FOETAL DISTRESS

by

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In recent years greatly improved obstetric care has caused dramatic fall in the rates of maternal mortality, still-births and early neonatal deaths, but various degrees of permanent anoxic damage of the foetus due to intra-uterine distress are of far-reaching importance. The aim should be that all infants go home from the hospital not only alive but with the maximum prowess and minimal behavioural abnormality. The present review is based on 760 cases of foetal distress out of 25,117 confinements in Eden Hospital during the period 1963-64. In these 760 cases (3.2%) one or more of the accepted signs of foetal distress were found, namely, the presence of meconium in the liquor amnii and/or alterations in the foetal heart sounds such as heart rate persistently going above 160 beats per minute or persistent slowing to below 110 beats a minute, or persisting irregularity of the rhythm.

According to the aetiological factors, the cases of foetal distress were distributed as follows:

TABLE I
Causes of foetal distress

Causes	No. of cases	Per cent
Pre-eclamptic toxaemia	180	23.76
Eclampsia	18	2.43
Postmaturity	226	29.7
Antepartum haemorrhage: both placenta praevia and accidental haemorrhage	136	17.9
Prolonged labour due to tonic contractions of the uterus	82	10.8
Prolonged compression of the foetal head due to C.P.D.	49	6.45
Placental insufficiency	26	3.65
Cord around the neck	10	1.4
Decompensated heart disease	8	1.1
Respiratory failure	6	0.8
Acute infections	4	0.5
Severe anaemia	15	1.9
Total	760	100.0

The incidence of foetal distress varied according to maternal age, parity, duration of pregnancy, labour (spontaneous or induced), method of induction, condition of the os at the time of induction and duration of labour.

Table II shows the significance of maternal age in foetal distress:—

TABLE II
Age distribution

Age in years	Per cent
Below 20	10.0
20-25	15.4
26-30	22.5
31-35	28.2
Above 35	23.9

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It is apparent that the incidence of foetal distress rises sharply with increased maternal age. About 50% cases were in the 25-35 years age group.

Table III shows the distribution of parity in these cases.

TABLE III

Parity

Parity	Per cent
Primigravida	23.7
Para 2	18.2
Para 3	14.6
Para 4	16.5
Para 5 and above	27.0

Foetal distress was commonest in the primigravidas and that too was influenced by maternal age. It increased from 3 per cent in young primigravida below the age of 30 years to 36 per cent in elderly primigravida above the age of 30 years.

Table IV shows the duration of pregnancy in these cases.

TABLE IV

Duration of pregnancy

Duration of pregnancy	Per cent
28-32 weeks	30.8
33-36 weeks	28.4
37-40 weeks	13.2
41-43 weeks	27.6

The incidence of foetal distress was minimum during 37-40 weeks (13.2%).. It was higher in premature labour under 36 weeks but increased considerably in prolonged pregnancy. At 43 weeks the incidence of foetal distress was 24.4%, whereas at 40 weeks, it was 6.2%.

Table V indicates the relationship between incidence of foetal distress and mode of onset of labour — whether spontaneous or induced and the latter again depending on method adopted in induction of labour.

TABLE V

Mode of onset of labour and foetal distress

	Per cent
Spontaneous labour	12.4
Induced labour	32.8
Medical induction	18.6
High rupture of membranes ..	10.2
Low rupture of membranes ..	4.0

The above table shows that in spontaneous labour the incidence of foetal distress was much less than in induced labour, 12.4% and 32.8% respectively. In induced labour again medical induction was more commonly associated with foetal distress (14.6%) than surgical induction (14.2%). In the latter, high rupture of membranes was more commonly associated with foetal distress than low rupture of membranes (10.2% and 4.0% respectively). Condition of the os at the time of rupture of membranes was also interlinked with the incidence of foetal distress. The more rigid the cervix the greater was the risk of developing foetal distress.

The increased incidence of foetal distress in the induced group can be explained by the fact that labour is usually induced in pre-eclamptic toxaemia, eclampsia, ante-partum haemorrhage post-maturity essential hypertension, chronic nephritis etc. and these are the causes for foetal distress.

Duration of labour had definite relation with the incidence of foetal distress. The latter was 3.5 per cent in

easy labour and 8.4 in difficult labour.

Significance of signs of foetal distress is shown in relation to the important signs of foetal distress in Table VI.

TABLE VI

Perinatal loss in relation to signs of foetal distress

Signs of foetal distress	No. of cases	Perinatal loss
Thin meconium	180	20
Thick meconium	262	30
Meconium & heart signs	224	45
Heart signs only	94	3
Total	760	98 (13%)

The above table shows that the perinatal loss increased with the thickness of meconium in liquor amnii and it was maximum when thick meconium was associated with abnormal foetal heart sounds. In the presence of clear liquor amnii foetal heart signs alone could not account for increased foetal loss.

TABLE VII

Perinatal loss in relation to abnormal foetal heart rate

Foetal heart rate	Meconium-stained liquor Perinatal loss	Clear liquor Perinatal loss
Heart normal	7.2%	—
Heart slow	2.5%	3.2%
Heart slow & irregular	14.8%	—
Heart fast	8.3%	—
Heart fast & irregular	11.2%	—

Table VII shows that when liquor amnii was clear there was no appreciable increase in foetal loss (3.2%) irrespective of the nature of the foetal heart. On the other hand when liquor amnii was meconium-stained there was considerable increase in the perinatal loss, particularly in presence of abnormal foetal heart rate. It was worst when foetal heart was slow and irregular. Even when foetal heart was normal perinatal loss was high (7.2%) in presence of meconium-stained liquor amnii.

Table VIII shows the effect of duration of foetal distress upon perinatal death rate.

TABLE VIII

Effect of duration of foetal distress upon perinatal death rate

Duration of foetal distress	Perinatal death rate
1- 4 hours	7.8%
4- 8 hours	10.3%
8-12 hours	14.2%

From the above table it is obvious that with the prolongation of duration of foetal distress there was proportionate increase in perinatal death rate.

Table IX shows the effect of complications associated with foetal distress on perinatal loss.

This table shows that without associated complications the perinatal loss was not much altered in foetal distress (6.8%) but with

TABLE IX

Effects of complications associated with foetal distress on perinatal death rate

Complications	Perinatal death
No recognized complication ..	6.8%
Cephalo-pelvic disproportion ..	19.3%
Pre-eclamptic toxaemia	7.5%
Uterine inertia	8.2%
Intra-uterine infection	16.2%

complications perinatal death rate was 15.8%. It was very high when foetal distress was associated with cephalo-pelvic disproportion, and intra-uterine infection (19.3% and 16.2% respectively).

Table X shows the effect of mode of delivery on perinatal death in foetal distress.

TABLE X

Effect of mode of delivery on perinatal death in foetal distress

Mode of delivery	P.N.D.
Spontaneous vaginal delivery ..	9.6%
Caesarean section	6.5%
Mid-forceps delivery	32.4%
Low forceps delivery	7.2%

From the above table it appears that the foetal survival rate was worst in difficult mid-forceps delivery in presence of foetal distress. There was very little difference in perinatal death rate whether baby was delivered spontaneously or by low forceps or by caesarean section. Possibly it was comparatively better when baby was delivered quickly by caesarean section in presence of foetal distress.

Table XI shows the effect of disproportion on perinatal death in foetal distress. The 760 cases of foetal distress were divided into two groups with and without cephalo-pelvic disproportion, and perinatal deaths were considered according to mode of delivery.

TABLE XI

Effect of disproportion on perinatal death in foetal distress

Mode of delivery	Disproportion present Perinatal loss	Disproportion absent Perinatal loss
Spontaneous vaginal delivery	11.6%	7.8%
Caesarean section	8.4%	6.1%
Mid-forceps	38.1%	14.4%
Low forceps	9.2%	7.1%
Average	20.6%	9.4%

The above table shows that the average perinatal death in presence of cephalo-pelvic disproportion was much higher than that in cases without any disproporton (20.6% and 9.4% respectively) whatever might be the nature of delivery. Comparatively the result was best in caesarean section and worst in difficult mid-forceps delivery in both the groups of cases.

Discussion

The diagnosis of foetal distress during the ante-partum or intrapartum period depends mainly upon the recognition of two signs, namely presence of meconium in the liquor amnii and alteration in the foetal heart sounds, either becoming slow or rapid or irregular. Abnormal foetal movement is not given much importance in this respect.

It is difficult to assess accurately any abnormality in foetal heart sounds. The latter can be studied by

(a) auscultation (b) foetal phonocardiograph and (c) foetal electrograph (Hon, 1959).

Pinkerton *et al.* (1961) suggested that foetal heart rate should be recorded at least every 15 minutes in early labour and every 5 minutes in late labour particularly in patients where the risk of foetal death is very high. There may be foetal tachycardia, bradycardia or irregular beats. All agree that foetal bradycardia means greater foetal distress, but there is difference of opinion on the exact point beyond which slowing of the heart rate becomes pathological. According to Gibberd (1947) and Greenhill (1951) 100 beats per minute should be considered as critical points.

Brews (1953) stands alone in regard to the foetal heart rate in distress. He believes that foetal tachycardia above 160 beats per minute should be considered a more important sign of foetal distress than foetal bradycardia.

Chasser Moir (1956) opines that steady slowing of the heart rate below 110 beats per minute is a certain evidence of foetal distress. If irregularity of foetal heart is superimposed on slowing that should be considered as an indication of impending foetal death.

Hon (1959) studied foetal heart rate in various abnormal conditions of pregnancy and labour by foetal phonocardiograph and electrocardiograph. He believes that prolonged foetal heart slowing during uterine contraction is a definite sign of foetal distress. Onset of this slowing occuring early in the contraction phase is due to prolapse of the umbilical cord, and late in the contraction it is due to anoxia from other causes.

All the authorities agree that foetus is in grave danger if the foetal heart is both slow and irregular or if bradycardia accompanies the passage of thick meconium in the liquor amnii.

Meconium staining of liquor amnii: This is considered by majority of the authors to be a most important sign of foetal distress. Presence of meconium in liquor amnii is supposed to be due to increased persistalsis of the gut in response to foetal anoxia.

Even in this regard there are lots of doubts and confusion about its meaning and significance in the diagnosis of foetal distress. Macafee and Bancroft Livingston (1958) are of the opinion that the presence of meconium in liquor amnii as a sign of foetal distress has been over-emphasized. There are reasons other than foetal anoxia which cause the passage of meconium in liquor amnii. It is influenced by the volume of liquor amnii, the speed in which liquor amnii is drained and the total amount of meconium passed by the foetus. If the passage of meconium is to be diagnosed during the early development of foetal distress and before spontaneous rupture of membranes, artificial rupture of membranes should be performed, particularly in patients with an increased risk of foetal anoxia or when the foetal heart rate is abnormal.

Lister and Buckanan (1957), on the other hand, believe that meconium staining of liquor amnii is the sign of ominous import in foetal distress. The present study confirms that if thick meconium is combined

with abnormal foetal heart rate foetal risk is definitely increased. The perinatal loss associated with meconium stained liquor is three times greater than when liquor is clear (Table VII). This increased foetal loss is further exaggerated if there is any associated obstetrical complication, e.g., cephalo-pelvic disproportion, pre-eclampsia, uterine inertia etc. (Table IX). Uncomplicated foetal distress, on the other hand is a relatively less dangerous condition, but nevertheless the foetal loss is greater than when no distress is present.

Management

Here also opinions vary much. According to Macafee and Bancroft Livingstone (1958) foetal distress is an indication to search for the cause rather than a demand for dramatic action.

Gibberd (1957) believes that caesarean section offers no guarantee to a live child which is already distressed. Perinatal death in caesarean section is the same as in vaginal delivery in the absence of any other complication like cephalo-pelvic disproportion, pre-eclamptic toxaemia etc.

N. Walker (1959) observes that active intervention in labour for foetal distress does not alter the prognosis for the baby. When foetal distress already develops, it goes to foetal death so rapidly that any active intervention may be too late to save the foetus.

J. Walker (1959), on the other hand, believes that foetal distress carries a fairly high mortality indicating interference and expediting delivery of the baby.

The present study also confirms that when the foetus is really distressed some interference is justified to hasten the delivery of the baby. That definitely improves the foetal salvage (Table 11).

Whenever foetal distress is detected oxygen inhalation should be given to the mother and search be made to find out the cause of foetal distress and associated complications like maternal age, parity, cephalo-pelvic disproportion, pre-eclampsia etc. Active intervention is indicated under the following conditions depending on the condition of the os: (a) elderly primigravida with relative subfertility, (b) pre-eclamptic toxaemia, (c) cephalo-pelvic disproportion. If labour is likely to be prolonged and not easy, caesarean section is justified.

If the os is nearly fully dilated and vaginal delivery is not contraindicated, forceps or vacuum extractor may be used to cut short the second stage of labour.

Internal version may have some place, particularly when a second twin is in distress, after the birth of the first one. We must always keep in mind that the birth of a live child is not necessarily the hall-mark of good obstetrics. A child subjected to hypoxia during labour may be mentally crippled afterwards. So the time for interference should be decided with utmost precision.

Summary

760 cases of foetal distress have been analysed from various angles.

Problems of diagnosis and management of foetal distress have been stressed.

Effects of various signs of foetal distress and associated obstetrical complications like cephalo-pelvic disproportion, pre-eclampsia, ante-partum haemorrhage on perinatal death have been critically studied.

A plea has been made for a thorough search for the cause rather than taking a dramatic step in cases of foetal distress.

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