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Foetal acid–base status in clinical foetal distress and high risk cases*

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Summary

The acid–base balance in 307 Chinese patients in labour was studied in Tsan Yuk Hospital, Hong Kong over a 15-month period. Fifty-six of these were normal cases, 200 cases exhibited clinical signs of foetal distress and the other fifty-one were high risk cases. The results showed that cases of clinical foetal distress had a significantly lower foetal pH than that in the normal control group. Of the different signs of clinical foetal distress, meconium was the least important and foetal tachycardia appeared to be more significant than the others. Cases of postmaturity and uterine inertia also gave a lower foetal pH than normal. It is concluded that foetal blood sampling is indicated in these types of cases.

THE conventional clinical signs of foetal distress, namely the presence of meconium in the liquor amnii and changes in the foetal heart rate, have been shown to be not sufficiently reliable for the accurate assessment of the foetus (Wood & Pinkerton, 1961; Day, Maddern & Beard, 1968; Farr, 1970). Foetal blood sampling provides direct information about the foetal acid–base balance and gives a more accurate picture of the state of the foetus (Saling, 1966a). Foetal scalp blood has been found to be suitable for this purpose (Beard & Morris, 1965; Kubli & Berg, 1966; Gare, Whetham & Henry, 1967). Good correlation has been established between the foetal pH and the condition of the infant at birth (Beard, Morris & Clayton, 1967; Bowe *et al.*, 1970).

This paper presents the results of a study of foetal blood sampling in Chinese patients with clinical foetal distress and in high risk cases undertaken in Hong Kong.

Material

From January 1969 to March 1970, foetal blood sampling was performed in 307 Chinese patients among 7166 deliveries at Tsan Yuk Hospital, Hong Kong, an incidence of 4·3%. These comprised fifty-six normal cases, 200 cases exhibiting clinical signs of foetal distress and fifty-one high risk cases as shown in Table 1.

The number of samples taken from each case ranged from one to five. A total of 387 foetal blood samples were obtained. In 149 of these, only the pH was determined. In the other 238 samples equilibration with carbon dioxide was carried out.

TABLE 1. Indications for foetal blood sampling

Normal control	56
Clinical foetal distress	
Meconium alone	168
Foetal tachycardia	5
Foetal bradycardia	10
Meconium and abnormal foetal heart rate	17
Total	200
High risk cases	
Toxaemia	14
Uterine inertia	13
Elderly primigravida	12
Postmaturity	7
Prolonged second stage	2
Diabetes mellitus	2
Foetal growth retardation	1
Total	51

The technique of obtaining foetal blood samples followed that of Morris & Beard (1965), but heparinized glass capillaries were used. The acid–base determinations were performed with the Astrup apparatus. Statistical comparison of the mean values was calculated using 'Student's' *t*-test.

Foetal acidosis was defined as a pH value lower than 7·20 (Lee, 1971).

Results

Normal labour

From the fifty-six cases of normal labour which served as control, the mean foetal pH in the Chinese was found to be $7{\cdot}30 \pm 0{\cdot}04$. The other values are shown in Table 2. The details of this group of normal cases have been reported separately (Lee, 1971).

Clinical foetal distress

The mean foetal pH of the 200 cases with clinical signs of foetal distress was $7{\cdot}28 \pm 0{\cdot}06$ which was

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TABLE 2. Mean acid-base values and incidence of foetal acidosis in cases with clinical foetal distress

	pH	pHqu40	P_{CO_2} (mmHg)	Standard bicarbonate (mEq/l)	Base deficit (mEq/l)	Buffer base (mEq/l)	Foetal acidosis (%)
Meconium alone	7·28 ±0·05	7·33 ±0·05	49·9 ±10·2	20·5 ±2·2	4·7 ±3·1	44·3 ±4·1	4·2
Tachycardia	7·24 ±0·07	7·26 ±0·09	51·8 ±11·2	17·4 ±3·5	9·5 ±4·9	41·5 ±9·8	40·0
Bradycardia	7·24 ±0·07	7·27 ±0·08	48·0 ±6·4	18·0 ±3·2	8·1 ±4·6	40·9 ±5·2	10·0
Meconium and abnormal foetal heart rate	7·26 ±0·05	7·31 ±0·05	50·5 ±6·9	19·6 ±2·3	5·8 ±3·1	42·8 ±5·2	25·5
Normal control	7·30 ±0·04	7·33 ±0·04	47·5 ±5·9	20·6 ±1·8	4·6 ±2·5	44·6 ±2·6	0

significantly lower than that of the normal cases ($P < 0·005$).

Foetal acidosis was detected in fourteen cases, an incidence of 7·0%. The clinical sign in seven of these cases was the presence of meconium alone; two cases had foetal tachycardia, one case had bradycardia and four cases were associated with meconium and an abnormal foetal heart rate.

When the cases are divided according to the different clinical signs of foetal distress, the acid–base values of the different groups together with the incidence of foetal acidosis are shown in Table 2. In every group the mean foetal pH was significantly lower than that in the normal control group.

Comparison among the four groups themselves shows that cases with presence of meconium alone had the highest mean foetal pH (7·28) and the lowest incidence of foetal acidosis (4·2%). The acid–base values were least favourable in cases with tachycardia and this group was also associated with the highest incidence of foetal acidosis (40·0%).

All the 200 infants were delivered alive. Two died neonatally, one on the first day after a difficult breech delivery and the other died on the eighth day because of intracranial haemorrhage. The perinatal mortality rate was therefore one in 200 or 5·0 per thousand.

High risk cases

In the fifty-one high risk cases without clinical foetal distress, the mean foetal pH was 7·28±0·05, pHqu40 7·33, P_{CO_2} 49·3 mmHg, standard bicarbonate 20·5 mEq/l, base deficit 4·8 mEq/l and buffer base 45·2 mEq/l. There was no significant difference between these values and those of the normal control group.

Foetal acidosis was detected in three cases (5·9%). There was no stillbirth or neonatal death among the fifty-one cases. One infant died 45 days after birth because of exomphalos.

When this group was divided according to the individual complication, the foetal pH in cases of postmaturity and uterine inertia (7·25 and 7·27 respectively) was found to be significantly lower than that of the control group ($P < 0·01$ and 0·05 respectively). The foetal acid-base values in the elderly primigravidae and toxaemic patients were similar to those of the normal cases.

Discussion

The incidence of foetal acidosis (pH less than 7·20) in cases of clinical foetal distress varied widely in reported series as shown in Table 3 (from 6·8% to 29·6%). In the present series of 200 cases 7% had a foetal pH of less than 7.20, a figure similar to those obtained by Coltart, Trickey & Beard (1969) and Saling & Schneider (1967).

There is still considerable controversy regarding which of the clinical signs of foetal distress or a combination of them is superior to the others for the detection of foetal asphyxia.

Retrospective studies on foetal distress indicate that no one clinical sign is superior to the others (Walker, 1959; Wood & Pinkerton, 1961; Fenton & Steer, 1965). These findings have been confirmed by Beard *et al.* (1967) who conclude that no single clinical sign of foetal distress or combination of them is clearly superior to another for diagnostic purposes,

TABLE 3. Reported incidences of foetal acidosis among cases with clinical foetal distress

Author	No. of cases with clinical foetal distress	No. of cases with foetal pH less than 7·20	Incidence (%)
Coltart *et al.* (1969)	295	20	6·8
Saling & Schneider (1967)	850	61	7·2
Wood *et al.* (1967)	67	11	16·4
Garud *et al.* (1969)	53	11	20·8
Beard *et al.* (1967)	176	52	29·6
Present series	200	14	7·0

irrespective of whether the Apgar score or acidosis is used as an index of foetal asphyxia. However, they have noted that more low scores and more low pH babies are found in association with passage of meconium and a foetal heart rate of below 100 beats/min than with any other clinical sign of foetal distress.

Foetal bradycardia has always been regarded as an ominous sign of foetal distress while foetal tachycardia is thought to be less serious. However, recent investigations have stressed the importance of foetal tachycardia as a sign of foetal anoxia. Beard (1968) reported thirty-seven cases with foetal pH 7·25 or less and found that foetal tachycardia was present in 49% whereas bradycardia was only present in 30%. He stressed the importance of foetal tachycardia as a sign of foetal asphyxia and exposed the extent to which bradycardia had been over-emphasized as a sign of foetal distress at the expense of foetal tachycardia. In the series of Coltart *et al.* (1969), among forty-five cases of foetal acidosis thirty-three presented with foetal tachycardia.

The present study confirmed the clinical impression that the presence of meconium alone was the least important sign. This group gave the highest mean foetal pH and the lowest incidence of foetal acidosis. Tachycardia did appear to be a more significant sign than the others, but the difference was not significant because of the small number of cases.

The high risk cases in the present series, mainly postmaturity, uterine inertia, toxaemia and elderly primigravida, were considered to carry an increased likelihood of placental insufficiency and foetal anoxia especially during labour. However, taken as a whole group, these cases gave normal acid–base values, which concurred with the results of Kubli & Berg (1965).

If the different complications were considered separately, a significantly lower foetal pH was obtained in cases of postmaturity and uterine inertia. The results in postmature cases were contrary to those of Paterson *et al.* (1970) who found no difference in pH, Po_2 and Pco_2 between postmature cases and the control. A lower foetal pH in cases of uterine inertia was not unexpected as progressive retraction of the uterus would jeopardize the placental circulation.

Toxaemic patients and elderly primigravidae had normal foetal pH during labour when there was no clinical sign of foetal distress. Similar results were previously reported by Saling (1966b).

The results obtained in the present study confirmed that foetal blood sampling should be performed in cases of clinical foetal distress, of postmaturity in labour and of uterine inertia in order to obtain an accurate and early diagnosis of foetal hypoxia.

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