which is cheap, simple, and accurate. Urinary purine chromatography will confirm the diagnosis, and spectrographic analysis of the stone is also conclusive. It is interesting that Professor Pierre Cartier has discovered by spectrography two examples of xanthine stones amongst an old collection of renal stones (Boissonat, 1957). This emphasizes that xanthine stones can be mistaken for uric acid unless specific analytical methods are used. There is no doubt that many xanthine calculi must be overlooked, though we would certainly not claim that the condition is a common one. It is surprising to find that the metabolic defect in our patient has produced predominantly unilateral renal disease.

Summary

A clinical description is given, with a four-year follow-up, of the patient with xanthinuria described by Dent and Philpot in 1954. No change in metabolism has occurred; but the patient has been studied again, new specific enzymatic methods being used to determine uric acid, xanthine, and hypoxanthine.

Traces of uric acid were found only when the patient was taking a normal purine-containing diet. Xanthine was the predominant oxypurine present both in plasma and in urine. The renal clearance of xanthine was very high, probably indicating complete failure of normal tubular reabsorption. Hypoxanthine was also present, but its concentration was variable and low.

A single biochemical lesion could account for this disorder. We suggest that in this condition there is a minor, inborn, genetically determined abnormality in the protein moiety of the enzyme xanthine oxidase; and we believe that this same protein also plays a part in the renal tubular reabsorption of xanthine.

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THE CASE FOR CONSERVATISM IN MANAGEMENT OF FOETAL DISTRESS

BY

NORMAN WALKER, M.B., Ch.B., D.Obst.R.C.O.G.

Registrar, Department of Gynaecology and Obstetrics, University of Natal, Durban

The diagnosis of foetal distress during the course of labour is based primarily upon the recognition of two signs—the presence of meconium in the liquor amnii and alterations in the foetal heart sounds.

Reference to five standard textbooks of obstetrics show general agreement that meconium-staining of the liquor means that the foetus is or has been distressed. Likewise, all agree that foetal bradycardia means distress, but there is some difference of opinion on the exact point beyond which slowing of the heart rate becomes pathological.

Munro Kerr (Chassar Moir, 1956) regards a steady slowing of the heart rate to below 110 beats a minute as certain evidence of foetal distress, and irregularity superimposed on slowing as indicative of impending foetal death: Gibberd (1947) accepts a heart rate of below 110 a minute as meaning foetal distress: Eden and Holland (Brews, 1953) accept 100 a minute as the critical level and regard progressive slowing as an indication for immediate delivery; Claye (1955) states that a drop in heart rate of 25 beats a minute is of serious significance. Greenhill (1951) declares 100 beats a minute to be the critical point, but stresses that on occasion the child may be born alive and well despite persistent intra-partum bradycardia.

Eden and Holland stand alone in regarding foetal tachycardia as a sign of distress, and set the upper limit of normality at 160 beats a minute.

All these authorities agree that the foetus is in grave danger if the heart rate is persistently slow, if it is both slow and irregular, or if bradycardia accompanies the passage of thick meconium in the liquor amnii. All but one agree that such severe foetal distress indicates a need to rescue the foetus immediately either by forceps delivery or by caesarean section, according to the conditions obtaining. Gibberd strikes the more cautious note and states: "It is not often that foetal distress of itself is a good indication for caesarean section, but it may sometimes be possible to save the baby, for instance when the cord is prolapsed, if operation is undertaken without delay." This conservative attitude serves as a reminder that caesarean section is not without risk to the mother, for, as shown in the Report on Confidential Enquiries into Maternal Deaths in England and Wales (Ministry of Health, 1957), the operation carries a mortality rate which is seven times greater than that of vaginal delivery. This risk can be justified only if it is reasonably certain that a foetus showing one or more of these signs of distress is in fact in such danger that it will inevitably succumb.

A survey of the literature indicates that this is not necessarily so, because there is doubt and confusion in regard to the meaning and significance of the very signs upon which the decision to operate must be based.

For instance, Lister and Buchanan (1957) state that meconium-staining of the liquor amnii is the one single sign of ominous import to the foetus; whereas Macafee and Bancroft-Livingston (1958) hold the opinion that the appearance of meconium is a danger that has been overemphasized. Similarly, Bartholomew (1925), Freed (1927), Lund (1943), White (1955), and Ginsburg (1957) indicate that a rapid foetal heart is of no consequence; Fitzgerald and McFarlane (1955) and James Walker (1959) take the opposite view and declare that a rapid foetal heart is of graver significance than a slow heart. Theobald *et al.* (1956), on the other hand, ignore the foetal heart completely provided that the umbilical cord is not occluded.

Nevertheless, as Dumoulin and Martin (1957) have pointed out, one in every hundred mothers admitted to maternity hospitals in Great Britain is subjected to caesarean section solely on the grounds of foetal distress.

In view of the fact that the obstetric unit of this department controls more than 12,000 deliveries in a year, it seemed that the amount of material available could offer an opportunity to inaugurate a large-scale study of the problem.

The Present Study

Over 12.000 deliveries were studied at King Edward VIII Hospital, Durban. In 700 (5.5%) 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 persistent slowing of the heart rate to below 110 beats a minute, persistent acceleration of the rate to above 160 beats a minute, or persistent irregularity of the rhythm.

Premature deliveries (babies weighing less than 5½ lb. —2,500 g.), breech presentations (where meconium is present normally in the liquor amnii), and cases of cord prolapse were excluded from the study. The parturients included both Indian and Bantu women, with the latter predominating in the ratio of 10 to 1.

The study consisted of two equal parts: the first was a pilot survey of 350 distressed babies; the second was a controlled study of a further 350 distressed babies—the total series comprising 700 cases.

There was no deliberate selection of cases in the pilot survey. Several obstetricians were responsible for these deliveries: those who believed in the radical treatment of foetal distress tended to use operative methods of delivery, whereas those who were more conservative tended to follow a policy of non-intervention, provided, of course, that intervention was not indicated on other grounds.

The method of delivery of these 350 distressed babies in the pilot survey is shown in Table I, together with the associated perinatal loss—that is, those babies born dead or dying within the first week of life. In this group of cases the perinatal loss (5.4%) from spontaneous vaginal delivery was no greater than that following caesarean section (6.5%). The appalling loss associated with mid-forceps delivery is discussed later.

The experience and impressions gained from this pilot survey provided the confidence required to embark upon the second part of the study, in which a policy of non-intervention for foetal distress was contrasted with the conventional approach of paying heed and offering treatment when the typical signs of foetal distress had been present for a definite period of time.

TABLE I.—*Mode of Delivery of* 350 *Distressed Babies in Pilot Survey*

Mode of Delivery	No. of Cases	Perinatal Loss
Spontaneous vaginal delivery ..	165	9 (5·4%)
Caesarean section	107	7 (6·5%)
Mid-forceps delivery	45	17 (37·7%)
Low-forceps ,, ...	33	1 (3%)
Total	350	34 (9·7%)

TABLE II.—*Comparison of Results of Non-intervention With Active Intervention in Treatment of* 350 *Cases of Foetal Distress*

Mode of Delivery	Non-intervention		Intervention	
	No. of Cases	Perinatal Loss	No. of Cases	Perinatal Loss
Spontaneous vaginal delivery	141	8	68	0
Caesarean section	12	3	48	4
Symphysiotomy	14	1	23	7
Mid-forceps delivery ..	5	0	19	3
Low-forceps ,, ..	4	0	16	0
Total	176	12 (6·8%)	174	14 (8%)

The method of delivery to be employed—operative or non-intervention—was determined by drawing a sealed envelope from a drum. The results of this controlled investigation are shown in Table II. A strictly controlled investigation was impossible to organize, because 36 of the 176 cases in the non-intervention group had ultimately to be delivered by operative means because of obstetrical difficulties unconnected with foetal distress. Likewise 68 cases in the intervention group delivered spontaneously because delivery was imminent when the signs of foetal distress became manifest. Even so, the perinatal loss in these two groups is virtually the same—6.8% in the non-intervention group as against 8% in the intervention group. It appears, therefore, that a policy of non-intervention is not necessarily fraught with dire consequences.

It may be thought that these comparisons are not valid because cases have been included of so-called "mild" foetal distress in which the foetal mortality may be so low as to vitiate a true comparison. As indicated earlier, many observers would ignore thin meconium, whereas others would pay great heed to the association of meconium with a slow heart; the implication being that there are definite grades of severity of foetal distress. The 700 cases have therefore been classified into four groups and analysed to determine if foetal distress can in fact be graded into "mild" or "severe."

Significance of Signs of Foetal Distress

The perinatal loss in all 700 distressed babies is shown in relation to the presenting signs of foetal distress in

TABLE III.—*Perinatal Mortality in Relation to Signs of Foetal Distress*

	No. of Cases		Perinatal Loss	
Thin meconium	162	622	18 (11·1%)	61 (9·8%)
Thick ,, ...	244		19 (7·8%)	
Meconium+heart signs ..	216		24 (11·1%)	
Heart signs only	78		2 (2·6%)	
Total	700		63 (9%)	

Table III. Four groups are described: thin meconium, thick meconium, meconium with heart signs, and heart signs alone. Meconium was described as thin when the liquor was merely stained, and thick when it resembled "pea soup."

The foetal loss is much the same when meconium is present in the liquor amnii, no matter whether it is thin (11.1%), thick (7.8%), or associated with heart signs (11.1%). On the other hand, when the liquor amnii is clear and heart signs alone are present the foetal loss is 2.6%.

Even when the cases are analysed in relation to the method of delivery the same pattern is manifest. As shown in Table IV, 374 of the total 700 distressed babies were allowed to deliver spontaneously per vaginam. The perinatal loss in all three meconium groups was much the same—thin meconium 6%, thick meconium 3.1%, and meconium associated with heart signs 6.7%. There was no loss when heart signs alone were present.

There were 167 cases delivered by caesarean section (Table V). In the meconium groups the perinatal loss was 11.1% (thin), 5.7% (thick), and 12.9% (meconium plus heart signs). No loss occurred when the liquor was clear.

There were 122 forceps deliveries (Table VI). The high loss from mid-forceps application, as is shown below, is related to the presence of disproportion, but even with disproportion there is no evidence to suggest that any one sign of foetal distress, or a combination of them, offers a worse prognosis than any other. In the meconium group the perinatal loss was 50%, 47%, and 23% respectively, and with heart signs alone the loss was 20%.

TABLE IV.—*Results of 374 Spontaneous Vaginal Deliveries*

	No. of Cases	Perinatal Loss
Thin meconium	97	6 (6%)
Thick ,,	162	5 (3·1%)
Meconium + heart signs	90	6 (6·7%)
Heart signs only	25	0
Total	374	17 (4·54%)

TABLE V.—*Caesarean Section for Foetal Distress*

	No. of Cases	Perinatal Loss
Thin meconium	36	4 (11·1%)
Thick ,,	35	2 (5·7%)
Meconium and heart signs	62	8 (12·9%)
Heart signs only	33	0
Total	167	14 (8·4%)

TABLE VI.—*Forceps Deliveries*

	Mid-forceps		Outlet Forceps	
	No. of Cases	Perinatal Loss	No. of Cases	Perinatal Loss
Thin meconium	12	6 (50%)	7	0
Thick ,,	17	8 (47%)	23	1 (4·3%)
Meconium and heart signs	30	7 (23%)	18	0
Heart signs only	10	2 (20%)	5	0
Total	69	23 (33%)	53	1 (2%)

TABLE VII.—*Delivery by Symphysiotomy*

	No. of Cases	Perinatal Loss
Thin meconium	7	2 (28·6%)
Thick ,,	9	3 (33%)
Meconium and heart signs	16	3 (18·7%)
Heart signs only	5	0
Total	37	8 (21·6%)

Thirty-seven patients were delivered by symphysiotomy (Table VII), all performed, of course, for cephalo-pelvic disproportion. The same pattern is again manifest, an increased perinatal loss with meconium-stained liquor—28.6%, 33%, and 18.7% respectively—and no loss, with heart signs and clear liquor.

There is thus no evidence to suggest that foetal distress can be graded in severity. Indeed, thin meconium would appear to be as significant as thick meconium. On the other hand, alterations in the foetal heart sounds do not appear to worsen the prognosis for the baby. Indeed, when heart signs alone are present the mortality rate (2.6%) is no higher than might be expected with uncomplicated delivery.

TABLE VIII.—*Perinatal Loss in Relation to Signs of Foetal Distress*

	Meconium-stained Liquor		Clear Liquor	
	No. of Cases	Perinatal Loss	No. of Cases	Perinatal Loss
Heart normal	406	37 (9·1%)	—	—
,, slow	159	20 (12·6%)	53	2 (4%)
,, ,, and irregular	25	2 (8·0%)	22	0
,, fast	31	2 (6·4%)	3	0
,, ,, and irregular	1	0	0	0
	622	61 (9·8%)	78	2 (2·6%)

TABLE IX.—*Time-lag Between Onset of Heart Abnormality and Delivery in 78 Cases with Clear Liquor Amnii*

Time in Hours	No. of Cases	Perinatal Loss
0–1	20	1 (5%)
1–2	33	1 (3·3%)
2–3	16	—
3–4	6	—
4–5	1	—
5–6	2	—
Total	78	2 (2·6%)

To exclude the possibility that a large number of cases with a rapid foetal heart have been included—this being a sign ignored by most authorities—the exact abnormality of the foetal heart in relation to meconium-stained liquor and clear liquor is shown in Table VIII. Abnormal foetal tachycardia occurred on only 34 occasions.

Furthermore, it is clearly shown that foetal heart abnormalities — irrespective of their nature — do not necessarily carry an increased foetal loss (2.6%), provided that the liquor amnii is clear. Moreover, Table IX indicates that this low foetal loss is not of necessity related to a short time interval between the onset of the heart disturbance and actual delivery of the foetus. Of the 78 cases, 20 (26%) were delivered in less than one hour, 33 (42%) between one and two hours, and the remaining 25 (28%) between two and six hours after the onset of the foetal heart disturbance.

These figures indicate, however, that foetal distress is nevertheless attended by a higher perinatal loss than occurs in normal cases. In a series of deliveries in our unit without foetal distress the perinatal loss was 2.7%, whereas the overall foetal loss in the total 700 distressed cases was 9%.

In order to arrive at a true assessment of the risks of foetal distress other complications such as cephalo-pelvic disproportion, pre-eclamptic toxaemia, uterine inertia, and intrauterine infection must be excluded.

Effect of Complications

These four complications are analysed in Table X. Where foetal distress existed without other complications the perinatal loss was 3.6% in 358 cases. In 233 cases with cephalo-pelvic disproportion the perinatal loss was 18%. In other words, there is a fivefold increase with disproportion. These figures are statistically highly significant ($P<0.001$ on χ^2).

In toxaemia (68 cases), uterine inertia (29 cases) and intrauterine infection (12 cases), though the numbers in these groups are fewer and therefore less convincing, the foetal loss is also higher than when no recognized complication exists, being 5.9%, 6.9%, and 16.7% respectively.

TABLE X.—*Effect of Various Obstetrical Complications on Perinatal Loss in Foetal Distress*

Nature of Complication	No. of Cases	Perinatal Loss
No recognized complication	358	13 (3·6%)
Cephalo-pelvic disproportion	233	42 (18%)
Pre-eclamptic toxaemia	68	4 (5·9%)
Uterine inertia and prolonged labour	29	2 (6·9%)
Intrauterine infection	12	2 (16·7%)
Total	700	63 (9%)

TABLE XI.—*Effect of Disproportion on Perinatal Loss in Foetal Distress*

Mode of Delivery	Disproportion Present		No Disproportion	
	No. of Cases	Perinatal Loss	No. of Cases	Perinatal Loss
Spontaneous vaginal delivery	54	4 (7·4%)	320	13 (4%)
Caesarean section	88	10 (11·3%)	79	4 (5%)
Symphysiotomy	37	8 (21·6%)	0	0
Mid-forceps extraction	46	20 (43·5%)	23	3 (13%)
Low-forceps ,,	8	0	45	1 (2·2%)
Total	233	42 (18%)	467	21 (4·5%)

The grave significance of cephalo-pelvic disproportion may be further stressed by dividing the 700 cases into two groups—disproportion and non-disproportion—and showing the perinatal loss in relation to the method of delivery (Table XI). It is obvious that whatever the method of delivery the perinatal loss is increased in the disproportion group. Of particular importance is the appalling loss when mid-forceps are used in disproportion cases with foetal distress: almost half the babies died (20 out of 46). Even when symphysiotomy was used to relieve the disproportion more than 20% of the babies were lost—presumably because excess moulding of the foetal head, prolonged anoxia, or perhaps cerebral haemorrhage had already occurred.

In sum, therefore, the overall perinatal loss in 342 deliveries associated with an obstetrical complication was 14.6%, whereas in 358 cases in which no complication was recognized the loss was 3.6%.

Perinatal Loss in Relation to Method of Delivery

The perinatal loss relating to the various methods used to deliver all 700 distressed babies is shown in Table XII. In 374 spontaneous vaginal deliveries the loss was 4.5%, whereas in 167 caesarean sections the loss was 8.4%.

The high loss following mid-forceps delivery (33.3%) and symphysiotomy (21.6%) is of course due to the associated factor of cephalo-pelvic disproportion. Low-forceps delivery, on the other hand, shows a perinatal loss (2%) which is no greater than the loss (2.7%) which might be expected to occur in vaginal deliveries without foetal distress. This suggests that the more frequent use of low-forceps application might save additional distressed babies.

TABLE XII.—*Method of Delivery of 700 Distressed Babies*

Method of Delivery	No. of Cases	Perinatal Loss
Spontaneous vaginal delivery	374	17 (4·5%)
Caesarean section	167	14 (8·4%)
Symphysiotomy	37	8 (21·6%)
Mid-forceps delivery	69	23 (33·3%)
Low-forceps ,,	53	1 (2%)
Total	700	63 (9%)

The low foetal mortality (4.5%) achieved by spontaneous vaginal delivery is not related to the inclusion of a large number of "mild" cases of foetal distress. Table IV shows that 97 (26%) of the 374 spontaneous vaginal deliveries presented with thin meconium; 162 (43%) presented with thick meconium; 90 (24%) with both meconium and heart signs; and 25 (7%) with heart signs and clear liquor amnii. It may be thought, however, that these good results following spontaneous vaginal delivery in foetal distress occurred as a result of rapid delivery following the onset of the signs of distress.

Effect of Duration of Foetal Distress Upon Perinatal Loss

The time lag between the recognition of foetal distress and actual delivery in the 374 spontaneous vaginal deliveries is shown in Table XIII.

Most of the patients (279) did deliver spontaneously within four hours; it will be noted, however, that even where foetal distress had lasted for more than 12 hours one baby was lost in 14 deliveries (7%). Furthermore, there were in fact 95 cases in which foetal distress lasted for more than four hours before delivery with a loss of 7.4%—a figure which is not markedly higher than the loss (3.6%) for deliveries in less than four hours.

TABLE XIII.—*Time-lag Between Onset of Distress and Delivery in 374 Cases of Foetal Distress Allowed to Deliver Spontaneously*

Time in Hours	No. of Cases		Perinatal Loss	
0–1	54		3 (5·6%)	
1–2	105		3 (2·8%)	
2–3	83	279	3 (3·6%)	10 (3·6%)
3–4	37		1 (2·7%)	
4–5	21		1 (4·8%)	
5–6	15		—	
6–7	9		1 (11·1%)	
7–8	17		2 (11·8%)	
8–9	8	95	1 (12·5%)	7 (7·4%)
9–10	5		—	
10–11	4		1 (25%)	
11–12	2		—	
Over 12 hours	14		1 (7%)	
Total	374		17 (4·54%)	

There is, nevertheless, a tendency for the perinatal loss to rise with prolongation of labour, but the actual figures are too few to permit of any firm conclusions.

Discussion

From the figures presented in Tables III to VIII it is clear that meconium-staining of the liquor amnii means foetal distress and is a sign which indicates a risk to foetal survival that is increased threefold.

There is, however, no statistical difference between perinatal loss associated with thin meconium (11.1%)

and thick meconium (7.8%) in this series. It seems, therefore, despite Resnick's (1955) findings to the contrary, that no useful purpose is served by maintaining this distinction, which, after all, is a matter of opinion and must vary from one observer to the next.

Furthermore, the association of meconium-stained liquor amnii and abnormal foetal heart sounds does not necessarily indicate an increased risk to foetal survival: the perinatal loss (11.1%) in the cases displaying this combination of signs was not significantly greater than when meconium was present alone (9%). This finding casts doubt on the generally accepted belief that meconium-stained liquor together with an abnormal foetal heart—particularly a slow or irregular heart—means severe foetal distress and generally presages death.

Moreover, in 78 cases in which the foetal heart was abnormal and the liquor amnii clear, the perinatal loss (2.6%) was no greater than that which could be expected to occur (2.7%) in deliveries without foetal distress.

James Walker (1959), from his extensive studies of foetal distress, has concluded that slowing of the foetal heart is of little significance provided that the liquor amnii is clear. He bases this opinion upon a perinatal loss of 1.9% in 270 such cases—figures which are even more impressive than those of this study.

However, he attributes this low perinatal mortality to the fact that most of his cases achieved delivery soon after slowing of the heart had become manifest, and intimates that perinatal loss must rise with prolongation of the interval between onset of slowing and actual delivery. Nevertheless, the figures presented above offer some evidence that foetal death is not necessarily an inevitable sequel even when foetal bradycardia has existed for several hours prior to delivery.

Furthermore, James Walker disagrees with the majority of observers regarding the significance of a rapid foetal heart and considers that foetal tachycardia above 160 beats a minute implies a grave risk to the foetus, more particularly in the presence of meconium-stained liquor. He bases this belief on a series of 38 such cases with 7 perinatal deaths (18%). In this study there were 2 (6.4%) perinatal deaths in 31 similar cases. Obviously neither series is large enough to be of statistical significance.

If the thesis is correct that thin meconium carries as bad a prognosis for the baby as meconium together with heart signs, is it justifiable to rescue the baby from its dangerous environment even when there is only a tinge of meconium in the liquor amnii? Not infrequently, meconium-staining of the liquor may pass unnoticed until after delivery because the membranes happen to remain intact, or because the escape of liquor is prevented by the presenting part acting in the birth canal like a stopper in a bottle. Under such conditions the obstetrician is content, willy-nilly, to allow labour to proceed, and it is common knowledge that the great majority of these babies are born alive and well. Moreover, the pursuance of a deliberate policy of non-intervention in foetal distress is not necessarily followed by disaster (Table II).

Nevertheless, the perinatal loss associated with meconium-stained liquor is three times greater than when the liquor is clear. This increased loss is to a great extent dependent on the presence of other complicating obstetrical factors (Table III), and herein lies the sinister reputation of foetal distress. Pre-eclamptic toxaemia, uterine intertia, and intrauterine infection are all responsible in some measure for the increased loss of babies—a finding confirmed by Hellman *et al.* (1958)—but, in this series at least, cephalo-pelvic disproportion is the major lethal factor, and there can be no real doubt that caesarean section is the treatment of choice when foetal distress develops in the presence of disproportion, unless it is of such minor degree that cervical dilatation and descent of the presenting part are proceeding satisfactorily.

It is interesting, at this stage, to recall that when von Winckel (1903–7) originally described the syndrome of foetal distress, cephalo-pelvic disproportion was as common in Europe as it is in South Africa to-day.

Uncomplicated foetal distress, on the other hand, is a relatively benign condition, but nevertheless the foetal loss is greater than when no distress is present.

The incidence of foetal distress in this study was 5.5%, and of these babies 4.5% died when left to deliver spontaneously in the absence of other complications. In other words, 55 babies in every 1,000 deliveries showed signs of foetal distress during labour, and of these, two failed to survive. It follows, therefore, that the problem of foetal loss from uncomplicated foetal distress is a minor one when applied to an obstetric unit as a whole. Nevertheless, the problem is one of great personal concern to the individual mother whose child develops distress during the course of labour, and, as such, will prove a source of anxiety to her obstetrician because he has no reliable means of determining which 4 babies in every 100 at risk will succumb. The accepted signs are of very little help in assessing the outcome for the foetus.

The question then arises: Is it justifiable to consider performing 100 caesarean sections for the sake of saving four babies when uncomplicated foetal distress arises during the course of the first stage of labour? Many obstetricians will nevertheless decide to operate, particularly if the patient is an elderly primigravida or a relatively infertile woman. Caesarean section, however, offers no guarantee that a live child will result. In this series of 167 caesarean sections (Table V) there was a perinatal loss of 8.4%, and even when the cases of disproportion were excluded four babies were lost in 79 deliveries, an incidence of 5% (Table XI). This experience accords with that of Dumoulin and Martin (1957), who reported a perinatal loss of 7.9% in 107 caesarean sections performed for foetal distress in three teaching hospitals in Great Britain. In other words, the perinatal loss in caesarean section performed for foetal distress is much the same as for spontaneous vaginal delivery in the absence of complication. Nevertheless the birth of a live child is not necessarily the hallmark of good obstetrics, and evidence is gradually accumulating that a child subject to hypoxia during labour or in the immediate neonatal period may bear the stigmata of this deprivation for life. Inquiry into the birth history of children showing evidence of mental retardation or cerebral palsy has revealed a story of hypoxia in a significant proportion of cases.

In this series, 43 (11.5%) of the 374 infants who were delivered spontaneously per vaginam required some form of resuscitation at birth apart from routine aspiration of the air passages. Similarly, 20 infants (12%) of the 167 delivered by caesarean section required resuscitation, and in 200 normal deliveries without foetal

distress 10 infants were in need of resuscitation. These figures indicate that a considerable number of infants showing signs of distress prior to delivery are subject to hypoxia both during labour and in the immediate neonatal period. The risks attendant upon hypoxia at birth have not been fully established, because as yet all investigations have been retrospective and in consequence fail to take cognizance of the large number of children who develop into normal adults despite such hypoxia. Indeed, Higgins (1958) believes that neonates are particularly tolerant of anoxia.

Furthermore, it is by no means certain that the passage of meconium and foetal bradycardia are invariably the result of hypoxia. Though James Walker (1959) has shown that meconium is passed when the oxygen saturation of the blood of the umbilical vein falls to 30 vols.%, Macafee and Bancroft-Livingston (1958) state that there are factors other than placental insufficiency which cause the passage of meconium.

Clinical experience has shown that hypoxia in the human foetus will invariably produce bradycardia—the response of the foetal heart when pressure is applied to the umbilical cord is proof of this—but is all foetal bradycardia caused by hypoxia ?

It has been shown (Table III) that the onset of foetal bradycardia during labour is not necessarily associated with an increased stillbirth rate, no matter whether the liquor amnii is clear or meconium-stained. In view of these findings it would seem reasonable to postulate that some factor other than hypoxia is often the cause of foetal bradycardia during labour.

In the physiological bradycardia which accompanies normal uterine action, the slowing of the heart begins a few seconds after the onset of the uterine contraction. In other words, it starts before there could possibly be time for the foetus to develop hypoxia, even if it be assumed that the foetal oxygen supply is cut off completely the instant the contraction starts. This factor suggests that hypoxia is not the primary cause of physiological bradycardia.

Prystowsky (1957) indicates that the idea that each contraction squeezes blood out of the intervillous spaces is incorrect. He finds that during a contraction the intervillous spaces contain an increased quantity of blood which is 50–60% saturated with oxygen. This is another point which contradicts the idea that physiological bradycardia is attributable to hypoxia.

If the skull of the newborn infant is gently compressed and the heart auscultated at the same time, it will be found that the heart rate falls rapidly, and if the pressure is continued irregularities develop.

If moderate pressure on the newborn skull can produce this effect it is reasonable to suppose that a similar effect may be produced by the moulding of the foetal head through a tight pelvis, the pressure of a firmly applied cervix, or by the pressure of the sacral promontory against the parietal bone as the head finds its way through a flattened pelvis. It is possible, therefore, that most, if not all, of these 184 cases of prenatal bradycardia (Table VIII) were in reality the result of pressure on the foetal skull and did not represent the pathological bradycardia of true hypoxic foetal distress.

These investigations have borne out Louw's (1955) contention that foetal distress is a poor indication for caesarean section because it is one that is apt to be abused. The onset of distress during labour should be the signal, not for dramatic action, but for thoughtful reassessment and a search for a cause, particularly cephalo-pelvic disproportion.

Summary and Conclusions

Seven hundred cases of foetal distress have been studied, including 350 cases which formed the basis of a controlled investigation in which a policy of non-intervention was contrasted with the conventional approach of paying heed and offering treatment when the signs of foetal distress become manifest.

Meconium staining of the liquor amnii is the one definite sign of foetal distress.

The behaviour of the foetal heart is of doubtful value in assessing the condition of the foetus *in utero*, provided that the umbilical circulation is not occluded.

The prognosis in foetal distress is not as gloomy as is generally believed.

The sinister reputation of foetal distress depends upon the presence of underlying complications: the onset of distress should therefore be an indication to search for the cause rather than a demand for dramatic action.

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In the province of British Columbia, while all racial groups showed a marked decline in mortality from tuberculosis, the most spectacular decline was among the Indians. In 1948 the death rate for this group was 557.1 per 100,000 (156 deaths), in 1957, 47.6 per 100,000 (17 deaths), and, in 1958, 24.1 per 100,000 (9 deaths). (*Province of British Columbia, Division of Tuberculosis Control, Department of Health and Welfare, Annual Report for 1958.*)