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THE EARLY DIAGNOSIS OF FOETAL DISTRESS

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THE problem of reducing perinatal mortality is closely related to the early and accurate recognition of foetal distress. Although the estimation of urinary oestrogen and/or pregnanediol before labour, and the continuous recording of the foetal heart rate during labour are of some value, the problem is far from solved. In this paper we present the results obtained with two less-known methods for the early diagnosis of foetal distress: the first is used before the onset of labour and before rupture of the membranes; the second is used after rupture of the membranes, and usually during labour.

(1) BEFORE RUPTURE OF THE MEMBRANES AND BEFORE THE ONSET OF LABOUR

Methods

Threats to foetal life may be anticipated by observing meconium in the amniotic fluid either by transabdominal amniocentesis or by amnioscopy. We have recently described our technique of amniocentesis (Kubli, 1962). Amnioscopy, which was introduced by Saling (1962a), can be used to determine the colour of the forewaters by inspecting them through intact membranes with an endoscope introduced into the cervical canal. This procedure can be carried out so long as the cervix admits one finger. Otherwise liquor amnii is obtained by amniocentesis, and we prefer this method before the 38th week of pregnancy, or when the results of amnioscopy are equivocal. The indications for using these procedures were: abnormal foetal heart rate before labour, suspected intra-uterine death and all maternal conditions frequently associated with placental insufficiency, such as toxaemia (as defined by Dieckmann, 1952), hypertension (blood-pressure 140/90 mm. Hg. or more), postmaturity (10 days past the expected date of delivery), maternal diabetes and prediabetes, and elderly primigravidae.

Special precautions must be observed when amniocentesis is performed in rhesus negative women (Kubli, 1965), and in such patients we therefore restrict its use to cases of suspected haemolytic disease.

Results

In the last three years amniocentesis has been performed 293 times in 257 women, and amnioscopy was used 163 times in 101 women. In 309 patients the final inspection of the amniotic fluid was carried out during the week before delivery, We have not observed any foetal or maternal complications following amniocentesis or amnioscopy.

Table I shows the appearance of the amniotic fluid in 309 women in the week before delivery in relation to the condition of the babies at birth. With clear liquor the perinatal mortality rate was 0·4 per cent (one intra-uterine death at 31 weeks), whereas when the amniotic fluid was stained with meconium it was 21·7 per cent (15 of 69 babies). But some of the babies were already dead at the time of the examination; if they are excluded, the perinatal mortality rate with clear liquor was nil, whereas with meconium-stained amniotic fluid it was 6·9 per cent. Neonatal asphyxia (an Apgar score of six or less) occurred in only 9 of 239 (3·8 per cent) live-born babies when the amniotic fluid was clear, compared with 16 of 57 (28·1 per cent) when the liquor was meconium-stained. In other words when the liquor was clear 230 of 240 (95·8 per cent) babies were vigorous at birth, compared with only 41 of 69 (59·4 per cent) when it was meconium-stained at the time of the last examination before the onset of labour.

TABLE I

Appearance of the Amniotic Fluid in the Week Before Delivery and the Condition of the Babies at Birth in 309 Cases

Appearance of the Amniotic Fluid at Amniocentesis or Amnioscopy	Condition of the Newborn				
	Vigorous	Asphyxiated (Apgar Score ≤6)		Stillborn	
		Survivors	Neonatal Death	Dead After Examination	Dead Before Examination
Clear 240	230 (95·8%)	9	—	—	1
Meconium-stained 69	41 (59·4%)	13	3	1	11

(2) AFTER RUPTURE OF THE MEMBRANES

Methods

The metabolic state of the foetus can be directly evaluated by analyzing small quantities of capillary blood taken from it. The technique of obtaining capillary blood from the presenting part of the foetus was described by Saling (1962b), and has been reported by Huntingford (1964). Blood can be obtained after rupture of the membranes before or during labour so long as the cervix is at least 1 cm. dilated. The presenting part of the foetus is examined through an endoscope, and, after making the skin hyperaemic, capillary blood is aspirated in a polyvinyl chloride tube under almost completely anaerobic conditions from a small stab incision 1 to 2 mm. deep. The acid-base balance is evaluated by using micro-methods of analysis (Astrup *et al.*, 1960; Siggaard-Andersen *et al.*, 1960). We are not concerned here with measurements of Po_2 and Pco_2, which are subject to error inherent in the methods of sampling and storing discussed in detail elsewhere (Kubli and Berg, 1964). The estimation of *p*H is, however, not significantly influenced by these factors. The accuracy (±two standard deviations) we obtained for clinical purposes for the actual *p*H was ±0·006 *p*H units, and for the equilibrated *p*H ±0·02 *p*H units. Using values for the equilibrated *p*H the errors for base excess and standard bicarbonate were estimated at ±1·2 and 1·0 mEq per litre respectively.

The indications for assessing the foetal acid-base balance included the maternal conditions that justified amnioscopy, or clinical evidence of foetal distress, i.e., the passage of meconium and alterations of the foetal heart rate.

Results

An analysis of the foetal acid-base balance was carried out 100 times in 62 cases. In 24 cases the pregnancy, delivery and condition of the baby at birth were normal throughout. In 10 cases there was some abnormal maternal condition, but no evidence of foetal distress or neonatal asphyxia. There was clinical evidence of foetal distress in 26 cases; in four of them the babies were asphyxiated at birth. Finally, there were two cases of intra-uterine asphyxia without any clinical evidence of foetal distress; one of these babies died during the first stage of labour.

During this study only orthodox classical indications determined intervention in the

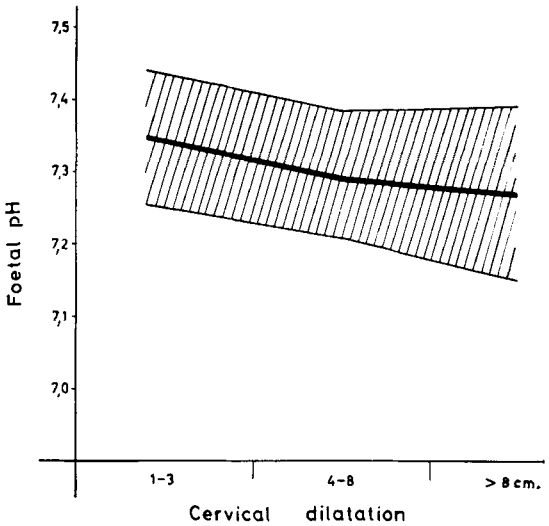


FIG. 1

Changes in foetal *p*H during labour in 24 normal cases. The bold line is the mean of 43 estimations; the shaded area includes two standard deviations of the mean.

TABLE II

Changes in Foetal Base Excess and Standard Bicarbonate During Labour in 24 Normal Cases

No. of Cases	Dilatation of the Cervix	Base Excess mEq/l Mean±2s	Standard Bicarbonate mEq/l Mean±2s
11	1–3 cm.	−3·9±5·7	20·9±4·2
12	4–8 cm.	−5·6±3·3	19·6±2·3
20	>8 cm.	−7·0±5·7	18·6±4·7

course of labour, i.e., decisions were based on the clinical evidence of foetal distress and not on the results of blood estimations.

The changes of *p*H during labour in the 24 normal cases are illustrated in Fig. 1. The mean *p*H fell from 7·35 at the onset of labour to 7·27 during the second stage of labour; the lower limits of normal were 7·25 and 7·15 respectively. The corresponding values for standard bicarbonate and base excess are shown in Table II.

The differences observed between the values at the onset of labour and those during the second stage were statistically significant for *p*H ($p<0·005$), standard bicarbonate ($p<0·02$), and base excess ($p<0·02$).

The results in the second group with various abnormal maternal conditions but with no evidence of foetal distress or neonatal asphyxia did not differ significantly from the normal cases.

The values for foetal base excess in the group

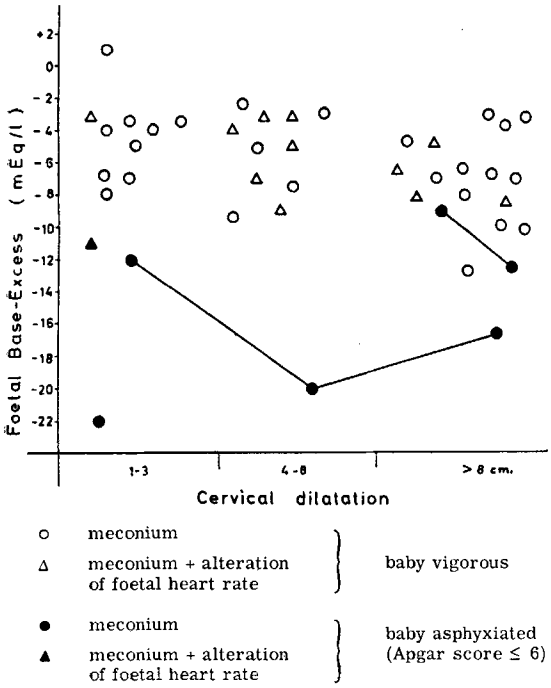


FIG. 2

Clinical signs of foetal distress and foetal base excess (44 analyses) in 26 cases.

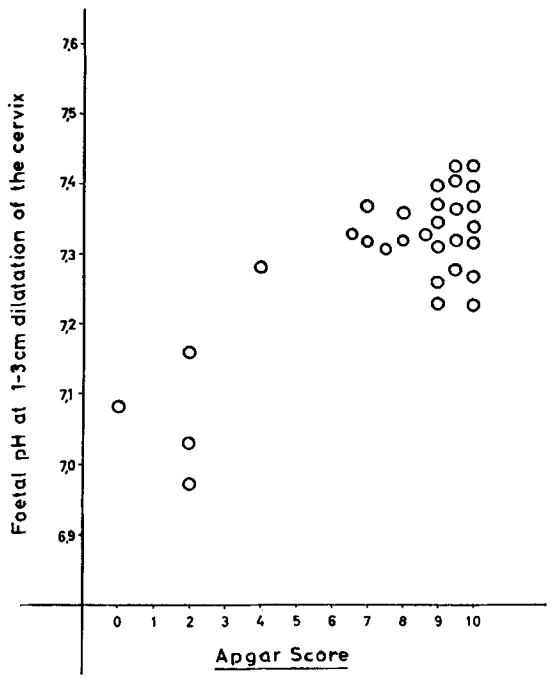


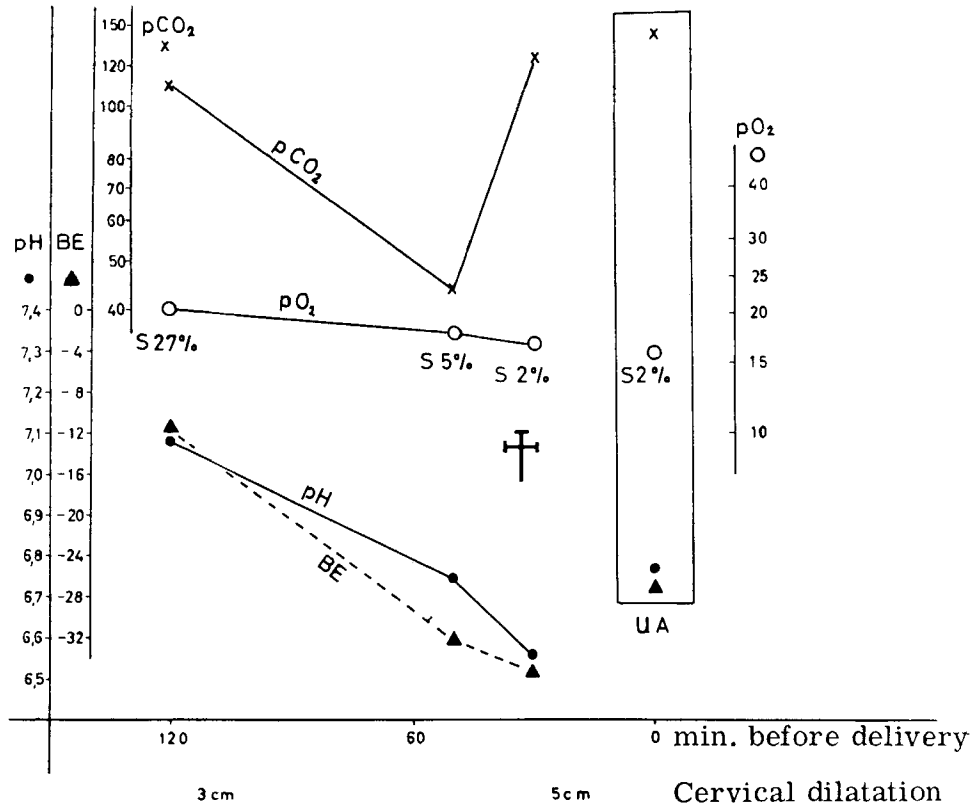
FIG. 3

Foetal *p*H in 30 cases before the onset of labour or in early labour compared with the Apgar score of the newborn. The difference in *p*H between the group with an Apgar score of 6 or less and the group with a score of 7+ is statistically significant ($p<0·001$).

of 26 cases with clinical evidence of foetal distress are shown in Fig. 2. No attempt has been made to correlate these observations with changes in the foetal heart rate as the number of cases was small and a continuous phonocardiographic recording was only available in a few of them. With the exception of a single estimation the base excess of the asphyxiated babies was below the lower limit of normal.

The accuracy of this method for early diagnosis of foetal distress is demonstrated in Fig. 3. Early in labour, when the cervix was 1 to 3 cm. dilated, the *p*H of foetuses that were eventually born in asphyxiated state was significantly lower than that of the control group ($p<0{\cdot}001$).

One foetus died during the first stage of labour. As the liquor amnii remained clear and there was no alteration in the foetal heart rate before death there was no clinical indication for operative delivery. Labour had been induced at 38 weeks by a continuous intravenous infusion of oxytocin, because of maternal pyrexia and a leucocytosis, the membranes having spontaneously ruptured two days previously. Although the same strain of streptococcus was found in the uterine cavity, and in the pharynx and



S oxygen saturation (calculated)

moment of foetal death

UA umbilical artery

FIG. 4

Foetal *p*H, base excess, P_{CO_2}, P_{O_2}, and oxygen saturation in a case of intrapartum foetal death without clinical signs of foetal distress.

cardiac blood of the baby, conclusive evidence that the baby died from an intra-uterine infection was lacking since there were no histological lesions in the placenta or umbilical cord. Neither was any other cause for intra-uterine death found. The changes in *p*H, base excess, P_{CO_2} and P_{O_2} (measured polarographically using the method described by Thews, 1962) preceding the death of this foetus are shown in Fig. 4. The extremely low values of *p*H and base excess are remarkable, whereas the P_{O_2} in this case was not markedly abnormal.

Discussion

Meconium-staining of the amniotic fluid is not a very reliable sign of intra-uterine asphyxia. In large series the mortality rate associated with this sign has varied between 5 and 10 per cent (Hellman *et al.*, 1958; Macafee and Bancroft-Livingston, 1958; Eastman, 1961), and the morbidity rate is about 20 per cent (Desmond *et al.*, 1956). Assessment of the colour of the amniotic fluid before the membranes are ruptured cannot, therefore, be regarded as an accurate method of diagnosis, but rather as a means of screening for cases at risk. Our results demonstrate that the foetal risk is indoubtedly increased, if the amniotic fluid is meconium-stained in the days preceding the onset of labour. Detection of meconium-staining of the amniotic fluid before labour therefore provides an indication for terminating the pregnancy, if the foetus is viable.

The diagnostic value of other changes in the amniotic fluid, such as carbon dioxide tension (Schreiner, 1964) and the level of lactic acid (Wood *et al.*, 1963), in relation to the foetal condition have yet to be confirmed.

The work of James *et al.* (1958) has demonstrated that the most reliable index of neonatal asphyxia is the degree of acidosis. The acid-base balance of capillary blood from the presenting part of the foetus seems to be closely related to the state of the foetus *in utero*. Although there may be errors in these estimations due to circulatory disturbances in the presenting part caused by the formation of the caput succedaneum and pressure differences, which are most pronounced during the second stage of labour, nevertheless, with few exceptions the values in capillary blood obtained a few minutes before delivery correspond very closely to those in the umbilical artery (Kubli and Berg, 1964).

The statistically significant decrease in foetal *p*H and buffer capacity during labour may, at least in part, be accounted for by maternal acidosis (Vedra, 1960; Derom, 1963; Rooth, 1964). However, we do not think that this accounts for the pathological foetal acidosis accompanying foetal distress; and this opinion is supported by the work of Derom (1963).

The assessment of foetal acid-base balance would seem to be of greatest value (Fig. 3) at the beginning of labour, or even before the onset of labour, since it is obvious that the chances of the foetus surviving are much greater if it begins labour with sufficient buffer reserves. There are of course some cases in which although the state of the foetus is good at the beginning it deteriorates during protracted labour, e.g., umbilical cord complications and foetal haemorrhage. If clinical evidence of foetal distress subsequently appears during labour, in spite of normal values at the onset, this provides an indication for re-examination of the foetal acid-base balance.

In principle our results confirm those of Saling (1964). Although the number of our cases is small we feel so strongly that this method provides the most reliable means for detecting foetal distress that we now make considerable use of it in the clinical management of our cases, particularly as a guide to when operative delivery is indicated.

Summary

The amniotic fluid was examined for meconium-staining either by amniocentesis or by amnioscopy within one week of delivery in 309 women. In 240 patients with clear liquor 230 of the babies were born in good condition (Apgar score 7+), whereas in 69 with meconium-stained liquor only 41 of the babies were vigorous at birth.

Micro-analyses of capillary blood obtained from the presenting part of the foetus after rupture of the membranes were performed 100 times in 62 cases before or during labour. The results were closely related to the condition of the babies at birth. This procedure provides one of the most reliable methods for the early diagnosis of foetal distress.

References

Astrup, P., Jørgensen, K., Siggaard-Andersen, O., Engel, K. (1960): *Lancet*, **i**, 1035.

Derom, R. (1963): *in De intrauterine hypoxie*, Arscia Uitgaven, Brussels.

Desmond, M. M., Lindley, J. E., Moore, J., and Brown, C. A. (1956): *J. Pediat.*, **49**, 540.

Dieckmann, W. J. (1952): *in Toxemias in Pregnancy*, Mosby, Comp., St. Louis.

Eastman, N. J. (1961): *Obstet. gynec. Surv.*, **16**, 72.

Hellman, L. M., Schiffer, M. A., Kohl, S. G., and Tolles, W. E. (1958): *Amer. J. Obstet. Gynec.*, **76**, 998.

Huntingford, P. J. (1964): *Lancet*, **i**, 95.

James, L. S., Weisbrot, I. M., Prince, C. E., Holaday, D. A., and Apgar, V. (1958): *J. Pediat.*, **52**, 379.

Kubli, F. (1962): *Geburtsh. u. Frauenheilk.*, **22**, 134.

Kubli, F. (1965): *Bibl. haemat. (Basel)*, **20**, 287.

Kubli, F., and Berg, D. (1964): *in Proceedings of the Symposium on "Effect of Labour on the Fetus and the Newborn"*. Montevideo, Oct. 1964, Pergamon Press, Oxford.

Macafee, C. H. G., and Bancroft-Livingston, G. H. (1958): *J. Obstet. Gynaec. Brit. Emp.*, **65**, 7.

Rooth, G. (1964): *Lancet*, **i**, 290.

Saling, E. (1962a): *Geburtsh. u. Frauenheilk.*, **22**, 831.

Saling, E. (1962b): *Arch. Gynäk.*, **197**, 108.

Saling, E. (1964): *Zschr. Geburtsh. Gynäk.*, **162**, 56.

Schreiner, W. E. (1964): *in Fruchtwasser und Fetus*, Karger, Basel/New York.

Siggaard-Andersen, O., Engel, K., Jørgensen, K., and Astrup, P. (1960): *Scand. J. clin. Lab. Invest.*, **12**, 172.

Thews, G. (1962): *Pflügers Arch. ges. Physiol.*, **276**, 89.

Vedra, B. (1960): *Biol. Neonat. (Basel)*, **2**, 121.

Wood, C., Acharya, P. T., Cornwell, E., and Pinkerton, J. H. M. (1963): *J. Obstet. Gynaec. Brit. Cwlth.*, **70**, 274.