J. Perinat. Med.
11 (1983) 278

Systematic pH-measurements in the umbilical artery: Causes and predictive value of neonatal acidosis

P. A. Lauener, A. Calame, P. Janecek, H. Bossart, J. F. Monod

Department of Pediatrics and Department of Obstetrics,
Centre Hospitalier Universitaire Vaudois (CHUV),
Lausanne, Switzerland

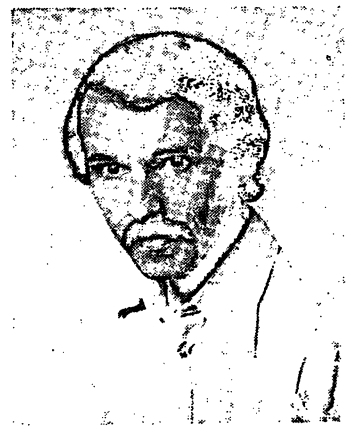
1 Introduction

The evaluation of fetal wellbeing has been a major concern for many years. Several methods, i. e. fetal heart rate monitoring [11, 21, 24, 25], scalp blood pH monitoring [18, 48, 54, 55, 67], continuous pH monitoring [29, 30,], continuous P_{O_2} monitoring [27], sub partu EEG recording [12] have been developed to recognize a threatening intrapartum asphyxia.

The APGAR score [2] was the first very useful attempt to assess the state of adaptation of a newborn baby. Over the past 20 years biochemical parameters such as blood gases, acid-base balance and lactic acid [7, 14, 18, 23, 28, 34, 38, 54, 65] have been studied. During these past few years many hopes have been placed on the routine umbilical arterial blood pH determination ($pH_{a.umb.}$) as a mean of assessing fetal asphyxia [5, 7, 14, 23, 28, 34, 50]. Causes of neonatal acidosis have been studied by several authors [3, 6, 32, 33, 40, 41, 49, 59]. Adaptational problems and higher morbidity in the early neonatal period have been found to be related to a low $pH_{a.umb.}$ [23, 28, 34, 65].

The aim of this study was to examine the long term outcome of acidotic babies at birth and to determine the relationship of acidosis to perinatal risk factors.

Curriculum vitae



PIERRE-ANDRE LAUENER *was born in 1948 in Zürich. He studied medicine at the University of Zürich from which he was graduated in 1975. In the following years he worked successively at the Hôpital Central, N'Djaména, Tchad, Service de Pédiatrie, CHUV, Lausanne and Universitäts-Kinderklinik, Zürich. Since 1982, he is chief resident at the Service de Pédiatrie, CHUV, Lausanne.*

2 Patients and methods

During a 16-month period 1922 deliveries were performed at the Department of Obstetrics of the University Hospital of Lausanne (CHUV), Switzerland. 1911 pH measurements were done and blood samples could not be obtained for technical reasons in 11 deliveries. Seven babies with a $pH_{a.umb.} < 7.15$ were excluded because of trisomy, congenital malformations, congenital infections or extreme prematurity (< 28 weeks gestational age).

1904 deliveries were included in this study. Acidosis was defined as a $pH_{a.umb.} < 7.15$ [35].

Blood for gas analysis was obtained by arterial punction of the umbilical cord immediately after delivery. The measurements were done by an ABL 1 apparatus (RADIOMETER, Copenhague). The obstetrical data were extracted from standardized charts. Fetal heart patterns could be recorded in 83% of the acidotic infants, to be reviewed by one of us (J. F. M.) and classified according to the HAMMACHER score [21].

Prolonged rupture of the membranes was defined as a minimal delay of 12 hours between rupture and delivery. Cord complications included cord around neck, cord around the body, short cord and true knot.

Asphyxia was defined as an APGAR score $\leqslant 4$ at 1 minute. APGAR scoring was done by midwives in uneventful deliveries or by pediatricians in all other cases. Gestational age was assessed according to DUBOWITZ scoring [16] and to obstetrical data. All babies were routinely examined by a pediatrician during the first 24 hours, then between day 5 and 7 according to a standardized protocol. The follow up was performed by pediatricians using the DENVER DEVELOPMENTAL SCREENING TEST [19]. Minimal frequency of controls was 5 times during the first year of life and 2 times during the second year. Tone abnormalities during the first week of life were defined as hypotonia with or without apathy and hypertonia with or without irritability. Major neurological complications in the first week of life included convulsions, absence of primitive reflexes and severe hypo- or hypertonia.

The significance of statistical differences was studied by the X^2-test (four-fold table with YATES' correction).

3 Results

Out of 1904 deliveries, 1783 cord blood samples had a $pH_{a.umb.} \geqslant 7.15$ and 121 had a $pH_{a.umb.} < 7.15$ (6.4%). 38 babies (2.0%) had a $pH_{a.umb.} \leqslant 7.10$. These date are shown in Tab. I.

A comparison was made of the incidence of perinatal risk factors in deliveries with a $pH_{a.umb.} \geqslant 7.15$ vs deliveries with a $pH_{a.umb.} < 7.15$ (Tab. II).

Tab. I. Total numbers and distribution.

$pH_{a.umb.}$	N	%
$\geqslant 7.15$	1783	93.6
7.11–7.14	83	4.4
$\leqslant 7.10$	38	2.0
Total	1904	100.0

The number of acidotic babies is significantly higher in primiparae, in deliveries with cord complications or prolonged rupture, and in deliveries with forceps or on peridural analgesia. There are no statistically significant differences when multiple pregnancies, meconium-stained amniotic fluids, cesarean sections, gestational age (28 through 37 weeks of gestation), and sex are considered.

The mean $pH_{a.umb.}$ ($\overline{pH}$) value was tabulated against HAMMACHER scores. The results are shown

Tab. II. Neonatal acidosis vs perinatal risk factors.

Risk factors	pH ⩾ 7.15 N = 1783 (%)	pH < 7.15 N = 121 (%)	X^2
Primiparae	848 (47.6)	75 (62.0)	$p < 0.01$
Cord complications	190 (10.7)	46 (38.0)	$p < 0.001$
Prolonged rupture	241 (13.5)	40 (33.1)	$p < 0.001$
Peridural analgesia	510 (28.6)	65 (53.7)	$p < 0.001$
Forceps delivery	160 (9.0)	24 (19.8)	$p < 0.001$
Multiple pregnancy	26 (1.5)	5 (4.1)	NS
Meconium stained amniotic fluid	222 (12.5)	18 (14.9)	NS
Cesarean section	300 (16.8)	21 (17.4)	NS
Prematurity 28–37 weeks	67 (3.8)	9 (7.4)	NS
Male	931 (52.2)	61 (50.4)	NS
Female	852 (47.8)	60 (49.6)	NS

Tab. III. $pH_{a.umb.}$ vs HAMMACHER score.

HAMMACHER score	N	$\overline{pH}_{a.umb.}$	range
0–2	35	7.11	6.9 –7.14
3–4	55	7.11	6.9 –7.14
5–6	11	7.02	6.58–7.13

Tab. IV. APGAR score at 1 min vs $pH_{a.umb.}$

$pH_{a.umb.}$	$Apgar_{1\,min} \leq 4$	$Apgar_{1\,min} \geq 5$
< 7.15	13/ 121 (10.7%)	108/ 121 (89.3%)
≥ 7.15	22/1783 (1.2%)	1761/1783 (98.8%)

in Tab. III. The pH was 7.11 for HAMMACHER scores of 0–2 and 3–4, as opposed to 7.02 for 5–6 scores.

Out of 121 acidotic infants, 13 (10.7%) had an APGAR score ≤ 4 at 1 minute as compared to 22 (1.2%) of 1783 with a $pH_{a.umb.} \geq 7.15$. The difference is significant ($p < 0.001$) (Tab. IV).

The follow up study refers only to these infants who were acidotic at birth ($pH_{a.umb.} < 7.15$). Follow up time ranged from 6 through 25 months (mean 15.0 months). Of 121 acidotic babies ($pH_{a.umb.} < 7.15$), 23 infants were lost for follow up or followed up for less than 6 months. The neurological outcome for the first week of life is summarized in Tab. V. Eighty babies (81.6%) were neurologically normal. Sixteen infants (16.4%) showed tone abnormalities, of whom all but two fully recovered within one week. Two (2.0%) infants developed major neurological problems: The first baby was born after prolonged Dip II decelerations and displayed clinical signs of severe asphyxia (APGAR score of 3/6/ –) as well as a $pH_{a.umb.}$ of 6.90. The second delivery was complicated by maternal anaphylactic shock on a spasmolytic drug and required major resuscitation manoeuvres. The baby was delivered by cesarean section and showed an APGAR score of 4/4/4 and a $pH_{a.umb.}$ of 6.58. Both babies were born at term. The mean values for the CGT score ($\overline{CGT}$), $pH_{a.umb.}$ ($\overline{pH}$) and APGAR score at 1 min ($\overline{APGAR}$) vs early neurological exam are shown in Tab. V.

Tab. V. Early neurologic outcome (first week of life) of acidotic babies ($pH_{a.umb.} < 7.15$).

Outcome	N (%)	Perinatal parameters		
		$\overline{CTG}$ score	$\overline{pH_{a.umb.}}$	$\overline{APGAR}$ score
Normal	80 (81.6)	2.2	7.10	7.7
Hypotonia/ apathy or Hypertonia/ irritability	16 (16.4)	3.3	7.10	7.0
Major neurologic neonatal complication	2 (2.0)	5/5	6.90/6.58	3/4
Total	98 (100.0)	2.4	7.09	7.5

Tab. VI. Long term outcome of babies acidotic at birth ($pH_{a.umb.} < 7.15$).

Outcome	N (%)	Mean follow up time (months)
Normal – uneventful	78 (79.6)	14.5
Normal – transient neurologic problems	18 (18.4)	16.6
Major neurologic complications (CP)	2 (2.0)	24/24
Total	98 (100.0)	15.0

The long term outcome is summarized in Tab. VI. 96 babies (98%) exhibited a normal psycho-motor development. Of these 18 (18.4%) displayed minor and transient neurological problems: Trouble of muscle tone maturation (12 infants), isolated motor retardation (2 infants), neuro-ophtalmic problems – transient squint, palpebral ptosis, anisocoria – (3 infants), and febrile convulsion (1 infant). Two children were showing signs of cerebral palsy and mental retardation at 24 months of age. In both of them, hypoxic-ischemic lesions were revealed on CAT-scans.

These two children are the same 2 babies who suffered major neonatal neurologic complications (Tab. V).

4 Discussion

As mentioned in previous reports an increased risk for perinatal acidosis has been found in primiparae [23, 40] and in deliveries complicated by cord entanglement [3, 6, 52, 62] or prolonged rupture of the membranes [41]. Neonatal acidosis is significantly more frequent in deliveries done with peridural analgesia. Studies on the effects of peridural analgesia on the fetus and the newborn have been made by different authors and yielded quite conflicting results [4, 8, 9, 13, 31, 39, 42, 44, 49, 51, 59, 64, 66, 68]. In addition to the above mentioned predisposing factors, other studies have shown the risk for neonatal acidosis to be increased by breech presentation [32, 65], by

small birth weights [65], by older maternal age [40], and by prolonged second stage of labor [23, 40]. Altered placental perfusion [20, 49] and impaired feto-placental circulation [3, 52, 62] are the common basic mechanisms leading to the accumulation of CO_2 and nonvolatile acids in the fetus. Relative maternal acidosis due to acid local anesthetics and/or a prolonged second stage of labor [49] might be partly responsible for a "transfusional acidosis" [50]. However, there is usually only a ΔpH gradient of 0.1 between the mother and the fetus. Even in this selected population $pH_{a.umb.}$ is markedly lower in infants whose HAMMACHER scores are greater than 4. The direct correlation between the HAMMACHER score and the $pH_{a.umb.}$ has well been proven in other more comprehensive studies [35].

This study demonstrates that the incidence of clinical asphyxia is greater in infants whose arterial cord blood pH is less than 7.15. Other authors have studied this relationship and found comparable results [7, 14, 28, 34, 50]. They, too, found a number of discordant values, i.e. acidotic babies with a normal APGAR score or asphyxiated babies with a normal $pH_{a.umb.}$. Their interpretation was, however, quite different: PONTONNIER [50] ascribed neonatal acidosis with a good APGAR score in most case to "transfusional acidosis"; on the other hand he attributed neonatal depression to the effect of maternal anesthesia. VOIGT [65] considered the measurement of the $pH_{a.umb.}$ to be more reliable a parameter since the assessment of the APGAR score is influenced by subjective factors. We believe that acute acidosis due to the predisposing mechanisms studied earlier in this paper does not necessarily influence neontatal adaptation and that it could explain discrepance between $pH_{a.umb.}$ and APGAR score.

When considering early developmental outcome, 81.6% of babies were found to be neurologically normal. 16.4% showed minor neurological problems, and 2.0% severe neurological complications. Other authors have studied the perinatal acidosis as it relates to early neurological outcome. LOW [37] found that severe cerebral symptoms in newborn infants were often associated with more severe metabolic acidosis at birth. In this study, however, clinically asphyxiated infants could not be segregated into neurologically symptomatic or asymptomatic on the basis of acid-base characteristics. LITSCHGI [34] found a greater incidence of infants suspect of having brain damage among those with a lower $pH_{a.umb.}$. The long term outcome of clinically asphyxiated babies has been studied by many authors [5, 15, 37, 46, 47, 63]. Little is known, however, about the influence of neonatal acidosis. LORENZ [36] presented recently a study similar to ours. However, his data are not yet published.

In our population of 98 acidotic babies, two infants have severe neurological sequalae (CP). They both presented with severe intrauterine asphyxia, extreme perinatal acidosis ($pH_{a.umb.}$ = 6.58 and 6.90) and adaptational problems followed by early and severe neurologic complications. The fact that the rate of glycolysis falls progressively with a decreasing pH and ceases altogether at a pH of 6.9 [48] could, at least partly, account for irreversible brain damage. The 96 infants who eventually displayed a normal psycho-motor development had all a $pH_{a.umb.} > 6.90$. Our study gives no clues as to the cause of minor transient neurologic problems that occured in 18.4% of these infants.

5 Conclusions

Whether it be associated with intrapartum asphyxia, with severe adaptational problems or with early and persistent neurologic problems perinatal acidosis has a poor prognosis. However, perinatal acidosis with a $pH_{a.umb.}$ between 6.91 and 7.15, not associated with severe perinatal asphyxia (documented by CTG or APGAR score) and not followed by early major neurologic complications, has a good prognosis. One or several predisposing factors might be responsible for the perinatal acidosis in these cases. A number of such infants present with varied minor and transient neurologic problems during their first year of life. Therefore, while $pH_{a.umb.}$ – except perhaps in extremely low values – cannot be taken alone as a prognostic factor for the long term outcome, the measurement of the $pH_{a.umb.}$ should be performed and evaluated in association with fetal heart rate monitoring and APGAR scoring to assess the well-being of the fetus and newborn infant.

Summary

Subpartal and neonatal blood gas analyses have attracted increasing interest during the past 20 years. Different studies have been carried out to investigate the causes and immediate consequences of perinatal acidosis. It was the aim of this study to examine the long term outcome of acidotic-born babies.

During 16 consecutive months all deliveries in the Obstetric Department of the Centre Hospitalier Universitaire Vaudois (CHUV) were investigated with regard to incidence and causes of a perinatal acidosis ($pH_{a.umb.} < 7.15$). The psycho-motor development of all acidotic newborns was followed up for an average of 15 months. Out of 1922 deliveries a blood sample was available in all but 11 cases (0.57%). Seven newborns were excluded from the study on the assumption that their acidosis and outcome might be related to the underlying condition (congenital malformation and infection, extreme prematurity) rather than perinatal events. From the remaining 1904 deliveries 6.4% (N = 121) had a $pH_{a.umb.} < 7.15$ (**Tab. I**). The incidence of certain perinatal factors was compared in the acidotic and the non acidotic groups (**Tab. II**). The percentage of acidotic newborns is significantly higher in primipareae, in deliveries done on peridural analgesia in cord complications and premature rupture of the membranes, and in forceps deliveries. No difference could be found with regard to multiple pregnancies, meconiumstained amniotic fluid, cesarean sections, prematurity of 28–37 weeks, and sex. The relationship between CTG score and $pH_{a.umb.}$ is summarized in **Tab. III**. The percentage of APGAR score ≤ 4 at 1 minute is significantly higher in infants with a $pH_{a.umb.} < 7.15$ compared to those with a $pH_{a.umb.} \geq 7.15$ (**Tab. IV**).

During the first week of life 81.6% of all babies had a normal neurological assessment, 16.4% had minor neurological problems and 2.0% had severe neurological complications (**Tab. V**). The latter had significantly worse values for CTG scores, $pH_{a.umb.}$ and APGAR scores; although 98% of infants had a normal psycho-motor development in the long term, 18.4% exhibited minor transient troubles, predominantly of muscle tone maturation. Two percent of all infants showed cerebral palsy and mental retardation (**Tab. VI**). The discordance between APGAR score and $pH_{a.umb.}$ can, at least partly, be explained by the above demonstrated predisposing factors.

No data can be found in the literature about the long term outcome of acidotic-born babies. The 2 infants with cerebral palsy observed in our study had severe perinatal complications, so that their neurological problems cannot be satisfactorily explained by the neonatal acidosis alone. Therefore, the $pH_{a.umb.}$ – except perhaps in extremely low values – cannot be taken alone as a predicitve factor for the long term outcome. It still retains, however, its value in the assessment of the health of the newborn, together with other perinatal parameters such as CTG and APGAR score.

Keywords: Acidosis, APGAR score, asphyxia, blood gases, neonatal acidosis, pH umbilical artery, psycho-motor development.

Zusammenfassung

Systematische pH-Messungen im Nabelarterienblut: Ursachen und Prognose einer neonatalen Azidose

Während der letzten 20 Jahre galt den subpartalen und neonatalen Blutgasanalysen ein ständig wachsendes Interesse. Verschiedene Untersuchungen wurden durchgeführt, um die Ursachen und unmittelbaren Folgen einer perinatalen Azidose zu erforschen. Ziel unserer Studie war die Untersuchung von Spätfolgen bei Kindern mit perinataler Azidose.

16 Monate lang wurden alle Entbindungen in der geburtshilflichen Abeilung der Universitätsklinik von Lausanne (CHUV) im Hinblick auf Inzidenz und Ursachen einer perinatalen Azidose ($pH_{N.A.} < 7.15$) untersucht. Bei allen azidotischen Kindern wurde die psycho-motorische Entwicklung bis zu einem durchschnittlichen Lebensalter von 15 Monaten verfolgt. Mit Ausnahme von 11 Fällen (0.57%) konnten bei allen 1922 Geburten Blutproben gewonnen werden. Sieben Neugeborene wurden aus der Studie herausgenommen, weil hier wahrscheinlich nicht perinatale Komplikationen, sondern andere Ursachen (congenitale Mißbildungen und Infektionen, extreme Frühgeburt) zur Azidose geführt haben. Von den verbleibenden 1904 Neugeborenen hatten 6.4% (N = 121) einen $pH_{N.A.} < 7.15$ (**Tab. I**). Wir haben die Inzidenz bestimmter Faktoren in der azidotischen bzw. nichtazidotischen Gruppe miteinander verglichen (**Tab. II**): So war der Anteil azidotischer Neugeborener bei den Erstgebärenden signifikant höher, ebenso bei Entbindungen in Periduralanästhesie, bei Nabelschnurkomplikationen, bei vorzeitigem Blasensprung und bei Zangengeburten, während bei Mehrlingsschwangerschaften, mekoniumhaltigem Fruchtwasser, bei Sectio caesarea und Frühgeburten der 28.–37. Woche sowie hinsichtlich des Geschlechts keine Unterschiede gefunden werden konnten. Der Zusammenhang zwischen CTG-Score und $pH_{N.A.}$ ist in **Tab. III** dargestellt. Der Anteil von Neugeborenen, die 1 Minute post partum einen APGAR-Score ≤ 4 hatten, ist bei Kindern mit einem $pH_{N.A.} < 7.15$ signifikant höher als bei Neugeborenen mit einem $pH_{N.A.} \geq 7.15$ (**Tab. IV**).

Während der ersten Lebenswoche waren 81.6% aller Kinder neurologisch unauffällig; 16.4% zeigten minimale neurologische Auffälligkeiten und 2% hatten schwere neurologische Komplikationen (**Tab. V**). In der letzten Gruppe wurden signifikant schlechtere CTG-Scores, $pH_{N.A.}$-Werte und APGAR-Scores gemessen. Obwohl sich 98% der Kinder psychomotorisch normal entwickelten, hatten 18.4% vorübergehend neurologische Störungen, hauptsächlich den Muskeltonus betreffend. Bei 2% traten Cerebralparesen und eine mentale Retardierung auf

(Tab. VI). Die Diskordanz zwischen APGAR-Score und $pH_{N.A.}$ kann zum Teil durch die oben beschriebenen prädisponierenden Faktoren erklärt werden.
In der Literatur finden sich keine Daten über die weitere Entwicklung azidotischer Neugeborener. Die beiden Kinder mit Cerebralparesen, die in unserer Studie beobachtet wurden, hatten schwerwiegende perinatale Komplikationen; ihre neurologische Symptomatik kann nicht allein durch die neonatale Azidose erklärt werden. Darum kann der $pH_{N.A.}$, abgesehen von extrem niedrigen Werten, nicht allein zur Prognose der weiteren Entwicklung herangezogen werden. Zusammen mit anderen perinatalen Parametern wie CTG und APGAR-Score ist er jedoch wertvoll bei der Beurteilung des neonatalen Zustandes.

Schlüsselwörter: APGAR-Score, Asphyxie, Azidose, Blutgase, neonatale Azidose, pH im Nabelarterienblut, psychomotorische Entwicklung.

Résumé

Détermination systématique du pH artériel ombilical: Causes et valeur prédictive de l'acidose néonatale
Durant ces 20 dernières années, l'analyse des gaz sanguins per-partum et à la naissance a fait l'objet d'un intérêt croissant. Plusieurs études ont été réalisées pour apprécier les causes et les conséquences immédiates de l'acidose périnatale. Le but de ce travail est de déterminer le devenir à long terme des enfants nés en état d'acidose.
Durant 16 mois consécutifs, l'incidence et la cause de l'acidose périnatale ($pH_{a.omb.} < 7.15$) ont été étudiées dans tous les accouchements du Département d'Obstétrique du Centre Hospitalier Universitaire Vaudois (CHUV), Lausanne. Le développement psychomoteur de tous les nouveaux-nés acidotiques a été suivi pendant une moyenne de 15 mois. Sur les 1922 accouchements, 11 n'ont pas eu de prélèvement sanguin (0.57 %). Sept nouveaux-nés ont été exclus de l'étude en raison de malformations congénitales, infections et prématurité extrême. Sur les 1904 naissances restantes, 6.4 % (N = 121) on eu un $pH_{a.omb.} < 7.15$ (**Tab. I**).
L'incidence de facteurs périnataux dans les groupes avec et sans acidose a été comparée (**Tab. II**). Le pourcentage de nouveaux-nés acidotiques est plus élevé de façon significative chez les primipares, en cas d'accouchement sous analgésie péridurale, en cas de complications du cordon ombilical, lors de rupture prématurée des membranes et lors de forceps. Aucune différence n'a été trouvée dans les grossesses multiples, en présence de liquide amniotique méconial, lors de césariennes, ni en relation avec la prématurité (âge de gestation 28 à 37 semaines) et le sexe. La relation entre le score du CTG et le $pH_{a.omb.}$ est résumée sur le **Tab. III**. Le pourcentage de score d'APGAR ≤ 4 à 1 minute est significativement plus élevé chez les enfants ayant un $pH_{a.omb.} < 7.15$ que chez ceux ayant un pH ≥ 7.15 (**Tab. IV**).
81.6 % des nouveaux-nés acidotiques ont un bilan neurologique normal pendant la première semaine de vie, 16.4 % ont des troubles neurologiques mineurs et 2 % ont des complications neurologiques graves (**Tab. V**). Ces derniers ont de façon significative des résultats moins bons à la détermination du score de CTG, du $pH_{a.omb.}$ et au score d'APGAR. Parmi les 98 % des enfants qui ont un développement psychomoteur normal à long terme, 18.4 % ont présenté des troubles transitoires minimes (maturation du tonus musculaire). Deux pour cent ont une infirmité motrice cérébrale (IMC) et un retard mental (**Tab. VI**). La différence entre le score d'APGAR et le $pH_{a.omb.}$ est partiellement explicable par les facteurs prédisposants périnataux étudiés.
Le follow-up à long terme des enfants nés avec une acidose a été peu étudié. Les 2 enfants atteints d'IMC ont eu des complications périnatales majeures et leur handicap ne peut probablement pas être expliqué uniquement par l'acidose néonatale. Le $pH_{a.omb.}$, donc, à l'exception des valeurs extrêmement basses, ne peut être considéré à lui seul comme un élément prédictif du devenir à long terme. Toutefois il conserve sa valeur pour l'évaluation de l'état de santé du fœtus et du nouveau-né au même titre que les autres paramètres périnataux que sont le CTG et le score d'APGAR.

Mots-clés: Acidose, acidose néonatale, asphyxie, développement psychomoteur, gaz du sang, pH dans l'artère ombilicale, score d'APGAR.

Bibliography

[1] ABRAMOVICI, H., J. M. BRANDES, K. FUCHS, I. TIMOR-TRITSCH: Meconium during delivery: A sign of compensated fetal distress. Amer. J. Obstet. Gynec. 118 (1974) 251
[2] APGAR, V.: A proposal for a new method of evaluation of the newborn infant. Anaesth. Analg. 32 (1953) 260
[3] ARTO-MEDRANO, F.: Influence of cord around the neck on fetal acid-base balance in vigorous newborn infants. Amer. J. Obstet. Gynec. 107 (1970) 1035
[4] BARRIER, G.: Le retentissement sur l'enfant de l'anesthésie péridurale. Ann. Anesth. Franç. XIX, 4 (1978) 241

[5] BEARD, R. W., R. P. A. RIVERS: Fetal asphyxia in labor. Lancet (1979) 1117
[6] BENDER, H. G., CH. WERNER, C. KARSTEN: Zum Einfluß der Nabelschnurstruktur auf Schwangerschafts- und Geburtsverlauf. Arch. Gynäk. 225 (1978) 347
[7] BOEHM, S.: Wert eines generellen Nabelarterien-pH für die Beurteilung der Azidosemorbidität. Zbl. Gynäk. 100 (1978) 842
[8] BOEHM, F. H., L. F. WOODRUFF JR., J. H. GROWDON JR.: The effect of lumbar epidural anesthesia on fetal heart rate baseline variability. Anesthesia and analgesia, current researches, 54 (1975) 779
[9] BOSSART, H., P. Y. DUBUIS, P. JANECEK, J.-F. MONOD, P. SUPPAN: Foetal and neonatal response to general and peridural anesthesia during elective caesarean section. 7th European Congress of Perinatal Medicine, September 2th–5th, Barcelona (1980)
[10] BROWN, J. K., R. J. PURVIS, J. O. FORFAR, F. COCKBURN: Neurological Aspects of Perinatal Asphyxia. Dev. Med. Child. Neurol. 16 (1974) 567
[11] CALDEYRO-BARCIA, R., C. MENDEZ-BAUER, J. J. POSEIRO, S. V. POSE, L. ESCARCENA, J. BIENARZ, O. ALTHABE, L. GULIN: La fréquence du cœur fœtal pendant l'accouchement. Signification de ses variations. In: XXI è Congrès de la Fédération des Sociétés de Gynécologie et Obstétrique de Langue Française – Lausanne 5–8 septembre 1965. Bulletin de la Féd. Soc. Gyn. et Obst. No. 1 bis et 5 bis, Masson et Cie, Paris (1965)
[12] CHACHAVA, K. V.: Fetal EEC. 5th European Congress of Perinatal Medicine Uppsala, Sweden, 9–12 June 1976. ROOTH, C., L. E. BRATTEBY (eds.): Almqvist & Wiksell International, Stockholm 1976
[13] CRAWFORD, J. S.: Continuous lumbar epidural analgesia for labour and delivery. Brit. Med. J. 1/6156 (1979) 72
[14] DEBDAS, A., N. N. R. CHOWDHURY: Fetal distress and pH value of umbilical cord blood. Amer. J. Obstet. Gynec. 107 (1970) 1044
[15] DE SOUZA, S. W., B. RICHARDS: Neurological sequalae in newborn babies after perinatal asphyxia. Arch. Dis. Childh. 53 (1978) 564
[16] DUBOWITZ, L. M. S., V. DUBOWITZ, C. GOLBERG: Clinical assessment of gestational age in the newborn infant. J. Pediat. 77 (1970) 1
[17] FEIGE, A., W. KÜNZEL, M. CORNELY, H. J. MITZKAT: Die Beziehung zwischen Glukosekonzentration und Säure-Basen-Status im maternen und fetalen Blut während der Geburt. Z. Geburtsh. Perinat. 180 (1976) 106
[18] FISCHER, W. M.: Untersuchung zum Säure/Basen-Gleichgewicht im fetalen Blut vor der Geburt. Arch. Gynäk. 200 (1965) 534
[19] FRANKENBURG, W. K., J. B. DODDS: The Denver Developmental Screening Test. J. Pediat. 71 (1967) 181
[20] GOODLIN, R. C.: Aortocaval compression during caesarean section. A cause of newborn depression. Obst. and Gynec. 37 (1971) 702
[21] HAMMACHER, K.: Diagnosis of fetal distress by means of cardio-tocographie. In Vth. World Congr. of Gynaec. and Obstet. Sept. 23–30, 1967, Sydney, Australia. Butterworths, Australia (1967)
[22] HAMMACHER, K.: Die semiquantitative Auswertung von Kardiotokogrammen anhand eines CTG-Scores. In: Perinatale Medizin in Band VI. DUDENHAUSEN, J. W., E. SALING, S. SCHMIDT (eds.): Thieme, Stuttgart (1975)
[23] HOCHULI, E., F. NAGL, O. DUBLER: Hypoxi- und Azidosemorbidität – Eine kritische Studie anhand von 3291 Nabelschnurblutgasanalysen und ihre Konsequenzen. Z. Geburtsh. Perinat. 179 (1975) 112
[24] HON, E. H., H. S. HUANG: The electronic evaluation of fetal heart rate. VII premature and missed beats. Obstet. and Gynec. 20 (1962) 81
[25] HON, E. H., E. J. QUILLIGAN: The classification of fetal heart rate. II: A revised working classification. Conn. Med. 31 (1967) 779
[26] HRBEK, A., P. KARLBERG, I. KJELLMER, T. OLLSON, M. RIHA: Clinical application of evoked electroencephalographic response in newborn infants. I: Perinatal asphyxia. Dev. Med. Child. Neurol. 19 (1977) 34
[27] HUCH, A., R. HUCH, H. SCHNEIDER, J. PEABODY: Experience with transcutaneous P_{O_2} (tcp_{O_2}) monitoring of mother, fetus and newborn. J. Perinat. Med. 8 (1980) 51
[28] HUISJES, H. J., J. G. AARNOUDSE: Arterial or venous pH as a measure of neonatal morbidity? Early Human. Dev. 3/2 (1979) 155
[29] JANECEK, P.: Surveillance néonatale par enregistrement continu du pH sous-cutané. Thèse – Lausanne. Buri Druck AG, Berne 1976
[30] JANECEK, P., H. BOSSART: Clinical aspects of continuous issular pH measurements of newborn and the fetus. Arch. Gyn. 226 (1978) 121
[31] JANECEK, P., P. SUPPAN, H. BOSSART, U. STOLL: Anesthésie péridurale continue en obstétrique. Résultats obstétricaux et impact psychologique. Congr. Soc. Suisse Gynéc., St-Gall 1980. Gynäk. Rundsch. 20 (Suppl. 1) (1980) 65
[32] KENNY, J. D., A. J. CORBET, J. M. ADAMS, A. J. RUDOLPH: Hyaline membrane disease and acidosis at birth in twins. Obstet. and Gynec. 50 (1977) 710
[33] KUBLI, F., H. RÜTTGERS, M. MEYER-MENK: Die fetale Acidosegefährdung bei vaginaler Geburt aus Beckenendlage. Z. Geburtsh. Perinat. 179 (1975) 1
[34] LITSCHGI, M., J. J. BEUZ, E. GLATTHAAR: Aktuelle und prognostische Bedeutung des arteriellen Nabelschnur-pH für die postpartale Zustandsdiagnostik. Z. Geburtsh. Perinat. 178 (1974) 23
[35] LITSCHGI, M., A. TSCHUMI: Vorschlag für eine Änderung der diagnostischen Kriterien der Azidose zwischen 0 und 60 Minuten post partum. Communication at the annual meeting of the Swiss Group for Neonatology, Neuchâtel, 1979
[36] LORENZ, U., G. ACEVEDO, S. TREILING, M. LEHMANN, R. FISCHER, H. RÜTTGERS, F. KUBLI: Neuromotor disturbances after pathologic antepartum cardiotocogram and/or acidosis in the umbilical artery. 8th European Congress of Perinatal Medicine, Brüssel 1982
[37] LOW, J. A., S. R. PANCHAM, W. N. PIERCY, D. WORTHINGTON, J. KARCHMAR: Intrapartum

fetal asphyxia: Clinical characteristics, diagnosis and significance in relation to pattern of development. Amer. J. Obstet. Gynec. 129 (1977) 857
[38] LOW, J. A., S. R. PANCHAM, D. WORTHINGTON, R. W. BOSTON: Acid-base lactate and pyruvate characteristics of the normal obstetric patient and fetus during the intrapartum period. Amer. J. Obstet. Gynec. 120 (1974) 862
[39] MAGNO, R., I. KJELLMER, K. KARLSSON: Anesthesia for caesarean section. III: Effects of epidural analgesia on the respiratory adaptation of the newborn in elective caesarean section. Acta Anaesth. Scand. 20 (1976) 73
[40] MANZL, J., A. BICHLER, K. SCHOLZ, H. HETZEL, H. FRISCH, W. GEIR: Einfluß des Alters Erstgebärender auf die perinatale kindliche Morbidität und Mortalität. Z. Geburtsh. Perinat. 181 (1977) 168
[41] MARTELL, M., J. M. BELIZAN, F. NIETO, R. SCHWARCZ: Blood acid-base balance at birth in neonates from labors with early and late rupture of membranes. J. Pediat. 89 (1976) 963
[42] MCDONALD, J. S., L. L. BJORKMAN, E. C. REED: Epidural analgesia for obstetrics. Amer. J. Obstet. Gynec. 120 (1974) 1055
[43] MILLER, F. C., D. A. SACKS, S. YEH, R. H. PAUL, B. S. SCHIFRIN, C. B. MARTIN, E. H. HON: Significance of meconium during labor. Amer. J. Obstet. Gynec. 122 (1975) 573
[44] MONOD, J.-F.: Césarienne élective: Anesthésie générale ou péridurale. Répercussions biophysiques et acido-basiques maternelles, fœtales et néonatales. Thèse – Lausanne, 1981
[45] MÜLLER-HOLVE, W., E. SALING, M. SCHWARZ: The significance of time interval in twin delivery. J. Perinat. Med. 4 (1976) 100
[46] MULLIGAN, J.-C., M. J. PAINTER, P. A. O'DONOGHUE, H. M. MCDONALD, A. C. ALLEN, P. M. TAYLOR: Neonatal asphyxia II. Neonatal mortality and long term sequalae. J. Pediat. 96 (1980) 903
[47] NELSON, K. B., J. H. ELLENBERG: APGAR scores as predictors of chronic neurologic disability. Pediatrics 68 (1981) 36
[48] PEARSON, J. F.: Maternal and fetal acid-base balance. In: Fetal Physiology and Medicine. BEARD, R. W., P. W. NATHANIELSZ (eds.) W. B. Saunders Philadelphia, London, Toronto 1976
[49] PEARSON, J. F., P. DAVIS: The effect of continuous lumbar epidural analgesia on the acid-base balance and arterial lactate concentration during the second stage of labor. J. Obstet. Gynaec. Brit. Cwlth. 80 (1973) 225
[50] PONTONNIER, G., H. GRANDJEAN, P. DERACHE, J. M. REME, M. BOULOGNE, J. DE MOUZON: Intrérêt de la mesure du pH sanguin dans la surveillance fœtale pendant l'accouchement. J. Gynec. Obstet. Biol. Reprod. 7 (1978) 1065
[51] RALSTON, D. H., S. M. SHNIDER: The fetal and neonatal effects of regional anesthesia in obstetrics. Anesthesiology 48 (1978) 34
[52] ROEMER, V. M., C. BOGDAN, R. BRUN DEL RE, P. DE GRANDI, K. HARMS, M. RAMZIN, K. HAMMACHER, M. HINSELMANN: Evaluation of fetal risk resulting from cord-entanglements during parturition. Arch. Gynäk. 216 (1974) 99
[53] ROEMER, V. M., E. RUPPIN, W. DIECKMANN, K. H. NEESER: Beitrag zur postpartalen Zustandsdiagnostik des Feten nach Schnittentbindungen. Geburtsh. Frauenheilk. 31 (1971) 936
[54] ROOTH, G., R. MCBRIDE, B.J. IVY: Fetal and maternal pH measurements, a basis for common values. Acta Obstet. Gynec. Scand. 52 (1973) 47
[55] SALING, E.: Das Kind im Bereich der Geburtshilfe. Thieme, Stuttgart, 1966
[56] SALING, E., K. H. WULF: Zustandsdiagnose bei Neugeborenen. Fortschr. Med. 89 (1971) 12
[57] SCOTT, H.: Outcome of severe birth asphyxia. Arch. Dis. Childh. 51 (1976) 712
[58] SHNIDER, S. M., G. LEVINSON: Anesthésie pour césarienne. Anesthésie et réanimation en obstétrique. SHNIDER, S. M., G. LEVINSON, Med. Paris, Chapitre VII (1980)
[59] STRASSER, K., P. HARNACKE, H. ALBRECHT, J. MORGENSTERN, H. SCHMIDT: Einfluß der lumbalen Periduralanästhesie mit Katheter auf den mütterlichen und kindlichen Säure-Basen-Haushalt und den 1-Minuten-APGAR-Wert. Z. Geburtsh. Perinat. 179 (1975) 163
[60] SUPPAN, P., P. JANECEK, H. BOSSART: Anesthésie péridurale continue en obstétrique. Technique et problème d'organisation et de formation. Congr. Soc. Suisse Gynec. St-Gall 1980. Gynäk. Rundsch. 20 (1980) 67
[61] SYKES, G. S., P. M. MOLLOY, P. JOHNSON, W. GU, F. ASHWORTH, G. M. STIRRAT, A. C. TURNBULL: Do APGAR scores indicate asphyxia? Lancet I (1982) 494
[62] TEJANI, N. A., L. I. MANN, M. SANGHAVI, A. BHAKTHAVATHSAIAN, R. R. WEISS: The association of ombilical cord complications and variable decelarations with acid-base findings. Obstet. and Gynec. 49 (1976) 159
[63] THOMPSON, A. J., M. SEARLE, G. RUSSEL: Quality of survival after severe birth asphyxia. Arch. Dis. Childh. 52 (1977) 620
[64] TRONICK, E., S. WISE, H. ALS, L. ADAMSON, J. SCANLON, T. B. BRAZELTON: Regional obstetric anesthesia and newborn behavior: Effect over the first ten days of life. Pediatrics 58 (1976) 94
[65] VOIGT, R., W. KRAUSE, P. VOIGT: Klinische Untersuchungen zur Azidosemorbidität. Z. Geburtsh. Perinat. 182 (1978) 440
[66] WINGATE, M. B., L. WINGATE, L. IFFY, J. FREUNDLICH, D. GOTTSEGEN: The effect of epidural analgesia upon fetal and neonatal status. Amer. J. Obstet. Gynec. 119 (1974) 1101
[67] WULF, M., W. KÜNZEL, V. LEHMANN: Vergleichende Untersuchungen der arteriellen Blutgase und des Säure-Basen Status im fetalen und maternen Kapillarblut während der Geburt. Z. Geburtsh. Gynäk. 167 (1967) 113
[68] ZILIANTI, S. M.: Fetal heart rate and pH of fetal capillary blood during epidural analgesia in labour. Obstet. and Gynec. 36 (1970) 881

Received March 28, 1983. Revised June 20, 1983.
Accepted July 19, 1983.

Dr. Pierre-André Lauener
Service de Pédiatrie, CHUV
CH-1011 Lausanne, Switzerland