

VALIDITY OF CTG MONITORING FOR THE DIAGNOSIS OF ACUTE FOETAL HYPOXIA

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Abstract

Acute foetal hypoxia (AFH) is one of the most serious pathological conditions in the prenatal and perinatal periods and is a major risk factor for neonatal mortality and morbidity. It is routinely diagnosed by cardiotocography (CTG). However, it often gives false positive results which may be an indication for caesarean section (SC), a delivery which has a low effect on infant morbidity but may increase both maternal morbidity and mortality. In our group of 100 pregnant women, who had SC due to foetal hypoxia diagnosed on the basis of pathological or suspected pathological cardiotocograms, 68% of births were healthy babies with no signs of hypoxia. It is suggested that, in order to improve the effectiveness of AHP diagnosis, additional methods should be used, such as foetal pulse oximetry and scalp blood analysis, to reduce an unnecessary performance surgical delivery in the mother.

Key words

acute foetal hypoxia, cardiotocography, caesarean delivery, foetal pulse oximetry

INTRODUCTION

Acute foetal hypoxia (AFH) is one of the most serious pathological conditions during the prenatal and perinatal periods, and is a frequent cause of neonatal mortality and morbidity. Low levels of partial oxygen pressure in the foetal blood, usually caused by gas exchange disturbance (e.g. umbilical cord compression), participate in the development of AFH. Simultaneously, there is an increase in CO₂ that produces respiratory acidosis. This develops into metabolic acidosis due to the lack of oxygen in body tissues (4). Both respiratory and metabolic acidosis result in accumulation of H⁺ ions and a decrease in pH values in foetal blood. All these factors may cause foetal death or neonatal defects as a result of hypoxic encephalopathy.

Cardiotocography (CTG) is usually the only method available for AFH diagnosis in most of the obstetric wards, although other, but less objective methods, e.g., amnioscopy or amniotic fluid measurement also exist. CTG, with the use of a two-channel recorder, monitors foetal heart frequency and uterine activity. Due to a multifactorial aetiology of AFH, CTG is not sufficiently reliable

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and may give false positive results which, in turn, may increase indications for SC that represent a risk for the mother (12). In the Czech Republic, the increase in caesarean deliveries (about 13% of all deliveries) is alarming. Although, in many cases, SC has no effect on perinatal morbidity, it may increase maternal morbidity and mortality (about 50% of perinatal maternal deaths are associated with SC). Therefore, a thorough and consistent analysis of all findings indicating the need for SC is necessary.

After birth the level of hypoxia can be assessed only indirectly. There are two approaches: subjectively, examination by the attending neonatologist (Apgar score determination) and, objectively, analysis of foetal blood collected from the umbilical artery (5). However, the definite diagnosis of AFH can be made only at the neonatal ward by repeated analysis of foetal blood gases and pulse oximetry, and by assessment of the levels of lactate in foetal blood (>4 mmol/L indicates AFH).

The objectives of this study were: 1) to evaluate the reliability of CTG for AFH diagnosis; 2) to determine how many SCs were indicated due to the presence of foetal hypoxia and how many due to false positive CTG tracings; 3) to suggest more accurate steps for AFH diagnosis.

MATERIALS AND METHODS

During 18 months in the 1997/98 period, 997 caesarean deliveries were carried out at the 1st Department of Gynaecology and Obstetrics, Faculty of Medicine, Masaryk University in Brno. Of these, 159 (15.95 %) were indicated due to AFH diagnosed on the basis of CTG tracing. However, the majority of newborn infants did not show any expected pathological findings.

A group of 100 patients (average age, 26.5; range, 16 to 40 years) were included in the study because they met the following criteria: 1) the presence of pathological or suspected pathological CTG findings suggestive of acute hypoxia of the foetus; 2) delivery by SC; 3) availability of information on gas analysis of umbilical artery blood.

A total of 105 infants were born to the 100 mothers. They were classified according to their blood pH values assessed immediately after birth as follows: group 1, pH lower than 7.0 indicating „serious acidosis“; group 2, pH 7.0–7.2 indicating „acidosis“; group 3, pH 7.2–7.3 indicating „normal state“.

Groups 1 and 2 were subsequently made into one group designated „acidosis“. This approach was found to be more suitable for the „yes“ or „no“ statistical analysis in which the following CTG parameters were included:

1) **Baseline tetree heart rate** (BTHR) measurement. Values of 110–150 beats/min were considered to be normal and values below 100 beats/min and above 170 beats/min were regarded as pathological (6±14). The average BTHR value was 139.1±15.3 beats/min (range, 90–190 beats/min). Most of the BTHR values were in the normal range. The BTHR values for the acidosis and normal groups are presented in *Table 1*. Differences between the groups evaluated by Student's *t*-test were not found statistically significant.

2) **Variability**. According to the oscillation interval (6,14) the following curves were distinguished: physiological including reduced variability and variability patterns, and pathological or suspected pathological including silent and saltatory patterns.

3) **Curve reactivity**. A reactive curve was regarded as physiological, non-reactive curves as suspected pathological or pathological.

4) **Decelerations** are short, persistent depressions of foetal heart rate, with BTHR values 10–15 beats/min, lasting less than 30 sec. Their classification is related to uterine contractions as follows: early decelerations (DIP I), foetal heart rate depressions coincide with uterine contractions; late decelerations (DIP II), depression occurs after a contraction; variable decelerations (DIP 0), heart rate depression has no relation to uterine contraction. DIP I was regarded as physiological, DIP II and DIP 0 as pathological or suspected pathological findings

The diagnosis of foetal hypoxia was verified on the basis of the Apgar score classification (Apgar score > 7, good condition; 4–6, risk of postpartum foetal depression; 1–3, serious risk; 0, stillborn child) and the analysis of umbilical artery blood gasses. The latter procedure was carried out with the use of an IL 1610 acid-base analyser.

RESULTS

1) Baseline tetree heart rate. The mean BTHR value in our sample was 139.1 ± 15.3 beats/min, with a range of 90–190 beats/min. The mean values for each group of newborns are presented in *Table 1*.

2) Variability. *Table 2* shows the occurrence of variability types, expressed as percentages, in individual groups. Interestingly, in the infants with acidosis there was a high occurrence of normal patterns (reduced variability and variability). For

Table 1

Baseline tetree heart rate frequency values (mean \pm SD) in the neonatal groups.

Serious acidosis	138.33 ± 17.32 beat/min
Acidosis	141.03 ± 16.47 beat/min
Normal	136.42 ± 35.52 beat/min
Acidosis	139.68 ± 14.44 beat/min
Normal	136.42 ± 35.32 beat/min

Table 2

Variability patterns (in %) in the neonatal groups

Pattern	Serious acidosis	Acidosis	Normal	Total
Silent	4.76	23.81	71.43	100
Reduced variability	13.33	24.44	62.22	99.99
Variability	6.52	30.44	63.04	100
Saltatory	20.0	10.0	70.0	100

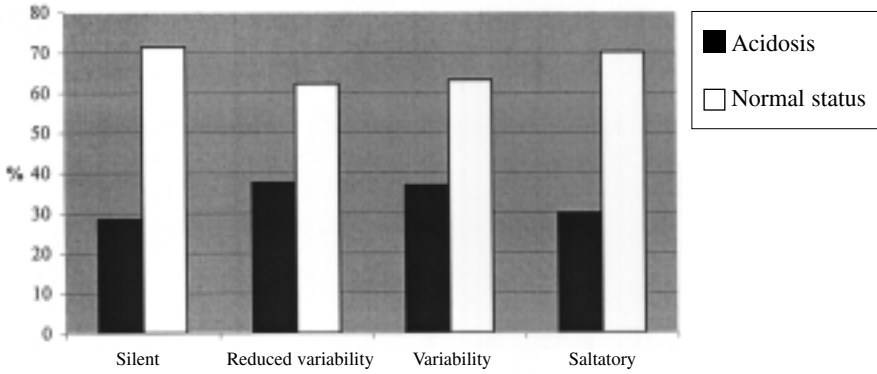


Fig. 1
Predictive value of variability patterns in the final groups

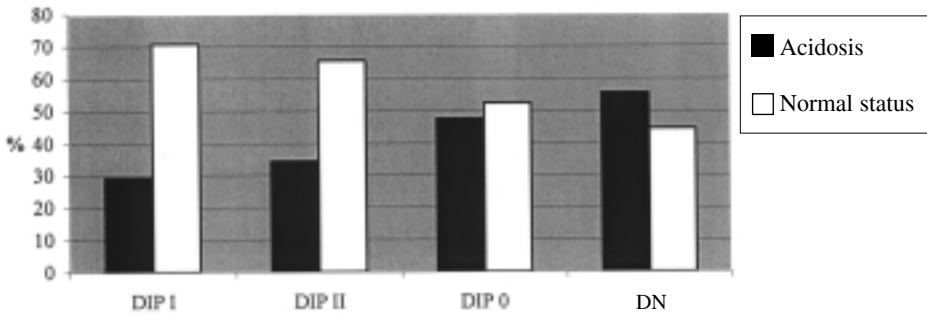


Fig. 2
Types of decelerations in the neonatal groups. DN, decelerations at normal tonus

Table 3
Reactivity of curves (in %) in the neonatal groups

Curve	Acidosis	Normal	Total
Reactive	37.23	62.77	100
Non-reactive	27.27	72.73	100

Table 4
Types of decelerations (in %) in the neonatal groups

Type	Acidosis	Normal	Total
DIP I	29.27	70.73	100
DIP II	34.48	65.52	100
DIP 0	47.62	52.38	100

Table 5
Average Apgar scores in the initial groups and their mean \pm SD in the final classification

Group	1-min Apgar score	5-min Apgar score	10-min Apgar score
Serious acidosis	5.44	7.22	7.89
Acidosis	6.83	7.76	8.41
Normal	7.87	8.76	9.04
Acidosis	6.50 \pm 2.05	7.63 \pm 2.83	8.29 \pm 2.96
Normal	7.87 \pm 1.72	8.76 \pm 1.43	9.04 \pm 2.36

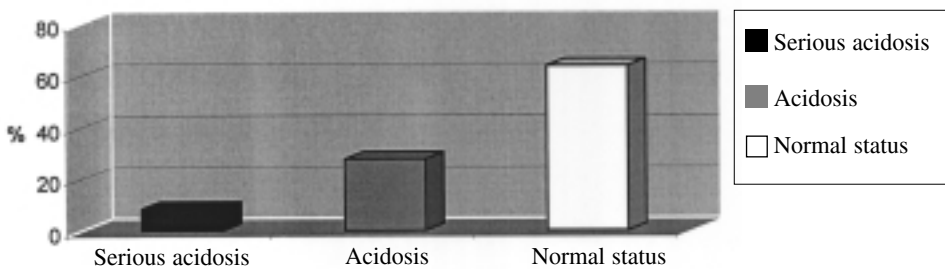


Fig. 3
Distribution of newborn babies, according to pH values of umbilical artery blood, into three initial groups (in %)

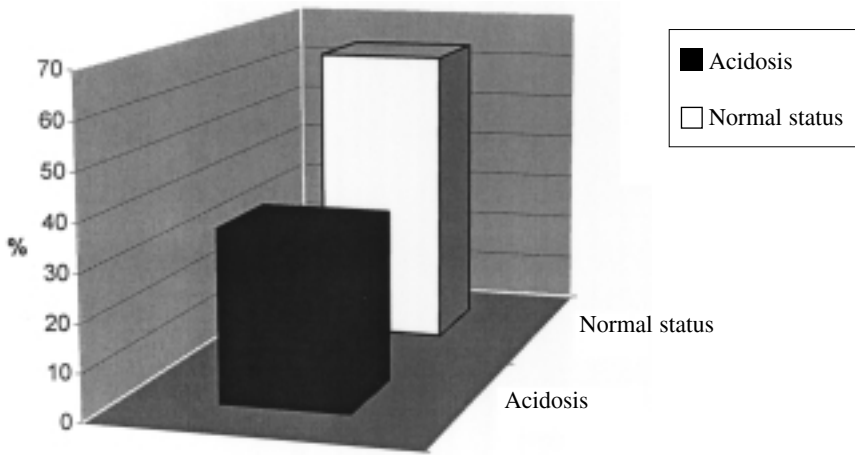


Fig. 4
Distribution of newborn babies in the acidosis and the normal group.

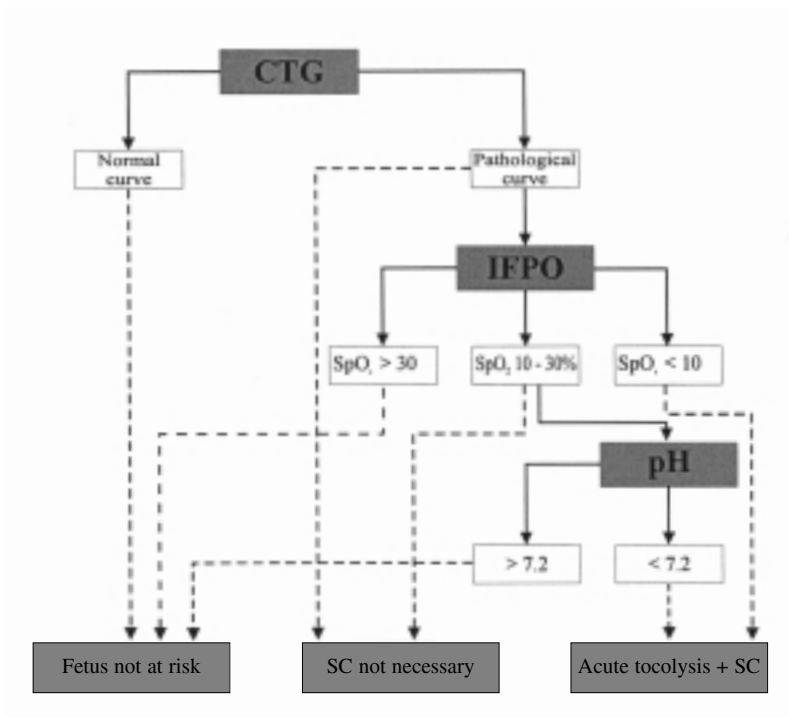


Fig. 5
Procedure (algorithm) suggested to make the diagnosis of acute foetal hypoxia more accurate.

Table 6
pH values of umbilical artery blood in the initial and final groups

Group	pH	No. of newborns	% of newborns
Serious acidosis	<7.0	9	8.57
Acidosis	7.0 - 7.199	29	27.62
Normal	7.2 – 7.34	67	63.81
Acidosis	<7.0 – 7.199	38	36.19
Normal	7.2 – 7.34	6	63.81

each type of variability, its predictive value was calculated. A low predictive value for the silent curve (28.6 %) indicated a high degree of probability that a hypoxic baby would be born. Saltatory curves were also found to have a low predictive value (30%) (*Fig. 1*).

3) Curve reactivity. The values for each group of newborn infants are in *Table 3*. The results show that, paradoxically, there was again a higher occurrence of non-reactive curves in the group of healthy infants than in the group of acidotic infants, and a higher occurrence of reactive curves in the group of babies with acidosis than in the healthy babies. When a reactive curve was present, the probability that a hypoxic infant would be born was calculated to be 27.3 %.

4) Decelerations. The highest occurrence of all three DIP types was found in the group of normal infants (*Table 4, Fig. 2*). The presence of DIP II, as an CTG indicator of acute foetal hypoxia, had a very low predictive value (34.5 %). When DIP O was detected, the probability that a hypoxic infant would be born was 47.6 %. It was interesting to find that, when DIP I (physiological type of deceleration) was present, the probability that the newborn infant would be hypoxic was 29.3 %.

Foetal hypoxia verification. The average values of Apgar scores were: 1-minute score, 7.37 ± 1.96 ; 5-minute score, 8.35 ± 2.58 ; 10-minute score, 8.77 ± 2.62 . The data for each group are shown in *Table 5*. The only statistically significant results were observed between the acidotic and the normal group at 1 and 5 min. Foetal blood gas analysis showed that the average pH value of umbilical artery blood was 7.22 ± 0.12 (range, 6.91–7.47). The distribution of newborn infants into the relevant groups according to their postpartum pH values is shown in *Table 6* and *Fig. 3*. In the 105 infants who were suspected of having acute foetal hypoxia, only 38 (36.2%) were born with this condition (*Fig. 4*). The most important finding was that 63.8 % of them, regardless of CTG tracing, were born healthy.

The drawing in *Fig. 5* shows a procedure consisting of several methods that is suggested by the author to reduce the risk of false positive findings in AFH diagnosis and, consequently, the risk of unnecessary caesarean delivery.

DISCUSSION

Cardiotocography is a method of prepartum and peripartum examination which has been established as the routine standard for foetal surveillance (15). Although this monitoring shows a considerable rate of false positive results and its evaluation is very subjective, it has also some advantages. It is completely non-invasive, is available under any hospital situation (out-patient care delivery room, etc.), is easy to operate, it is possible to use it in the absence of an obstetrician and, especially, it is financially accessible. But its low validity due to almost 64% of false positive results stands against these advantages. In spite of this fact, CTG will probably remain the screening method for the diagnosis of acute foetal hypoxia, also because there has not been any other, more objective, non-invasive prepartum technique (5). In the future, it will be desirable to supplement CTG with other diagnostic methods, such as intrapartum foetal pulse oximetry (IFPO) (3,14) or scalp blood analysis, which can quickly assess the clinical condition of the foetus and prevent a possible, unnecessary surgical intervention (10,11). Even if CTG findings are pathological and indicate foetal hypoxia with respiratory and metabolic acidosis, the infant can survive about 10 min of oxygen deprivation without serious consequences. If oxygen supply is not available for about 25 min, the foetus can still be resuscitated, but hypoxic damage to numerous organs including the heart may be so severe that death usually occurs within a short time (1,7,8). For effective diagnosis of foetal hypoxia, the 10-minute period can be used to insert a transmission sensor of pulse oximetry, which is a very valid monitoring of arterial oxygen saturation (13), or to make a bedside acid-base analysis. However, these examinations have also their contraindications in patients who, for instance, have intact membranes, low placental insertion, partial placenta abruption, metrorrhagia of an unknown origin, genital infection or uterine malformation. In order to increase foetal oxygen saturation in these situations, we can administer a therapeutic oxygen supply to the mother to ensure oxygen saturation for her baby during the critical period of time .

Although CTG examination is, in most obstetric wards, the only „objective,, method of AFH diagnosis, our results show that it is not a method accurate valid to determine whether hypoxia is present or not. In our study, it showed a high number of false positive results. Out of 100% of pathological or suspected pathological cardiotocograms, only 36.2 % were valid (i.e., the infants had hypoxia depression after birth). The remaining 63.8 % of infants, although their CTG findings were suggestive of AFH, were born healthy and SC was probably not necessary. Therefore, it appears that the truly objective evaluation of foetal

intrauterine condition and correct indication for SC should, in the future, be sought with the use of other diagnostic methods for AFH such as IPFO or scalp blood analysis.

ACKNOWLEDGEMENTS

I wish to thank Prof. MUDr. Aleš Roztočil, CSc. for his supervision of my project and help with writing this paper, and to Prof. MUDr. Pavel Ventruba, DrSc. for his valuable comments.

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VALIDITA CTG JAKO METODY DIAGNOSTIKY AKUTNÍ HYPOXIE PLODU

S o u h r n

AHP představuje jednu z nejzávažnějších patologií prenatálního a perinatálního období, která je obávaným faktorem časné či pozdní novorozenecké morbiditě či mortality. K diagnostice AHP se v současnosti rutinně využívá CTG vyšetření. Toto vyšetření, ačkoli má řadu předností – zejména neinvazivnost, snadnost obsluhy, vysokou dostupnost a finanční nenáročnost je charakteristické také výskytem velkého počtu falešně pozitivních výsledků. Na jejich základě se poté indikují císařské řezy, které tak neřeší novorozeneckou morbiditu, ale spíše zvyšují morbiditu i mortalitu mateřskou – téměř 64% novorozenců označených CTG vyšetřením za hypoxické se narodilo zcela v normě. Proto je doporučováno k optimalizaci a zvýšení efektivity diagnostiky AHP použít další metody, jako např. IPFO, nebo odběr krve z hlavičky plodu a předejít tak zbytečnému vystavení maminek případnému operačnímu riziku.

REFERENCES

1. *Brann AW, Myers RE.* Central nervous system findings in the newborn monkey following severe in utero asphyxia. *Neurology* 1973;25:327–38.
2. *Court DJ, Parer JT.* Experimental studies of fetal asphyxia and fetal heart rate interpretation. In: Nathanielsy PW, Parer JT, eds. *Research in Perinatal Medicine.* New York: Perinatology Press 1984:113.
3. *Čech E, Hájek Z, Maršál K, Srp B, et al.* Porodnictví [Obstetrics]. Praha: Grada Publishing, 1999:369–73.
4. *Herbst A, Wölner-Hansen P, Ingemarsson I.* Risk Factors for Acidemia at Birth. *Obstet Gynecol* 1997;90, 125–130.
5. *Low JA.* Intrapartum fetal asphyxia: Definition, diagnosis, and classification. *Am J Obstet Gynecol* 1997; 176: 957–9.
6. *Martius G, Breckwoldt M, Pfleiderer A.* Gynekologie a porodnictví [Gynecology and Obstetrics]. Martin: Vydavatelstvo Osveta 1996: 199 –255
7. *Myers RE.* Two patterns of perinatal brain damage and their conditions of occurrence. *Am J Obstet Gynecol* 1972; 112: 246–76.
8. *Myers RE, Beard R, Adamson K.* Brain swelling in the newborn rhesus monkey following prolonged partial asphyxia. *Neurology* 1969;19:1012–8.
9. National Center for Health Statistics. Annual summary of births, marriages, divorces, and deaths. *Monthly vital statistics report* 1993;42:13.
10. *Pearson J, Rees G.* Technique of caesarean section. In: Chalmers I, Enkin M, Keirse MJNC, eds. *Effective Care in Pregnancy and Childbirth.* Oxford University Press 1989:1234–1269.
11. *Nelson KB, Dambrosia JM, Tricia YT, Grether JK.* Uncertain value of electronic fetal monitoring in predicting cerebral palsy. *New Engl J Med* 1996;334:613–18.
12. *Parer JT, Livingston EG.* What is fetal distress? *Am J Obstet Gynecol* June 1990; 162:1421–23.
13. *Richardson B, Nodwell A, et al.* Fetal oxygen saturation and fractional extraction at birth and the relationship to measures of acidosis. *Am J Obstet Gynecol* 1998;178:572–579

14. *Roztočil A. a kol.*: Vyšetřovací metody v porodnictví a gynekologii [Diagnostic Methods in Obstetrics and Gynaecology]. Brno: Institut pro další vzdělávání pracovníků ve zdravotnictví, 1998:123–132.
15. *Shy KK, Larson EB, Luthy DA*. Evaluating a new technology: The effectiveness of electronic fetal heart rate monitoring. *Ann Rev Public Health* 1987;8:165–190.