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Recognition and significance of maternogenic fetal acidosis during intensive monitoring of labor

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The rapid propagation of techniques for intensive monitoring of the fetus-at-risk during labor, i. e. fetal heart rate (FHR) monitoring and fetal blood sampling (FBS), has resulted in the accumulation of considerable clinical experience in this area within a short period of time.

The problem of choosing between these two methods for routine use now seems to have been solved; it has become clear that the two are mutually complementary [3, 9, 14, 15, 31, 34, 42]. In general, the purpose of FBS and fetal pH determination is to reduce the margin of error inherent in assessing the fetal condition by means of the cardiometer tracing. The clinical and acid-base status of the infant at birth may in fact be good, not only when the FHR has remained normal during labor (regular, or with early deceleration), but also in a high percentage of cases showing abnormal or even highly abnormal deceleration (late deceleration) [3, 12, 22, 27, 31, 34, 42].

Monitoring of FHR allows for continuous assessment of the fetal status during labor, whereas microanalysis of pH from FBS is at present still intermittent. In practical terms this means that both methods may be employed to advantage: FHR monitoring should be used for preliminary screening of all cases at risk, and microanalysis of the fetal blood as a guide to the obstetric management of labor once pathological changes in the FHR have been detected. If clinical management is to be based on the actual condition of the fetus, it becomes imperative to obtain an exact picture

of the fetal acid-base balance in appropriate cases. The main problem in this regard is one of false positives or falsely abnormal results, i. e., the cases (by no means rare) in which a low fetal pH during labor does not reflect a state of fetal hypoxia [2, 6, 20, 27, 31]. The good clinical condition of these infants at birth (APGAR ≥ 7) indicates that rapid extraction of the fetus is not justified in such cases. A number of methods have been proposed for the recognition of this type of acidosis during labor, but certain questions regarding the validity of these methods and their ability to meet the particular clinical requirements of the delivery room [2, 19, 26, 28, 31] are still unanswered. The data presented below were compiled at the First Clinic of Obstetrics and Gynecology of the Milan University during the period 1968—1972.

1 Cases and methods

59 normal cases (normal pregnancy, normal onset of labor at term, clear amniotic fluid, regular FHR, spontaneous delivery, APGAR 8—10 at 90 seconds, weight at birth $> 2,500$ g), and 335 cases regarded as being at risk (maternal disease, presence of meconium stained amniotic fluid and/or abnormalities in FHR) were followed during labor by monitoring of FHR and microanalysis of fetal blood.

1.1 Monitoring of the FHR and uterine contractions

Biophysical monitoring was carried out by means of a HEWLETT-PACKARD 8020 A cardiotokograph.

The FHR was taken from the fetal ECG recorded with a silver suction electrode applied to the scalp [40], and more recently with a HON clip [16] or spiral [17] electrode. Uterine contractions were recorded with an abdominal lead, or when possible, with a transvaginal intra-amniotic catheter. The tracings were interpreted according to HON [16].

1.2 Determination of acid-base balance

Microsamples of fetal blood were obtained by the method of SALING [30]. Simultaneous samples of the mother's blood were taken from the tip of the finger after arterialisation with ethyl chloride and smearing with vaseline. Both samples were subjected to identical chemical analysis, and all measurements were carried out in duplicate.

pH and pH qu 40 were measured with a SALING-TUROWSKI pH meter [30].

Hemoglobin concentration was determined with a BECKMAN spectrophotometer, after the addition of DRABKIN's solution.

Hemoglobin-O₂-saturation (%) was measured photometrically using an OSM 1 RADIOMETER Oxygen Saturation Meter [1, 39].

Base deficit (BD) and **Base deficit_{Hb5} (BD_{Hb5})** were calculated from the SIGGAARD-ANDERSEN nomogram [35] using the pH qu40 value and the hemoglobin concentration; BD values were corrected for hemoglobin-O₂-saturation. The P_{CO₂} value was indirectly derived, and the BD_{Hb5} calculated at the predetermined Hb level of 5 g/100 ml [37]. **Materno-fetal (M/F) Δ BD_{Hb5}** (the difference between the fetal and maternal base deficits at a fixed Hb level of 5 g/100 ml) was calculated by subtracting the corrected fetal from the maternal base deficit [19].

M/F actual Δ pH, M/F Δ pHqu 40 are the materno-fetal differences in actual pH (26) and in pHqu 40 [31] respectively. Samples of maternal blood and of arterial and venous cord blood were taken immediately after delivery and subjected to identical microanalyses. The clinical condition of the infant 90 seconds after birth was evaluated by the APGAR score.

2 'Falsely abnormal' cases and maternogenic fetal acidosis

Fetal pH values obtained shortly before delivery normally compare well with those of blood from the umbilical artery, and with the clinical condition of the infant at birth [6]. In some cases, however, the fetal pH during labor does not agree with the clinical condition at birth. A low pH (< 7.20) in these instances leads one to expect the delivery of a distressed infant, whereas the APGAR score proves to be ≥ 7 [2, 6, 20, 27, 31].

The overall incidence of such "falsely abnormal" cases — false because the infant is in good condi-

Tab. I. Incidence of 'falsely abnormal' cases (fetal pH < 7.20 within 30 minutes of delivery; APGAR score 1—2 minutes after birth ≥ 7) in 4 series.

Author	No. of cases	'Falsely abnormal' (%)
BEARD R. W. et al. [5]	176	13.1
KUBLI F. [21]	77	12.9
BOWE E. T. et al. [6]	355	7.6
Present series*	179	11.2

* Cases at risk

tion at birth, and abnormal because the fetal acid-base state during labor was found to be abnormal [20] — is nearly identical in all reported series [5, 6, 21]; in our own series they comprised 11.2% [29] of the cases at risk (Tab. I). In the absence of measures to detect these cases, rapid extraction of the fetus will be erroneously indicated in at least one out of every 10 cases at risk.

As a first step in solving this problem it is necessary to determine the cause of this type of fetal acidosis without associated hypoxia.

Investigations both in human subjects and in animals have shown that artificially induced acidosis or alkalosis in the mother causes a shift of the fetal acid-base balance in the same direction [8, 10, 13, 24]. The detailed mechanism of this dependence and the part played by the placenta are far from clear, but in normal labor an increase in the content of fixed acid radicals in the mother (mainly due to the uterine contractions) is accompanied by a similar increase in the fetus [11, 19]. This does not normally lead to any significant decrease in fetal pH, but the latter may be considerably decreased in cases where uterine contractions are particularly strong (spontaneous or iatrogenic hyperkinesia) and/or the clinical condition of the mother is such as to produce severe metabolic acidosis (prolonged labor, toxemia, diabetes, inadequate nutrition and care during labor, drugs, etc.). This secondary fetal acidosis does not appear to be harmful to the fetus, and according to some reports [7, 23] it could actually favour oxygenation.

The following parameters have been proposed to distinguish fetal hypoxic acidosis from

maternogenic acidosis and to ensure that the measurement of fetal pH during labor maintains its diagnostic and prognostic value: F/M Δ BD [2], M/F Δ pH_{qu 40} [31], M/F actual Δ pH [26], and M/F Δ BD_{Hb5} [19, 28].

3 Methods for the diagnosis of maternogenic fetal acidosis

3.1 Difference between fetal and maternal BD (F/M Δ BD)

On the basis of the pathophysiologic observations outlined above it appears that hypoxic fetal acidosis may be distinguished from acidosis of maternal origin by comparison of the metabolic component of the acid-base balance in the mother and in the fetus. In maternogenic fetal acidosis the relationship between the two should be normal, since the fetal acidosis is merely a reflection in the fetus of the metabolic acidosis in the mother. In fetal hypoxic acidosis on the other hand, there should be a change in the relationship owing to the accumulation of lactic acid in the fetus as a result of anaerobic metabolism [2, 19]. BEARD et al. [2] have shown that when the BD (not corrected for the O₂ saturation of the hemoglobin) is used as a parameter for the measurement of the metabolic component of the acid-base balance, the fetal values are normally slightly higher than the maternal values (F/M Δ BD = 2.0–3.0 mEq/l). This value has therefore been proposed as the boundary between the two forms of acidosis, and would be exceeded only in fetal hypoxic acidosis.

Difficulties may arise in the use of the BD for the differential diagnosis of fetal acidosis unless it is corrected for the O₂ saturation of the hemoglobin, for the following reasons:

(a) The uncorrected fetal BD is higher than the true value

The BD is read directly from the SIGGAARD-ANDERSEN nomogram [35], after construction of the buffer line by equilibration of the blood sample with gas mixtures containing O₂. During the equilibration the fetal Hb in the reduced state becomes oxygenated, and the greater acidity of oxygenated Hb in comparison with reduced Hb causes an increase in the in vitro BD in comparison with the in vivo value*. This error is invariably of considerable degree, owing to the

* According to SIGGAARD-ANDERSEN's definition of base deficit, the BD would be calculated at the actual hemoglobin oxygen saturation [38].

Fig. 1. Calculation of BD according to fetal Hb oxygen saturation: Actual pH 7.20, pH_{qu 40} 7.24, Hb 15 g/100 ml, O₂ saturation 33.3%

First line — uncorrected BD (10 mEq/l)

Second line — corrected BD (7 mEq/l)

high percentage of reduced Hb present in fetal blood. Particular variation may exist from case to case, since it depends (see the formula of SIGGAARD-ANDERSEN and ENGEL [38]) on two extremely variable factors, i. e. the amount of Hb present and its percentage oxygen saturation. Even with normal fetal values for Hb (15–20 g/100 ml) and for the oxygen saturation (20–50%), the error caused by ignoring correction for oxygenation amounts to an increase in BD varying between 2.2–4.8 mEq/l (Fig. 1).

(b) The fetal BD is higher than the maternal value

In view of the false increase in fetal BD, the materno-fetal BD difference (M/F Δ BD) is negative in normal labor, i. e. the fetal BD is higher than the maternal.

Analysis of the metabolic components of maternal and fetal acid-base balance shows [19] that the maternal BD is actually higher than the fetal value in vivo (see below).

3.2 Differences between maternal and fetal pH_{qu 40} (M/F Δ pH_{qu 40})

The pH_{qu 40} [30] is the pH of the blood after equilibration with CO₂ at a partial pressure of 40 mm Hg, and corresponds to the standard bicarbonate. It has the advantage of expressing the metabolic component of the acid-base balance in terms of pH.

SALING [31] has proposed the simultaneous determination of maternal and fetal pH_{qu 40} for the diagnosis of fetal acidosis of maternal origin. By his definition a fetal acidosis of maternal origin would be restricted to a difference of not greater

than 0.05 pH units between the maternal and the fetal values (M/F Δ pHqu 40), this limiting value having been established empirically [31].

The pHqu 40 is a parameter that can be determined quickly and easily, but the technique requires complete oxygenation of the blood sample. In the SIGGAARD-ANDERSEN nomogram, the buffer line giving the BD for fully oxygenated blood passes through the pHqu 40 value (Fig. 1). The use of the pHqu 40 is therefore open to the same criticism as is BD (see foregoing paragraph).

3.3 Differences between the actual maternal and fetal pH (M/F actual Δ pH)

The mean fetal pH determined during normal labor may vary considerably from one maternity unit to another [26]. In view of the close relationship between the maternal and the fetal acid-base balance this is probably due to variations in maternal metabolic acidosis during labor in different Units.

To overcome this difficulty, and to enable all maternity units to express the fetal acid-base condition in units that are truly comparable, it has been suggested that actual fetal pH should be replaced by difference in maternal and fetal actual pH (M/F actual Δ pH) [26]. **Fetal pH-normality, pre-acidosis, and frank acidosis would thus be redefined and characterized respectively by an M/F actual Δ pH of < 0.15, M/F actual Δ pH of between 0.15–0.19, and M/F actual Δ pH $\geq$ 0.20.** This classification is based on the close correlation found between M/F actual Δ pH and fetal P_{O_2} [26]. The use of M/F actual Δ pH instead of the actual pH in the evaluation of the fetal condition during labor would clearly also overcome the problem of differential diagnosis between fetal acidosis due to hypoxia and that of maternal origin.

A preliminary analysis suffices to show that it would be unwise to use the difference in pH between mother and fetus for the diagnosis of fetal hypoxia, or to thusly estimate its degree of severity. The same concentration of hydrogen ions may be expressed by very different Δ pH values, and vice versa, since pH is logarithmic. In Tab. II, for example, the same hydrogen ion concentration (23.2–23.3 nmol/l) corresponds both to an M/F actual Δ pH of 0.20 (actual pH: M 7.40; F 7.20),

Tab. II. Disagreement between Δ pH and Δ [H⁺]. Continuous lines enclose different Δ pH values for identical Δ [H⁺]; broken lines enclose identical Δ pH values for different Δ [H⁺] values.

pH M	pH F	Δ pH (M/F)	Δ [H ⁺] nmol/l
7.40	7.30	0.10	10.3
7.30	7.20	0.10	13.0
7.20	7.10	0.10	16.3
7.40	7.25	0.15	16.4
7.30	7.15	0.15	20.7
7.25	7.10	0.15	23.2
7.40	7.20	0.20	23.3
7.30	7.10	0.20	29.3

Tab. III. Correlations: M/F actual Δ pH/fetal P_{O_2} and M/F actual Δ pH/fetal Hb-O₂ saturation.

Author	No. of cases	M/F actual Δ pH/ P_{O_2} F		M/F actual Δ pH/ sat. O ₂ F	
		r	p	r	p
ROOTH G. [26]	31	~0.68	<0.001	0.72	<0.001
Present series	327	0.02		0.28	<0.001

~ = calculated from graphically reported data [26].

and to an M/F actual Δ pH of 0.15 (actual pH: M 7.25; F 7.10).

Moreover, a retrospective study of our own series had failed to confirm any close correlation between M/F actual Δ pH and fetal scalp blood P_{O_2} (Tab. III).

For all these reasons it is our opinion that **the clinical use of this parameter does not provide an accurate evaluation of the fetal acid-base balance during labor, and does not solve the problem of recognizing 'falsely abnormal' cases.**

3.4 Materno-fetal difference in base deficit of the extracellular fluid (M/F Δ BD_{EBF})

3.4.1 Base deficit of the extracellular fluid (BD_{EBF})*

As indicated above, any accurate comparison of the metabolic component of the maternal and fetal acid-base balance with the BD as the parameter necessitates correction of the BD for O₂

* In this paper the term BD_{EBF} is used in order to reflect the way the data are obtained. However, the modern terminology prefers to use the term BD_{ECF} (base deficit of the extracellular fluid).

hemoglobin saturation (Fig. 1). This correction may be performed by means of the formula of SIGGAARD-ANDERSEN and ENGEL [38].

However, another correction is necessary to eliminate the false increase in the BD that occurs in vivo with hypercapnia, which is almost a normal occurrence in the fetus during labor. In cases involving hypercapnia the increased CO_2 comes out of the intracellular compartment and enters the red cells, where bicarbonate is generated. This bicarbonate from the red cells diffuses out of the blood into the interstitial fluid. The intravascular

Fig. 2. Model showing bicarbonate redistribution in vivo in a case of hypercapnia. Buf^- = all buffers other than bicarbonate. For every Buf^- consumed, one bicarbonate ion is produced, some of which "leaks" into the interstitial fluid. Base deficit therefore increases [41].

Fig. 3. Calculation of fetal BD at the prefixed Hb value of 5 g/100 ml, O_2 saturation 33.3%

First line — uncorrected BD (10 mEq/l)

Second line — corrected BD (7 mEq/l)

Third line — BD for Hb 5 g/100 ml (5 mEq/l)

space therefore loses bicarbonate and blood BD increases, even though there is no real increase in the degree of metabolic acidosis present (Fig. 2) [41].

To prevent variations in P_{CO_2} from being reflected in the BD, i. e. to ensure that the BD value includes bicarbonate that has diffused from the blood into the interstitial fluid, SIGGAARD-ANDERSEN proposes using the BD of the extracellular fluid [37]. This is calculated on the basis of the amount of red cells distributed throughout the interstitial space, at a Hb concentration of 5 g/100 ml (BD_{Hb5}) (Fig. 3).

It has been shown experimentally that in both maternal and fetal blood the BD_{Hb5} is independent of the P_{CO_2} , and that the values obtained from the SIGGAARD-ANDERSEN nomogram are identical with those calculated on the basis of pH, P_{CO_2} , plasma protein, and Hb concentration [25].

3.4.2 M/F $\Delta\text{BD}_{\text{Hb5}}$ in normal labor

Workers in Sweden were the first to use the BD_{Hb5} for the evaluation of the acid-base balance during labor and the M/F $\Delta\text{BD}_{\text{Hb5}}$ for the detection of 'falsely abnormal' cases [19, 25]. JACOBSON has shown that in normal labor [19] the fetal BD_{Hb5} remains lower than the maternal value, particularly during expulsion.

Probability analyses on the results of this study [28] show that the mean M/F $\Delta\text{BD}_{\text{Hb5}}$ during labor is $+2.5 \text{ mEq/l} \pm 2.6 \text{ (SD)}$. This was confirmed in our own series of normal cases, the criteria adopted being the same as those used by the Swedish workers. Our mean was $+4.0 \text{ mEq/l} \pm 2.8 \text{ (SD)}$.

3.4.3 Detection of 'falsely abnormal' cases by means of the M/F $\Delta\text{BD}_{\text{Hb5}}$

It appeared desirable to confirm that this method could be used for the detection of maternogenic fetal acidosis in "falsely abnormal" cases. For this purpose it was assumed that a positive M/F $\Delta\text{BD}_{\text{Hb5}}$ in the presence of a low pH still represents normal placental function, and hence indicates that the acidosis is of maternal origin, since a value of zero for M/F $\Delta\text{BD}_{\text{Hb5}}$ equals $1\frac{1}{2} \text{ SD}$ (4.0—4.2 mEq/l) downwards from normal cases.

Tab. IV. Incidence of maternogenic fetal acidosis in "falsely abnormal" cases (actual pH < 7.25 during final 30 minutes of labor, APGAR ≥ 7 at 90 seconds after delivery).

Actual fetal pH	M/F Δ pHqu 40 ≤ 0.05	M/F Δ BD _{Hb5} > 0
<7.25 (39 cases)	35.9%	79.4%
7.25—7.20 (19 cases)	52.6%	84.2%
<7.20 (20 cases)	20.0%	75.0%

In a series of 179 cases at risk, the M/F Δ BD_{Hb5} was positive in 79.4% of 'falsely abnormal' cases (actual pH < 7.25, APGAR ≥ 7).

When the series was further delineated according to the degree of acidosis, the M/F Δ BD_{Hb5} remained positive in 84% of the cases of pre-acidosis (actual pH 7.25—7.20) and in 75% of the cases of frank acidosis (actual pH < 7.20) (Tab. IV).

The decrease in fetal pH in most of the "falsely abnormal" cases in our series was therefore due to increased metabolic acidosis in the mother. If the M/F Δ pHqu 40 had been used, on the other hand, the maternal origin of the fetal acidosis would have been recognised in considerably fewer cases; it was in fact less than 0.05 units in 35.9% of fetuses with an actual pH < 7.25, in 52.6% of those with actual pH values between 7.25 and 7.20, and in 20% of those with an actual pH < 7.20 (Tab. IV). The difference in the ability of the two criteria to identify maternal origin of fetal acidosis is not without clinical importance, since a decision

Tab. V. Incidence of maternogenic fetal metabolic acidosis during labor and after delivery.

Author	No. of cases	Parameter	First stage	2nd stage	Delivery
<hr/>					
SALING E. [31] Present series	118	<u>Fetal pHqu 40 < 7,25</u>			
		M/F Δ pHqu 40	18.0%	26.0%	31.0%
	101	M/F Δ pHqu 40	8.5%	28.0%	55.0%
		M/F Δ BD _{Hb5}	64.4%	66.7%	90.1%
<hr/>					
Present series	139	<u>Fetal actual pH < 7,25</u>			
		M/F Δ pHqu 40	31.9%	15.8%	59.7%
		M/F Δ BD _{Hb5}	75.3%	68.4%	88.5%

as to the future management of labor depends on the diagnosis.

It has recently been reported [31, 33] that when the M/F Δ pHqu 40 is used as the indicator, the incidence of maternogenic fetal metabolic acidosis is 18% and 26% during the first and second stages of labor respectively. In our own series the same parameter gave 8.5% in the first stage and 28% in the second, whereas the M/F Δ BD_{Hb5} gave a much higher incidence (64.4% and 66.7% respectively). The incidence is even higher in cases with actual pH < 7.25 (Tab. V).

3.4.4 Correlation between M/F Δ BD_{Hb5} and Δ pHqu 40

The different results obtained when the M/F Δ pHqu 40 and the M/F Δ BD_{Hb5} are used for the identification of "falsely abnormal" cases are at variance with the good statistical correlation found between the two parameters. The coefficient of correlation in a recent series was high ($r = 0.98$) [33], and good correlation was also found ($r = 0.80$) [28] in our own series (578 microsamples from 303 cases of labor) (Fig. 4).

Fig. 4. Correlation between M/F Δ pHqu 40 and Δ BD_{Hb5} during labor for cases at risk

No. of cases: 303

No. of blood microsamples: 578

Fig. 5. Correlation between M/F Δ pHqu 40 and Δ BD_{Hb5} in fetal acidosis (actual fetal pH < 7.25), and its clinical implications (see text)

No. of cases: 87
No. of samples: 117

If a system of axes with the ordinate at 0.05 pH units and the abscissa at a Δ BD_{Hb5} value of 0 (limiting values of the two parameters for distinguishing hypoxic and maternogenic fetal acidoses) are inserted in the context of the correlation between Δ BD_{Hb5} and Δ pHqu 40 (fetuses with actual pH < 7.25), two points of interest emerge. Firstly, in our own experience there are no negative values for M/F Δ BD_{Hb5} when the M/F Δ pHqu 40 is less than 0.05. Secondly, the Δ BD_{Hb5} may have a positive value even when the Δ pHqu 40 is greater than 0.05 (Fig. 5).

In clinical terms this means that in fetal acidosis, a M/F Δ pHqu 40 $\leq$ 0.05 pretty well rules out fetal hypoxia as the cause [28, 33], whereas a value greater than 0.05 does not necessarily rule out a maternal etiology [28].

3.4.5 Prognostic value of M/F Δ BD_{Hb5} in the presence of variable FHR during labor

The diagnostic and prognostic value of the M/F Δ BD_{Hb5} during labor was pointed out in 1970 [27]. In cases where the FHR varied during the 90 minutes preceding delivery, the M/F Δ BD_{Hb5} proved to be negative in every case where the subsequent APGAR score was $\leq$ 4, and positive when it was $\geq$ 8. Intermediate clinical

Fig. 6. M/F Δ BD_{Hb5} in cases of variable and late decelerations, subdivided according to APGAR score 90 seconds after birth

+ : M/F Δ BD_{Hb5} $\geq$ 0
- : M/F Δ BD_{Hb5} < 0

scores (APGAR 5—7) were found to occur with both positive and negative values (Fig. 6). Further evidence that fetal acidosis is due to hypoxia when the M/F Δ BD_{Hb5} is negative is provided by the distribution of the latter values in relation to Hb oxygen saturation in fetuses presenting with marked bradycardia. If cases of late deceleration in our own series are classified by degree of Hb oxygen saturation, the M/F Δ BD_{Hb5} is invariably positive whenever oxygen saturation is within normal limits. Negative values are encountered only in the presence of fetal hypoxia (O₂ saturation less than 1 SD of normal cases) (Tab. VI).

Tab. VI. Correlation between M/F Δ BD_{Hb5} and fetal Hb oxygen saturation in cases with late decelerations.

	Late deceleration		Normal labor
	Sat. O ₂ < 29.2**	Sat. O ₂ $\geq$ 29.2**	Sat. O ₂ 40.1 $\pm$ 10.9*
No. of cases	22	15	54
M/F Δ BD _{Hb5}	0.8 $\pm$ 3.1* (10)	4.1 $\pm$ 2.3* (0)	4.0 $\pm$ 2.8* (0)
NS			
p < 0.001			

In brackets: number of cases with M/F Δ BD_{Hb5} < 0 (see text).
* mean value $\pm$ SD
** 29.2 is the mean value of Hb oxygen saturation —1 SD in normal cases
NS = not significant

3.4.6 Technical observations

There have been doubts in the past regarding the clinical applicability of a parameter such as the $M/F \Delta BD_{Hb_5}$, since the test takes longer than the exigencies of the delivery room allow [32].

If the buffer line is drawn on the SIGGAARD-ANDERSEN nomogram by the double equilibrium technique [35] or by the SALING method [30] (pH_{qu} 40 and Hb), it is in fact necessary to correct the BD for Hb oxygen saturation [38]. Having thus obtained the actual P_{CO_2} , it is then necessary to read off the BD data at the predetermined Hb level of 5 g/100 ml (Fig. 3). The procedure is considerably simplified and hence much faster (sufficiently fast for use in the delivery room) if reliable values for the actual pH and the P_{CO_2} can be obtained directly. The BD_{Hb_5} of both mother and fetus can then be read off instantly from the SIGGAARD-ANDERSEN nomogram (36) (Fig. 7).

4 Clinical observations

The fact that "falsely abnormal" cases do occur necessitates further diagnostic measures to establish the true condition of the fetus. Whenever the FHR is abnormal and the pH indicates acidosis, steps must be taken to ascertain the cause (i. e. hypoxic or maternogenic) of the acidosis.

It should be emphasized that this differentiation must be as precise as possible, since it represents the final stage of diagnosis and consequently is not subject to subsequent check. Thus, it directly determines the further obstetric management of labor. **The safest of the various parameters suggested so far appears to be the $M/F \Delta BD_{Hb_5}$.** The most obvious clinical result of using this instead of the other parameters is the **marked**

Summary

FHR monitoring and microanalysis of fetal blood are mutually complementary procedures, and optimal knowledge of the fetal state is achieved by making use of both, the former for the preliminary screening of all cases at risk and the latter for the purpose of deciding on obstetric management where pathological changes are evident in the FHR.

The major difficulty in obtaining a precise value for the fetal acid-base balance lies in the occurrence of "falsely abnormal" cases, i. e. cases in which the fetal pH falls during labor but the clinical condition at birth is good ($APGAR \geq 7$). In our own series the incidence of such cases among fetuses at risk was 11.2% (Tab. I).

Fig. 7. Calculation of BD_{Hb_5} from the SIGGAARD-ANDERSEN nomogram. Actual pH = 7.20, P_{CO_2} = 58 mmHg, BD_{Hb_5} = 5 mEq/l

decrease in the number of erroneously diagnosed cases of fetal distress, particularly during the final stage of labor, and hence in the number of operative interventions. As has been said elsewhere [31], such a decrease is by no means to be excluded from the goals of perinatal medicine.

In the majority of these cases the fetal acidosis is thought to be a result of increased metabolic acidosis in the mother (maternogenic fetal metabolic acidosis).

The importance of the maternogenic fetal acidosis during labor lies in the fact that unless it is recognised, rapid extraction of the fetus will appear necessary on clinical grounds, although it is in fact unnecessary, since this form of acidosis has no adverse effect on the fetus.

Various parameters have been proposed for the differential diagnosis of the maternogenic fetal acidosis. These include the fetomaternal difference in base deficit ($F/M \Delta BD$), the materno-fetal differences in pH_{qu} 40 ($M/F \Delta pH_{qu} 40$), the materno-fetal difference actual pH (M/F actual ΔpH),

and the materno-fetal difference in base deficit of the extracellular fluid ($M/F \Delta BD_{Hb5}$).

A critical analysis of these parameters has been carried out on the results of microtests performed during a 5 year period (1968—1972) at the First Clinic of Obstetrics and Gynecology of Milan University. The cases comprised 59 regarded as normal (normal course of pregnancy, spontaneous commencement of labor at term, clear amniotic fluid, regular FHR, spontaneous birth, APGAR at 90 sec between 8 and 10, weight at birth > 2500 g), and 335 considered to be at risk (maternal disease, presence of meconium stained amniotic fluid and/or abnormal changes in FHR).

In all of these cases the FHR was recorded by cardiotokography, and the tracings were interpreted according to HON. Microsamples of blood were taken from both mother and fetus during labor and the following determinations were carried out: actual pH, pH_{qu} 40, Hb concentration, hemoglobin oxygen saturation, base deficit Hb 5 (BD_{Hb5}). The materno-fetal differences were then calculated. The same determinations were carried out on samples of maternal blood and of arterial and venous cord blood taken immediately after delivery.

The clinical condition of the infant was evaluated by the APGAR score at 90 seconds after birth.

Methods for the diagnosis of maternogenic fetal acidosis

1. Feto-maternal ΔBD : $F/M \Delta BD$

The use of this parameter poses certain problems, in that the BD is not corrected for the Hb oxygen saturation, and it cannot therefore be expected to provide an accurate comparison of the metabolic components of the maternal and fetal acid-base balance. The fetal BD appears higher (by 2.2—4.8 mEq/l for normal Hb and oxygen saturation levels of fetal blood) than the true value, and hence higher than the maternal value (Fig. 1).

2 Difference between maternal and fetal pH_{qu} 40 values: $M/F \Delta pH_{qu} 40$

According to SALING a materno-fetal difference of less than 0.05 pH units indicates that fetal acidosis during labor is of maternal origin. The parameter is quickly and easily obtainable, but has the same disadvantages as the $F/M \Delta BD$ (Fig. 1).

3 Difference between the actual pH values of mother and of fetus: $M/F \text{ actual } \Delta pH$

The use of the $M/F \text{ actual } \Delta pH$ has been suggested in order to overcome the difficulty arising from the fact that the mean fetal pH during labor varies from one maternity unit to another. The substitution of the $M/F \text{ actual } \Delta pH$ for the fetal actual pH would provide a solution to the problem of recognizing maternogenic fetal metabolic acidosis. In practice, however, this does not seem to be possible, since pH is logarithmic, and the same concentration of hydrogen ions may therefore be expressed by very different ΔpH values and vice versa (Tab. II).

Moreover, close correlation between the $M/F \text{ actual } \Delta pH$ and the degree of fetal oxygenation as assessed

by fetal scalp blood P_{O_2} has not been confirmed (Tab. III).

4 Difference between maternal and fetal base deficits of the extracellular fluid: $M/F \Delta BD_{Hb5}$

Base deficit of the extracellular fluid (BD_{Hb5})

Any valid comparison between the metabolic components of the maternal and fetal acid-base balance requires not only correction for the hemoglobin O_2 -saturation but also subsequent correction of the false increase in BD encountered in vivo in hypercapnia, a condition that is almost the rule rather than the exception in the fetus during labor. The increase in the concentration of CO_2 , and hence of bicarbonate, in the peripheral blood causes the bicarbonate to move into the interstitial fluid, with a consequent increase in the BD of the peripheral blood (Fig. 2). In order to prevent variations in P_{CO_2} from being reflected in the BD, SIGGAARD-ANDERSEN has suggested using the BD of the extracellular fluid, which may be determined from the amount of red cells distributed throughout the interstitial space, i. e. at a hemoglobin concentration of 5 g/100 ml (BD_{Hb5}) (Fig. 3).

Materno-fetal ΔBD_{Hb5} in normal labor

According to JACOBSON the fetal BD_{Hb5} remains below the maternal value during normal labor, particularly during expulsion. Our own series confirmed this finding: the mean $M/F \Delta BD_{Hb5}$ during normal labor was $+ 4.0 \text{ mEq/l} \pm 2.8 \text{ (SD)}$.

Recognition of falsely abnormal cases by means of the $M/F \Delta BD_{Hb5}$

We have confirmed that a maternogenic etiology of fetal acidosis can be detected in "falsely abnormal" cases by the $M/F \Delta BD_{Hb5}$.

For this purpose a positive value of $M/F \Delta BD_{Hb5}$ in the presence of a low pH is regarded as indicating normal placental function, and hence pointing to the maternal origin of the acidosis. A value of zero corresponds approximately to the mean materno-fetal ΔBD_{Hb5} minus $1\frac{1}{2}$ SD ($4.0 - 4.2 \text{ mEq/l}$) for normal cases.

In a series of 179 cases at risk, the value of $M/F \Delta BD_{Hb5}$ was positive in 79.4% of the "falsely abnormal" cases (actual pH < 7.25, APGAR ≥ 7), including 84% of cases of pre-acidosis (actual pH 7.25—7.20) and 75% of cases of frank acidosis (pH < 7.20) (Tab. IV).

If instead, the materno-fetal $\Delta pH_{qu} 40$ parameter had been used, the maternal origin of the acidosis would have been recognised in a lower percentage of cases, i. e. 35.9% of fetuses having an actual pH < 7.25, 52.6% with a pH between 7.25 and 7.20, and 20% of those with actual pH < 7.20 (Tab. IV).

In our own series using the materno-fetal $\Delta pH_{qu} 40$ as the parameter, the incidence of maternogenic fetal metabolic acidosis during labor was 8.5% in the first stage and 28% in the second stage of labor. With the $M/F \Delta BD_{Hb5}$, on the other hand, the percentages increased to 64.4% and 66.7% respectively (Tab. V).

This difference in the ability of the two indices to allow recognition of maternogenic fetal acidosis is of consider-

able clinical importance, since this directly determines the future management of labor.

Correlation between materno-fetal ΔBD_{Hb5} and $\Delta pH_{qu 40}$

The differing results obtained with the two methods do not agree with the good statistical correlation between the parameters, which was high in the series recently reported: $r = 0.98$ [33] and $r = 0.80$ (Fig. 4). Further examination of the correlation shows, however, that a materno-fetal $\Delta pH_{qu 40} \leq 0.05$ rules out fetal hypoxia as the cause of the acidosis, whereas a value > 0.05 does not necessarily exclude a maternogenic etiology (Fig. 5).

Prognostic value of materno-fetal ΔBD_{Hb5} in the presence of changes in FHR during labor

In cases showing abnormal changes in the FHR (variable and late decelerations) during the 90 minutes preceding delivery, the materno-fetal ΔBD_{Hb5} was negative in all of our cases that subsequently were graded with ≤ 4 APGAR score, and positive in those that were scored ≥ 8 . The cases in which the APGAR score range 5–7 included both positive and negative values (Fig. 6). If the late decelerations

are divided according to Hb oxygen saturation, it can be seen that when the latter is within normal limits the materno-fetal ΔBD_{Hb5} is positive. Negative values of $M/F \Delta BD_{Hb5}$ occur only in fetal hypoxia (Tab. VI).

Conclusions

The results described above appear to indicate that:

1. the metabolic component of fetal acid-base balance is reflected more accurately in the BD_{Hb5} than in $pH_{qu 40}$ or the uncorrected BD;
2. in the diagnosis of maternogenic fetal acidosis, the materno-fetal ΔBD_{Hb5} is a more reliable test than the $\Delta pH_{qu 40}$, despite the close statistical correlation between the two.

When the actual fetal pH during labor is found to be below, subsequent recognition of the fetal acidosis as being maternal in origin allows for a decision against rapid extraction of the fetus. Any decrease in the number of cases requiring intervention will increase with the accuracy of the parameter used to compare the metabolic components of maternal and fetal acid-base balance.

Keywords: Acid-base balance, base-deficit Hb5 (BD_{Hb5}), bradycardia, ΔBD_{Hb5} (M/F), $\Delta pH_{qu 40}$ (M/F), ΔpH_{act} (M/F), fetal-blood-analysis, fetus, heart-rate-monitoring, labor, maternogenic fetal acidosis, pH.

Zusammenfassung

Erkennung und Bedeutung der maternogenen fetalen Azidose während der Intensivüberwachung der Geburt.

Die Überwachung des fetalen Herzschlages und die Fetalblut-Analyse sind sich ergänzende Überwachungsmethoden; ein optimaler Einblick in die augenblickliche Situation des Feten wird durch den Einsatz beider Methoden ermöglicht: die erstere für ein vorläufiges Herausfiltern aller Risikofälle und die letztere, um geburtshilfliche Entscheidungen herbeizuführen, wenn pathologische Veränderungen der fetalen Herzschlagfrequenz auftreten.

Die Hauptschwierigkeit bei der Beurteilung des fetalen Säure-Basen-Haushaltes liegt im Auftreten von „falsch pathologischen“ Fällen, d. h. von Fällen, in denen der pH-Wert während der Geburt absinkt, der klinische Zustand bei der Geburt jedoch gut ist ($APGAR \geq 7$). In unserer eigenen Untersuchungsreihe war die Häufigkeit solcher Fälle unter den Risikofällen 11,2% (Tab. I).

In der Mehrzahl dieser Fälle kann man davon ausgehen, daß die fetale Azidose Folge einer erhöhten metabolischen Azidität der Mutter ist (maternogene fetale metabolische Azidose).

Die Bedeutung der maternogenen fetalen Azidose während der Geburt beruht auf der Tatsache, daß, wenn sie nicht erkannt wird, eine schnelle Entwicklung des Feten aufgrund von klinischen Hinweisen notwendig erscheint, obwohl diese rasche Entwicklung in Wirklichkeit nicht notwendig ist, weil diese Form der Azidose keinen schädlichen Einfluß auf den Feten hat.

Es wurden verschiedene Parameter vorgeschlagen für die Differentialdiagnose der maternogenen fetalen Azidose; diese schließen ein: die feto-maternale Differenz hinsichtlich des Basen-Defizits ($F/M \Delta BD$), materno-fetale Unterschiede im pH_{qu40} ($M/F \Delta pH_{qu40}$), materno-fetale Differenz des aktuellen pH ($M/F \Delta pH_{act}$) und die materno-fetale Differenz hinsichtlich des Basen-Defizits der extrazellulären Flüssigkeit ($M/F \Delta BD_{Hb5}$).

Eine kritische Analyse dieser Parameter wurde anhand der Ergebnisse von Mikroblutuntersuchungen im Verlauf der Jahre 1968–1972 an der Ersten Geburtshilflichen und Gynäkologischen Klinik an der Universität Mailand durchgeführt. Die Fälle umfaßten 59 normale Geburten (normaler Schwangerschaftsverlauf, spontaner Wehenbeginn am Termin, klares Fruchtwasser, unauffällige fetale Herzschlagfrequenz, Spontangeburt, APGAR nach 90 Sekunden zwischen 8 und 10, Geburtsgewicht über 2500 g) und 335 Risikogeburten (mütterliche Erkrankungen, grünes Fruchtwasser und/oder abnorme Herzschlagfrequenz-Kurven).

In allen diesen Fällen wurde die Herzschlagfrequenz durch Kardiotokographie überwacht, und die Herzschlagfrequenzmuster wurden in Anlehnung an HON bewertet. Mikroblutproben wurden von Mutter und Fet während der Geburt entnommen; die folgenden Bestimmungen wurden durchgeführt: pH_{act} , pH_{qu40} , Hb, Sauerstoffsättigung des Hb, Basen-Defizit Hb5 (BD_{Hb5}). Es wurden dann die materno-fetalen Differenzen berechnet. Die gleichen Bestimmungen wurden im mütterlichen Blut und im Blut von Nabelschnurarterie und -vene unmittelbar nach der Geburt durchgeführt.

Der klinische Zustand der Kinder wurde entsprechend dem APGAR-Score 90 Sekunden nach der Geburt bewertet.

Methoden für die Diagnose der maternogenen fetalen Azidose.

1 Der fetoto-maternale ΔBD : F/M ΔBD

Die Verwendung dieses Parameters ist insofern problematisch, als der BD nicht auf die reale Sauerstoffsättigung des Hämoglobin korrigiert ist und demzufolge auch nicht erwartet werden kann, daß er einen präzisen Vergleich der metabolischen Komponenten des mütterlichen und fetalen Säure-Basenhaushalts ermöglicht. Der fetale BD (Basendefizit) liegt scheinbar höher (um 2,2 bis 4,8 mÄq/l bei normalem Hämoglobingehalt und normaler Sauerstoffsättigung des fetalen Blutes) als der wirkliche Wert und demzufolge auch höher als der mütterliche Wert (Fig. 1).

2 Unterschiede zwischen mütterlichen und fetalen pH_{Qu 40}-Werten: M/F $\Delta pH_{Qu 40}$

Nach SALING bedeutet eine materno-fetale Differenz von weniger als 0,05 pH-Einheiten, daß die fetale, subpartuale Azidose maternogenen Ursprungs ist. Der Parameter kann schnell und leicht bestimmt werden, hat jedoch dieselben Nachteile wie der F/M ΔBD (Fig. 1).

3 Unterschied zwischen den aktuellen pH-Werten der Mutter und dem Feten: M/F ΔpH_{akt}

Um die Schwierigkeit zu überwinden, daß das mittlere fetale pH sub partu von einem Klinikum zum anderen Schwankungen zeigt, wurde der M/F ΔpH_{akt} eingeführt und verwendet.

Es war zu vermuten, daß der Austausch des aktuellen fetalen pH gegen den M/F ΔpH_{akt} eine Lösung des Problems der Erkennung der maternogenen fetalen metabolischen Azidose mit sich bringen würde. Dies scheint in praxi jedoch nicht möglich zu sein, da der pH-Wert eine logarithmische Größe ist und gleiche Wasserstoffionenkonzentrationen demzufolge durch sehr unterschiedliche ΔpH -Werte dargestellt werden können und vice versa (Tab. II). Darüberhinaus konnte keine gute Korrelation zwischen dem M/F $\Delta pH_{aktuell}$ und dem Grad der fetalen Oxygenation gesichert werden (Tab. III).

4 Unterschied zwischen mütterlichem und fetalem Basendefizit im extrazellulären Raum: ΔBD_{Hb_5}

Das Basendefizit im extrazellulären Raum (BD_{Hb_5})

Jeder brauchbare Vergleich zwischen dem metabolischen Komponenten des mütterlichen und fetalen Säure-Basenhaushalts schließt nicht nur die Korrektur auf die reale Sauerstoffsättigung des Hämoglobin ein, sondern auch anschließend die Korrektur auf die hyperkapniebedingte vorgetauschte Zunahme des BD in vivo — ein Zustand, der beim Feten unter der Geburt fast normalerweise gegeben ist. Die Zunahme der CO_2 -Konzentration und damit auch des Bikarbonat im peripheren Blut veranlaßt das Bikarbonat, in den interstitiellen Raum zu diffundieren, was einen Anstieg des BD im peripheren Blut zur Folge hat (Fig. 2). Um den BD unabhängig von P_{CO_2} -Veränderungen zu

machen, schlug SIGGAARD-ANDERSEN das Basendefizit im extrazellulären Raum vor, das bestimmt werden kann, wenn man sich die roten Blutkörperchen über den gesamten interstitiellen Raum gleichmäßig verteilt denkt, d. h. bei einer Hämoglobinkonzentration von 5 g/100 ml (BD_{Hb_5}) (Fig. 3).

Mütterlich-fetale ΔBD_{Hb_5} -Werte bei normaler Wehentätigkeit

Nach JACOBSON bleiben die fetalen BD_{Hb_5} -Werte unter jenen der Mutter bei normaler Wehentätigkeit und besonders auch in der Austreibungsperiode. Unsere eigenen Untersuchungen bestätigen diesen Befund: der mittlere ΔBD_{Hb_5} bei normaler Wehentätigkeit lag bei $+ 4,0 \text{ mÄq/l} \pm 2,8 \text{ (SD)}$.

Erkennen von falsch abnormalen Fällen mit Hilfe des M/F ΔBD_{Hb_5}

Wir konnten zeigen, daß die mütterliche Ursache einer fetalen Azidose in falsch abnormalen Fällen mit Hilfe des M/F ΔBD_{Hb_5} aufgedeckt werden kann. Zweckmäßigerweise wird ein positiver ΔpH_{Hb_5} in Gegenwart eines tiefen (fetalen) pH-Wertes als Indikator für eine normale Plazenta-funktion und demzufolge als ein Hinweis für den mütterlichen Ursprung der Azidose angesehen. Ein Wert von Null entspricht ungefähr dem mittleren materno-fetalen $\Delta BD_{Hb_5} - 1\frac{1}{2} \text{ SD}$ ($4,0 \text{ mÄq/l} - 4,2$) bei Normalfällen. Bei 179 Risikofällen war der M/F ΔBD_{Hb_5} in 79,4% positiv bei den falsch abnormalen Fällen (aktuelles pH $< 7,25$, APGAR ≥ 7), in 84% der Fälle mit Präazidose (aktueller pH $7,25 - 7,20$) und in 75% der Fälle mit klarer Azidose (pH $< 7,20$) (Tab. IV).

Mit dem materno-fetalen $\Delta pH_{Qu 40}$ würde der mütterliche Ursprung der Azidose in einem geringeren Prozentsatz der Fälle diagnostiziert worden sein, nämlich bei 35,9% der Feten mit einem pH von $< 7,25$, in 52,6% mit einem pH-Wert zwischen 7,25 und 7,20 und in 20% bei jenen Feten mit aktuellen pH-Werten unter 7,20 (Tab. IV). Die Häufigkeit der maternogenen fetalen metabolischen Azidose sub partu lag mit der Meßgröße des materno-fetalen $\Delta pH_{Qu 40}$ bei unserem eigenen Material bei 8,5% in der Eröffnungsperiode und bei 28% in der Austreibungsperiode, mit dem Parameter M/F ΔBD_{Hb_5} dagegen stiegen diese Prozentsätze an auf 64,4% bzw. 66,7% (Tab. V). Diese Differenz der Eignung der beiden indices für die Erkennung einer maternogenen fetalen Azidose ist von gewisser klinischer Bedeutung, da diese die zukünftige Geburtsleitung direkt bestimmend beeinflusst.

Korrelation zwischen dem materno-fetalen ΔBD_{Hb_5} und $\Delta pH_{Qu 40}$

Die mit den beiden Methoden gewonnenen unterschiedlichen Resultate harmonisieren nicht mit den guten statistischen Korrelationen der beiden Parameter untereinander: der Koeffizient lag bei den kürzlich mitgeteilten Serien mit $r = 0,98$ (33) und $r = 0,80$ (Fig. 4) recht hoch. Eine eingehende Prüfung der Korrelationen zeigt jedoch, daß ein materno-fetaler $\Delta pH_{Qu 40} < 0,05$ ein fetale

Hypoxie als Azidoseursache anzeigt, daß jedoch ein Wert von $> 0,05$ eine maternogene Ursache nicht notwendigerweise ausschließt (Fig. 5).

Der prognostische Wert des materno-fetalen ΔBD_{Hb5} beim Vorliegen von fetalen Herzfrequenzalterationen sub partu

Bei jenen Fällen mit Herzfrequenzalterationen (variable und späte Dezelerationen während des 90-Minuten-Intervalles vor der Geburt) und mit einer APGAR-Zahl von ≤ 4 war der materno-fetale ΔBD_{Hb5} negativ, bei jenen Fällen mit APGAR ≥ 8 war er positiv. Fälle, die zwischen APGAR 5 und 7 rangierten, wiesen sowohl positive wie negative Werte auf (Fig. 6).

Trennt man die Fälle mit Spätdezelerationen auf hinsichtlich der Sauerstoffsättigung des fetalen Hämoglobins, so zeigt sich, daß der materno-fetale ΔBD_{Hb5} positiv ist in jenen Fällen mit diesbezüglich normalen Werten.

Negative Werte des M/F ΔBD_{Hb5} kommen nur bei fetaler Hypoxie vor (Tab. VI).

Schlüsselwörter: Basendefizit, Bradycardie, Dezeleration, Fetalblutanalyse, Fetus, Herzschlagfrequenz, maternogene fetale Azidose, Säure-Basen-Haushalt, Wehen.

Résumé

Diagnostic et signification de l'acidose métabolique foetale maternogénique dans le travail

Le contrôle FHR et la microanalyse du sang foetal sont des procédés réciproquement complémentaires qui permettent d'étudier au maximum l'état foetal, le premier en détectant tous les cas menacés et la deuxième pour décider du traitement obstétrical là où se manifestent des altérations pathologiques dans le FHR.

La difficulté majeure pour obtenir une valeur précise pour l'équilibre acidobasique foetal réside dans l'apparition de cas „faussement anormaux”, c. à. d. de cas où le pH foetal tombe durant le travail, mais où la condition clinique s'avère bonne à la naissance (APGAR ≥ 7). Dans nos propres séries d'observations, l'incidence de tels cas parmi les foetus menacés s'est chiffrée à 11,2% (Tab. I).

Dans la majorité de ces cas, l'acidose foetale est supposée résulter d'une acidose métabolique accrue chez la mère (acidose métabolique foetale maternogénique).

L'importance de cette acidose foetale maternogénique durant le travail réside dans le fait qu'à moins de la reconnaître, il paraît nécessaire de procéder à une extraction rapide du foetus pour des raisons cliniques bien que cela soit inutile en réalité puisque cette forme d'acidose n'a pas d'effet néfaste sur le foetus.

Des paramètres variés ont été proposés pour le diagnostic différentiel de l'acidose foetale maternogénique. Ils incluent la différence foeto-maternelle dans le déficit basique (M/F ΔBD), les différences materno-foetales dans le pHqu40 (M/F $\Delta pHqu40$), la différence materno-foetale pH actuel (M/F ΔpH_{actuel}), et la différence materno-foetale dans le déficit basique du liquide extracellulaire (M/F ΔBD_{Hb5}).

Une analyse critique de ces paramètres a été effectuée sur les résultats de microtests étagés sur une période de 5 ans (1968

Schlußfolgerungen

Die oben beschriebenen Resultate scheinen zu belegen, daß:

1. die metabolische Komponente im fetalen Säure-Basenhaushalt präziser durch den BD_{Hb5} beschrieben wird als durch den pHqu 40 oder den unkorrigierten Basenexzeß;
2. für die Diagnose einer maternogenen fetalen Azidose der maternofoetale ΔBD_{Hb5} eine zuverlässigere Testgröße ist als der $\Delta pHqu 40$ trotz der engen statistischen Korrelationen zwischen den beiden Größen.

Die Erkenntnis, daß beim Vorliegen eines tiefen aktuellen pH-Wertes unter der Geburt diese fetale Azidose maternogenen Ursprungs ist, ermöglicht die Entscheidung gegen eine rasche Extraktion des Feten. Mit der Zunahme der Genauigkeit jenes Parameters, der für den Vergleich der metabolischen Komponenten des maternalen und fetalen Säure-Basenhaushaltes zur Anwendung kommt, wird die Zahl der Fälle, die ein Eingreifen des Geburtshelfers nötig machen, abnehmen.

—1972) à la Première Clinique d'Obstétrique et de Gynécologie de l'Université de Milan. 59 de ces cas étaient considérés comme normaux (grossesse normale, déclenchement spontané du travail au terme prévu, liquide amniotique clair, FHR régulier, naissance spontanée, APGAR à 90 sec. entre 8 et 10, poids à la naissance > 2500 g) et 335 comme menacés (maladie maternelle, présence de liquide amniotique coloré de méconium et/ou changements anormaux dans le FHR).

Dans tous ces cas, le FHR a été enregistré par cardiotocographie, et les tracés ont été interprétés en HON. Des microspécimens de sang ont été prélevés sur la mère et le foetus en cours de travail avec les calculs suivants: pHactuel, pHqu40, concentration Hb, saturation d'oxygène de l'hémoglobine, déficit basique Hb5 (BD_{Hb5}). Les différences materno-foetales ont alors été calculées. Les mêmes déterminations ont été faites sur des spécimens de sang maternel et de sang ombilical artériel et veineux prélevés immédiatement après l'accouchement.

La condition clinique des nouveaux-nés a été évaluée par le score APGAR à 90 secondes après la naissance.

Méthodes pour le diagnostic de l'acidose foetale maternogénique dans le travail

1. ΔBD materno-foetal

L'emploi de ce paramètre pose certains problèmes du fait que le BD n'est pas corrigé pour la saturation d'oxygène de l'hémoglobine et qu'il ne peut pas, en conséquence, servir à comparer avec précision les facteurs métaboliques de l'équilibre acidobasique maternel et foetal. Le BD foetal paraît plus élevé (2,2 à 4,8 mEq/l pour Hb et niveaux de saturation d'oxygène normaux du sang foetal)

que la valeur véritable, et, par conséquent, plus élevée aussi que la valeur maternelle (Fig. 1).

2. Différence entre les valeurs pH_{qu 40} maternelles et foetales: M/F Δ pH_{qu 40}

Selon SALING, une différence materno-foetale inférieure à 0,05 unités pH indique que l'acidose foetale durant le travail est d'origine maternelle. Le paramètre peut être obtenu rapidement et facilement, mais présente les mêmes inconvénients que le M/F Δ BD (Fig. 1).

3. Différence entre les valeurs pH actuelles de la mère et du fœtus: M/F Δ pH actuel

Le recours au M/F Δ pH actuel a été suggéré pour remédier à la difficulté causée par le fait que le pH foetal moyen varie pendant le travail d'une maternité à l'autre. La substitution du M/F Δ pH actuel au pH actuel foetal fournirait une solution au problème du dépistage de l'acidose métabolique foetale maternogénique. En pratique néanmoins, cela ne semble pas possible, car le pH est logarithmique et, par conséquent, la même concentration de ions hydrogène peut être exprimée par des valeurs Δ pH très différentes et vice versa (Tab. II). De plus, il n'a pas été possible de confirmer une étroite corrélation entre le M/F Δ pH actuel et le degré d'oxygénation foetale (Tab. III).

4. Différence entre les déficits basiques maternels et foetaux du liquide extracellulaire: M/F Δ BD_{Hb5}

Déficit basique du liquide extracellulaire (BD_{Hb5})

Toute comparaison valable entre les facteurs métaboliques de l'équilibre acidobasique maternel et foetal requiert non seulement une correction pour la saturation d'oxygène de l'hémoglobine, mais aussi une correction subséquente du faux accroissement du BD rencontré in vivo dans l'hypercapnie, une condition qui est presque toujours la norme dans le fœtus pendant le travail. L'augmentation de la concentration de CO₂ et, par conséquent, de bicarbonate dans le sang périphérique provoque la pénétration du bicarbonate dans le liquide interstitiel, avec une hausse conséquente du BD du sang périphérique (Fig. 2). Afin d'éviter que les variations P_{CO2} ne se reflètent sur le BD, SIGGAARD-ANDERSEN a suggéré d'utiliser le BD du liquide extracellulaire, qui peut être déterminé à partir des cellules rouges réparties dans l'espace interstitiel, c. à d. pour une concentration de Hb de 5 g/100 ml (BD_{Hb5}) (Fig. 3).

Δ BD_{Hb5} materno-foetal dans le travail normal

Selon JACOBSON, le BD_{Hb5} foetal reste au-dessous de la valeur maternelle durant le travail normal, et en particulier durant l'expulsion ainsi que l'ont prouvé nos propres observations: le M/F Δ BD_{Hb5} moyen a été de + 4,0 mEq/l $\pm$ 2,8 DS durant le travail normal.

Dépistage de fausses anomalies au moyen du M/F Δ BD_{Hb5}

Nous avons confirmé que l'étiologie maternogénique de l'acidose foetale peut être dépistée dans les fausses anomalies par le M/F Δ BD_{Hb5}.

A cet effet, une valeur positive du M/F Δ BD_{Hb5} en présence d'un pH bas est considérée comme témoignage d'une fonction placentaire normale et, en conséquence, de l'origine maternelle de l'acidose. Une valeur de zéro correspond approximativement à la valeur moyenne du M/F Δ BD_{Hb5} —1½ DS (4,0 mEq/l—4,2) pour les cas normaux.

Dans un groupe de 179 cas à usque, la valeur du M/F Δ BD_{Hb5} a été positive dans 79,4% des fausses anomalies (pH actuel < 7,25, APGAR $\geq$ 7), compris 84% de cas de préacidose (pH actuel 7,25—7,20) et 75% de cas d'acidose nette (pH < 7,20) (Tab. IV).

Avec le Δ pH_{qu 40} materno-foetal, l'origine maternelle de l'acidose aurait été reconnue dans un pourcentage moins élevé de cas, c. à d. 35,9% des fœtus ayant un pH actuel < 7,25, 52,6% avec un pH entre 7,25 et 7,20, et 20% de ceux avec un pH actuel < 7,20 (Tab. IV).

Dans nos propres séries, avec le Δ pH_{qu 40} materno-foetal comme paramètre, l'incidence de l'acidose métabolique foetale maternogénique durant le travail a été de 8,5% dans la période de dilatation et de 28% dans la deuxième phase; avec le M/F Δ BD_{Hb5}, d'autre part, les pourcentages se sont élevés respectivement à 64,4% et 66,7% (Tab. V). La différence de capacité des deux indices pour le dépistage de l'acidose foetale maternogénique est de remarquable importance clinique, puisqu'elle détermine directement la conduite future du travail.

Corrélation entre le Δ BD_{Hb5} et le Δ pH_{qu 40} materno-foetal

Les résultats différents obtenus avec les deux méthodes ne concordent pas avec la bonne corrélation statistique entre ces paramètres, corrélation qui est très élevée dans les séries communiquées récemment: $r = 0,98$ [33] et $r = 0,80$ (Fig. 4). Un examen ultérieur de la corrélation montre, néanmoins, qu'un Δ pH_{qu 40} $\geq 0,05$ materno-foetal élimine l'hypoxie foetale comme cause éventuelle de l'acidose, tandis qu'une valeur > 0,05 n'exclut pas nécessairement une étiologie maternogénique (Fig. 5).

Valeur de pronostic du Δ BD_{Hb5} materno-foetal en présence de changements du le FHR durant le travail

Dans les cas présentant des changements du FHR (décélérations variables et tardives durant les 90 minutes précédant l'accouchement), le Δ BD_{Hb5} materno-foetal a été dans nos séries: a) négatif dans les cas classés par la suite comme APGAR < 4; b) positif pour ceux classés APGAR $\geq$ 8; c) pour APGAR entre 5 et 7 les valeurs ont été à la fois positives et négatives (Fig. 6).

Si on divise les décélérations tardives par rapport à la saturation d'oxygène de l'hémoglobine, on peut considérer que lorsque celle-ci est normale, le Δ BD_{Hb5} materno-foetal est positif. Les valeurs négatives du Δ BD_{Hb5} surviennent seulement en cas d'hypoxie foetale (Tab. VI).

Conclusions

Les résultats précisés ci-dessus semblent indiquer que:

1. le facteur métabolique de l'équilibre acidobasique foetal se reflète davantage dans le BD_{Hb5} que dans le pH_{qu 40} ou dans le BD non corrigé;

2. dans le diagnostic d'acidose foetale maternogénique, le ΔBD_{Hb5} materno-foetal est un test plus sûr que le ΔpH_{Hqu} 40, malgré l'étroite corrélation statistique entre les deux.

Si pendant le travail le pH foetal est bas et on reconnaît que cette acidose foetale est d'origine maternelle, on peut

décider de ne pas procéder à une extraction rapide du foetus; et la diminution du nombre des cas requérant une intervention augmentera proportionnellement à la précision du paramètre utilisé pour la comparaison des facteurs métaboliques de l'équilibre acidobasique maternel et foetal.

Mots-clés: Acidose métabolique foetale maternogénique, analyse du sang foetale, bradycardie, déficit basique, équilibre acidobasique, pH_{qu} 40, travail.

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