil | 24 | 48 | 14 | 28 |
| 1 Sq. Cm. | 18 | 36 | $12_{(2)}$ | 24 |
| 1.5 Sq. Cm. | 6 | 12 | $16_{(1)}$ | 32 |
| More than 5 Sq. Cm. | 2 | 4 | $8_{(3)}$ | 16 |

Figures in brackets indicate still birth.

fication is found more frequently in postterm placenta.

In the present study calcification was graded as 1+ or ++; postterm placenta did show ++ calcification more frequently (Table IIIa). Thirty-four (68%) out of 50 postdated placentae had no gross calcification, though foetal distress, stillbirth, neonatal deaths, dysmaturity did occur in this group. However, there was definite increase in number of stillbirths and dysmaturity in postdated cases with calcified placenta (Table IIIb).

Fox (1964) found that the incidence of foetal distress and neonatal asphyxia is considerably more in the group with calcified placentae. Wentworth (1965) did not consider calcification to be of any clinical significance.

TABLE III a

*Calcification in Control and Postdated Placentae*

| Calcification | Control | | Post-dated | |
|---|---|---|---|---|
| | No. | % | No. | % |
| 0 | 38 | 78 | 34 | 68 |
| + | 9 | 18 | 10 | 20 |
| ++ | 2 | 4 | 6 | 12 |
| | 50 | 100 | 50 | 100.0 |

*Infarction*

Siegel (1963) found that the white infarct which is common in postterm placentae, does not cause foetal death.

In the present study, only 6% of the control placentae as compared to 38% of

TABLE III b

*Correlation of Placental Calcification and Foetal Outcome in Postdated Pregnancy*

| | Calcified 16 | | Non-calcified 34 | |
|---|---|---|---|---|
| | No. | % | No. | % |
| Number of stillbirth | 3 | 18.75 | 3 | 8.82 |
| Number of foetal distress | 5 | 31.25 | 10 | 29.4 |
| Dysmaturity | 10 | 62.5 | 6 | 17.64 |
| Neonatal death | 2 | 12.5 | 3 | 8.82 |

the postterm placentae had white infarct involving more than 10% of placental volume.

In 3 out of 6 postterm placentae with stillbirth, infarct was more than 10% of the volume, in 2 it was 5 to 9.9 per cent of volume, while one occurred with infarct of less than 5 per cent volume. No haemorrhagic infarct was found in postterm placentae (Table IV).

TABLE IV

*Degree of Infarction in Control and Postdated Placenta*

| Percentage volume of placentae involved in infarction | Control 50 | | Postdated 50 | |
|---|---|---|---|---|
| | No. | % | No. | % |
| Below 5 | 27 | 54 | 18 (1) | 36 |
| 5 to 9.9 | 20 | 40 | 13 (2) | 26 |
| 10 or above | 3 | 6.0 | 19 (3) | 38 |

Figures in brackets show number of stillbirths in each group.

*Syncitial Knots*

With aging of the placenta, there is thinning of the syncitial membrane and aggregation of syncitial nuclei. The higher frequency of syncitial knots in postdated placenta has been noted by various workers, the presence of knots in more than 30 per cent of the villi is reported by Merril (1963), Kubli and Budlinger (1963), Fox (1964), Malkani and Bhasin (1968) and Mehrotra *et al* (1972). In the present study, syncitial knot count above 30% was found in 52 per cent of postdated placentae, but also in 10% of the control placentae (Table Va). The frequency of high syncitial count increases with the duration of gestation (Table Vb).

TABLE Va

*Syncital Knot Count in Control and Postdated Placenta*

| Period of gestation | Less than 11% | 11-20% | 20-30% | More than 30% |
|---|---|---|---|---|
| Control group (50) | 19 | 12 | 14 | 5 |
| Per cent | 38% | 24% | 28% | 10% |
| Postdated group (50) | 3 | 4 | 17 | 26 |
| Per cent | 6% | 8% | 34% | 52% |

TABLE Vb

*Incidence of High Syncitial Count With Duration of Pregnancy*

| Duration of pregnancy | Total No. | No. with syn. count More than 30% | Percentage |
|---|---|---|---|
| 37-38 weeks | 20 | 0 | 0 |
| 38-40 weeks | 30 | 5 | 16.6 |
| 41 weeks | 10 | 4 | 40.0 |
| 42 weeks | 25 | 12 | 48 |
| 43 weeks or more | 15 | 10 | 66.6 |

Getzowa and Sadowski (1950) considered syncitial knot formation as indicative of placental inactivity, while Thomson (1954) considered it to be a result of ischaemia due to decreased maternal blood flow. Fox (1954) thought it resulted from decreased foetal blood as syncitial knots were often associated with non-sinusoid capillaries in villi. Tominaga and Page (1966) found that aggregation of syncitial nuclei occurs when the oxygen tension in the chamber containing villus tissue is reduced, indicating it to be the result of hypoxia.

Very few workers have correlated syncitial knots with foetal outcome, Fox (1964) found no correlation.

Out of total 50 postdated placentae in the present study, 26 had syncitial count above 30% and these 26 cases were associated with 5 of the total 6 stillbirths, 3 out of 5 neonatal deaths, 12 out of 15 cases with foetal distress and all 16 cases of dysmaturity, suggesting a definite correlation of high syncitial count and poor foetal outcome (Table Vc).

*Langhan Cell Layer*

For several years, persistence of Langhans cells throughout pregnancy has been suspected, finally it was proved by use of Haily's fluid fixation and Heidenhains iron haemotoxylin stain by Wigglesworth (1962). Fox (1964) after extensive study concluded that Langhan cells were seen in less than 20% of the villi in 62.5% of normal term placentae. If present in more than 20%, the count was considered high and in more than 40% of villi, it indicated a marked degree of proliferation. Fox (1964) found the count high in prolonged pregnancy. The ischaemia due to decreased maternal blood is supposed to cause the cytotrophoblastic proliferation, Brown and Veal 1953; Morris *et al*, 1965; Mackay, 1958; Fox, 1964.

In the present study (Table VIa) Langhan cells were detected in 60% of control placentae and in all the postdated placentae. None of the control placentae had Langhan cells in more than 20 per

TABLE Vc

*Correlation of Foetal Outcome of Syncitial Knot Count*

| Syn. Knot | Less than 30% (24) | | More than 30% (26) | |
|---|---|---|---|---|
| | No. | % | No. | % |
| Foetal distress | 3 | 12.5 | 12 | 46.15 |
| Stillbirth | 1 | 4.12 | 5 | 18.16 |
| Neonatal death | 2 | 8.33 | 3 | 11.53 |
| Dysmaturity | – | – | 16 | 61.53 |

TABLE VIa

*The Presence of Langhans Cells in Control and Postdated Placenta*

| Villi containing Longhans Cells | Control 50 | | Postdated 50 | |
|---|---|---|---|---|
| | No. | % | No. | % |
| Nil | 20 | 40 | — | — |
| 5-10% | 25 | 50 | 28 | 56 |
| 11-20% | 5 | 10 | 16 | 32 |
| Above 20 | — | — | 6 | 12 |

cent of the villi, while 16 (32%) postdated placentae had Langhans cells in 11-20 per cent villi and 6 (12%) had Langhans cells in more than 20 per cent villi. These 6 placentae were significantly associated with 3 stillbirths, 4 dysmature babies and foetal distress in 3 cases (Table VIb).

TABLE VIb

*Correlation of Foetal Outcome With the Presence of Langhans Cells in Postdated Group*

| Percentage of villi containing Langhans cells | Less than 20% (44) | | More than 20% (6) | |
|---|---|---|---|---|
| Foetal distress | 12 | 27.27% | 3 | 50% |
| Stillbirth | 3 | 6.82% | 3 | 50% |
| Dysmaturity | 12 | 27.27% | 4 | 66.61% |

## *Summary and Conclusions*

At present there is a lack of understanding about the exact nature of placental insufficiency and the cause of low foetal reserve in postdated pregnancy.

There are progressive morphological changes and functional alterations with aging of the placenta. There is increased average weight of the placenta which may be due to increased blood trapping. The placental co-efficient, calcification and white infarct also increase with the duration of pregnancy. There is increased syncitial nuclear aggregation and cytotroblastic cell proliferation, which probably indicates ischaemia and anoxia of the chorionic villus.

The foetal outcome was studied in relation to these morphological changes of the placenta.

There was more often evidence of dysmaturity in the newborn with increased placental co-efficient and calcification, but the extent of white infarct and subchorionic fibrin had no correlation with the condition of the foetus.

The high syncitial knot count i.e. the presence of syncitial knots in more than 30% of the villi increased steadily from 38 weeks onwards and all foetal paranatal deaths and dysmaturity were increased with high syncitial count. Similar deterioration of foetal condition was found with the Langhan cell proliferation i.e. when these were seen in more than 20% of the villi. Such high count was not found in the term placentae. These trophoblastic changes with poor foetal outcome reflect a deterioration in the intrauterine milieu or in the foetal-placental functional unit. Ischaemia and hypoxia is supposed to be the cause of these trophoblastic changes. Though of no prospective value, these trophoblastic alterations may help in allocating the cause of foetal death in retrospect.

## *Acknowledgement*

We wish to thank Dr. S. Acyaya, the Principal, Lady Hardinge Medical College & Hospital for Women, New Delhi and Dr. P. Madan, Professor of Obstetrics & Gynaecology, for granting the permission to collect the data and publish this paper, we are grateful to various members of the staff of the department of Obstetrics & Gynaecology and the Department of Pathology for their co-operation.

*References*

1. Browne, J. C. M. and Veall, N.: J. Obst. & Gynec. Brit. Emp., **60**: 141, 1953.

2. Fox, H.: J. Obst. & Gynec. Brit. Cwlth., 70: 980, 1963.
3. Fox, H.: J. Obst. & Gynec. Brit. Cwlth., 71: 759, 1964.
4. Fox, H.: J. Obst. & Gynec. Brit. Cwlth., 71: 885, 1964.
5. Garrow, J. S. and Hawes, S. F.: J. Obst. & Gynec. Brit. Cwlth., 78: 22, 1971.
6. Getzowa, S. and Sadowsky, A.: J. Obst. & Gynec. Brit. Emp., 57: 388, 1950.
7. Kubli, F. and Budlinger, H.: Geburtsh. u. Frauenheilk. 23: 37, 1963.
8. Little, W. A.: Obst. & Gynec., 15: 109, 1960.
9. Mackay, D. G.: Obst. & Gynec., 12: 1, 1958.
10. Malkani, P. K. and Bhasin K.: J. Obst. & Gynec. India, 18: 666, 1968.
11. Marias, W. D.: J. Obst. & Gynec. Brit. Cwlth., 69: 944, 1962.
12. Masters, M. and Clayton, S. G.: J. Obst. & Gynec. Brit. Emp., 47: 457, 1940.
13. Mehrotra, V. G., Mukherjee, K., Pande, M., Gurtu, P. and Mukherjee, M.: J. Obst. & Gynec. India, 22: 248, 1972.
14. Merril, J. R.: Clin. Obst. & Gynec., 6: 96, 1963.
15. Morris, E. D. and Beard R. W.: J. Obst. & Gynec. Brit. Cwlth., 72: 489, 1965.
16. Nesbitt, R. E. L.: Clin. Obst. & Gynec., 1: 349, 1958.
17. Potter, E.: Pathology of the Foetus and Newborn. Chicago Year book Publishers Inc. 1952.
18. Siegel, P.: ZBL Gynec., 85: 1327, 1963.
19. Thomson, K.: Arch. Gynec., 185: 221, 1954.
20. Tominaga, T. and Page, W. E.: Am. J. Obst. & Gynec., 94: 679, 1966.
21. Wentworth, P.: J. Obst. & Gynec. Brit. Cwlth., 72: 215, 1965.
22. Wigglesworth, J. S.: J. Obst. & Gynec. Brit. Cwlth., 69: 385, 1962.
23. Wigglesworth, J. S.: J. Obst. & Gynec. Brit. Cwlth., 69: 934, 1962.
24. Wilkin, P.: Irish J. Med. Sci. 6th Ser. 200, 1961.