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Solution of Controversy in Fetal Heart Rate Patterns

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Abstract

Aims: To solve the controversy in the subjective pattern classification of electronic fetal heart rate (FHR) monitoring.

Methods: FHR changes were quantitatively analyzed to calculate FHR score in the detection of abnormal outcome, instead of FHR pattern classification.

Results: Fetal outcome was abnormal when the FHR score determined in the 1st stage of labor was 10-19 and highly abnormal when the score was 20 or more.

Conclusion: Fetal outcome was predicted by a quantitative and objective evaluation by using FHR score. Further study was more objective neural network analysis published elsewhere. Various FHR controversies will be solved by the actocardiogram published in succeeding J Health Med Inform issue.

Keywords: Fetus; CTG; FHR pattern; Objective analysis; FHR score

Introduction

Fetal well-being was estimated by the enlargement of maternal abdomen, palpation of fetus on the abdomen, maternal perception of fetal kicking, and listening to fetal heart sound with obstetric stethoscope, in old age. Antique diagnosis of fetal life was fetal QRS found on limb lead electrocardiogram (ECG) studied by Cremer, which was progressed to fetal ECG amplifier composed of vacuum tube recorded on maternal abdomen and fetal phonocardiogram (PCG) in 1950s studied by EH Hon, R Caldeyro-Barcia, K Maeda and others. Listening to amplified fetal heart sounds using microphone was a method of intrapartum fetal monitoring, but it was unable to detect fetal sinusoidal heart rate, then fetal monitoring was shifted to electronic fetal heart rate (FHR) monitoring, initially recorded with direct lead fetal ECG through maternal abdominal and uterine walls by R Caldeyro-Barcia in 1950s, then shifted to FHR tracing with scalp lead fetal ECG by EH Hon in 1960. Since the effect of uterine contraction was emphasized, simultaneous tracing of intrauterine pressure was common. Although perinatal mortality, neonatal asphyxia and cerebral palsy reduced after introduction of intrapartum FHR monitoring [1-3], there remained controversy on HR patterns in the electronic fetal monitoring (EFM) and cardiotokography (CTG).

Electronic fetal monitoring and cardiotokogram

Edward H. Hon [4] created the EFM in USA, of which FHR curve was reciprocal interval of fetal QRS peaks, and labor contraction was recorded by intrauterine pressure. He reported a primary EFM machine in 1960, which recorded FHR and contraction curves on chart of 3 cm/min, and fetal ECG was recorded by another ECG speed recorder. The EFM was widely distributed in the world with Hon's classification of FHR deceleration patterns into early, late and variable decelerations, where the late decelerations, severe variable decelerations and the loss of baseline irregularity were fetal distress. R. Caldeyro-Barcia [5] classified the transient FHR decreases into Typ I and Typ II. Hon and Caldeyro-Barcia negotiated to agree that Typ II dip was the same as late deceleration in 1968. However, Hon did not reported the lag time of late decelerations diagnosed by visual observation, which was 20 or more sec in late decelerations later in the study of Chik et al. [6].

The Cardiotokogram was the external fetal monitoring of Konrad Hammacher [7] 1960s in Germany, which was composed of fetal heart rate triggered by fetal heart sound detected by a microphone, and labor contraction was recorded by external tocodynamometry. Single triggering signal was obtained from two fetal heart tones using a special algorithm. Fetal well-being was determined not only in the labor but also during pregnancy, that was unable by the EFM recorded by internal monitoring, although fetal QRS trigger enabled to study short term FHR variability. Hammacher also used FHR pattern classification but particularly emphasized the FHR variability (Oszillation), which was classified into normal, excessively high, and the loss of vaiability (Silente) was fetal distress. Various FHR pattern classifications were proposed by the world researchers.

The first production of FHR monitor in Japan

Maeda designed a FHR and contraction monitor in 1960s to trigger FHR curve by fetal heart sound, of which two tones were changed single trigger using a flip-flop multi-vibrator, or fetal scalp lead ECG (Figure 1). Uterine contraction was recorded by intrauterine pressure or external tocodynamometer, which detected the hardness of contracted uterus, and still it is used at present. The first fetal monitor, which enabled internal as well as external monitoring, was provided by TOITU (Tokyo) in 1964 (Figure 1) then distributed in Japan and other countries. Since Japanese obstetricians would not like the scalp clip electrode due to its invasive nature, instead they preferred fetal heart sound microphone, which was mandatory before the rupture of membrane and in antepartum monitoring. Ultrasonic external monitoring is the main technique in the FHR diagnosis at present.

The Japan Association of Maternal and Fetal Welfare (present Japan Association of Obstetricians and Gynecologists) promoted the fetal monitoring to members by designing an external FHR monitor using fetal heart microphone and tocodynamometer in 1970s, and manufacturers produced the machine, then the FHR monitoring was

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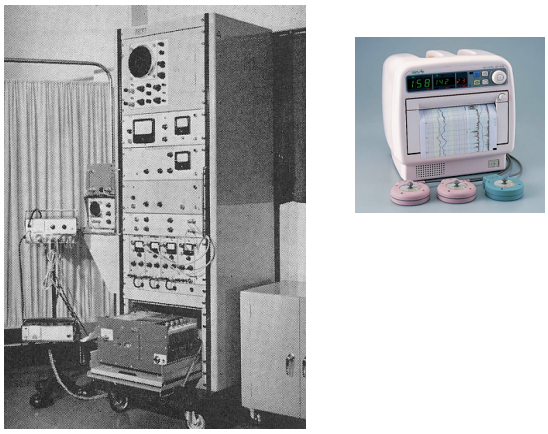

Figure 1: Japan first fetal heart rate monitor designed by Maeda and produced by TOITU (Tokyo) (left) and MT-516 actocardiograph for twins at present provided by TOITU (Tokyo) (right).

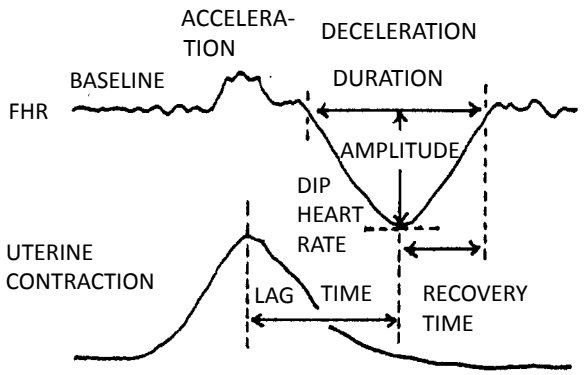

Figure 2: The quantitative analysis of FHR changes, of which data were used in the calculation of FHR score (Table 1).

widely distributed in Japan, and as the results, neonatal asphyxia, perinatal mortality and cerebral palsy decreased [1-3].

Diagnosis of nonreassuring Fetal States (NRFS, Fetal Distress) by FHR monitoring

Severe FHR changes appeared in CTG records before fetal demise in high risk pregnancies including pregnancy hypertension, preeclampsia, fetal growth restriction (FGR) and others. FHR changes prior to fetal deaths were the loss of FHR variability, bradycardia, prolonged decelerations, severe late decelerations, severe variable decelerations or sinusoidal FHR in 1970s. As the C-section was recommended when severe FHR abnormalities appeared during pregnancy, no fetal demise of unknown cause was experienced in our clinic. It was the merits of fetal monitoring.

Quantitative Nrfs diagnosis in the FHR monitoring

The classification of FHR patterns followed by their diagnosis of NRFS (fetal distress) was disseminated in the world; however there were inter observer differences in FHR pattern recognition, then more objective diagnostic method was desired.

Maeda intended to exclude the subjective pattern recognition from the fetal diagnosis by quantitative analysis of FHR changes (Figure 2) and to evaluate fetal condition with the scoring determined by the percentage of Agar score lower than 7 in non-inteventional labor. FHR curve received quantitative analysis (Figure 2), and the FHR score was calculated in the objective procedure (Table 1).

Sum of evaluation scores in 5 min is FHR score, which is the comprehensive index of the fetus, who is abnormal if FHR score is 10-19 points, and 20 or more is highly abnormal.

The largest FHR score (X) in the first stage of labor closely correlated 1min Apgar score (Y) and umbilical arterial blood pH (Z), i.e. neonatal Apgar score was 9 and umbilical arterial blood pH was 7.4 when the FHR score was 0, while Apgar score was 6 (mild neonatal asphyxia) when the FHR score was 10, and Apgar was 3 (severe asphyxia) and umbilical arterial blood pH was 7 (acidosis) when the first stage FHR score was 20. Further high FHR score resulted more severe neonatal asphyxia and severe acidosis (Table 2). Thus a rapid delivery was indicated when the FHR score was high even in early stage of labor. In continuous monitoring of a high risk pregnancy, Cesarean section was indicated to prevent fetal demise when the FHR score is high, because comprehensive fetal state is shown by FHR score (Table 2).

FHR score in the 1st stage of labor (X) closely correlated 1min Apar score (Y) and umbilical arterial pH (Z), i.e.

$Y=9.361-0.335X$, $R^2=0.71$, $N=20$

$Z=7.37-0.02X$, $R^2=0.99$, $p<0.001$.

Controversy in the CTG diagnosis

As the author pointed out, FHR pattern diagnosis was vague when the FHR pattern recognition is the diagnostic basis, i.e. there was the difference of pattern recognition among observers because the pattern was classified by subjective visual observation of CTG. Although the drawback was overcome by quantitative FHR analysis and numeric scoring, the development mechanism of FHR changes were unknown, and the cause of FHR acceleration, deceleration, bradycardia and variability were only supposition, but not based on facts, i.e. mild variable deceleration was decided to be caused by nervous reflex to cord compression but severe variable deceleration was hypoxia, all

FHR	Abnormal sign	Evaluation score
FHR baseline	110-130 or 160-180 bpm	1
	<110 or >180 bpm	3
Deceleration		
Duration	>60 sec	3
Amlitude	>50 bpm	2
Nadir heart rate	<100 bpm	2
Recovery time	>40 sec	3
Lag time	>40 sec	3
No associated acceleration		2
Loss of variability,	W-shaped dip	4

Table 1: Calculation of the FHR score.

Largest FHR score in the 1st stage of labor	1 min Apgar score	UApH
0	9	7.37
5	8	7.32
10	6 (asphyxia)	7.27
15	4 (asphyxia)	7.07 (acidosis)
20	3 (severe asphyxia)	6.97 (acidosis)
25	1 (severe asphyxia)	6.87 (acidosis)

Table 2: FHR score in the 1st stage of labor, Apgar score and umbilical arterial pH.

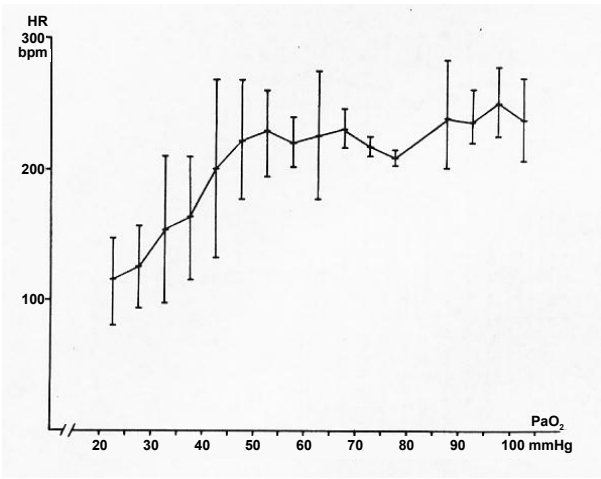

Figure 3: The adult rabbit heart rate did not change when the PaO_2 changed in the ranges higher than 50 mmHg, while the heart rate closely correlated to PaO_2 in the range lower than 50 mmHg. Since human fetal umbilical arterial PaO_2 is usually lower than 50 mmHg, the FHR is bradycardia in the hypoxia.

of late decelerations were decided ominous even in very mild late deceleration, developmental process of acceleration was unknown, the cause of bradycardia was fetal deterioration, baseline variability was told to be caused by the competition of sympathetic and vagal tones.

In the studies on heart rate (HR) and arterial blood PaO_2 of hypoxic adult rabbits induced by nitrogen gas inhalation, the heart rate did not correlated to PaO_2, if the PaO_2 was higher than 50 mmHg, while heart rate and PaO_2 closely correlated when the PaO_2 was lower than 50 mmHg (Figure 3) [8], and the umbilical arterial blood PaO_2 was lower than 50 mmHg [8], i.e. fetal bradycardia below 110 bpm was the sign of PaO_2 lower than 50 mmHg, and the bradycardia below 110 bpm was the sign of hypoxia. Since the bradycardia did not appear when the animal was anesthesized by urethane, the awake parasympathetic center of the medulla oblongata was excited by the hypoxia in normal state, i.e. fetal deceleration and bradycardia is caused by the hypoxia. Continuous bradycardia is caused by persisted hypoxia and therefore it is dangerous. Repeated nadirs lower than 100 bpm lasting longer than 1 min are also dangerous by repeated hypoxia in severe variable decelerations. Prolonged deceleration longer than 2 min is also dangerous by the prolonged hypoxia, while shorter duration of nadir than 1 min above 100 bpm is not dangerous in mild and short hypoxia in mild decelerations [9,10].

The other objective technique of the author was the artificial neural network analysis of FHR, which was more objective than FHR score [11]. In literatures, the empirical mode decomposition [12] and toward quantitative fetal heart rate monitoring [13] would be references of objective diagnosis.

Developmental mechanisms of physiologic sinusoidal FHR pattern, FHR acceleration and long term FHR variability (LTV) could not be explained using the CTG, but they could be discussed in the chapter of actocardiogram, using fetal movements and fetal brain reaction.

The FHR monitors utilized fetal heart sound microphone in the external monitoring. However, after introduction of ultrasonic Doppler fetal heart detector, its introduction into labor monitoring was requested, however, ultrasound safety to the fetus was not established, the ultrasound use was not allowed in FHR monitoring in 1970s. Since Maeda et al declared that the ultrasound, of which intensity lower than 240 mW/cm^2, had no effect to suppress cultured cell growth [14] and Japan Industrial Standard limited the medical diagnostic ultrasound intensity below 10 mW/cm^2 [15,16], the safety of medical ultrasound on the fetus was established. In addition, the disturbance of FHR baseline in the use of Doppler ultrasound in the instantaneous heart rate meter was solved by Takeuchi and Hogaki of Japan [17] to record the FHR curve as clear as the fetal scalp ECG by using the autocorrelation heart rate meter and Doppler fetal heart signals, all FHR monitor changed to ultrasound Doppler autocorrelation monitor not only in Japan, but also in the world. Thus, the FHR monitoring changed to external monitoring through pregnancy and delivery. This is the present state of FHR monitoring in Japan. Since the global standard of ultrasound safety is lower than 1.0 thermal index, where ultrasound intensity is close to but lower than 240 mW/cm^2 in 1.0 thermal index, thus the thermal index is accepted also in Japan.

Conclusion

As the FHR pattern classification method for the diagnosis of fetal NRFS (fetal distress) will not be recommended from the inter-observer difference of subjective FHR pattern recognition, instead quantitative analysis of FHR changes and FHR score calculated by objective FHR data are recommended, because the method predicts fetal outcomes even in the first stage of labor. The problem due to subjective FHR pattern recognition was solved by the objective FHR scoring, which also enables computerized analysis. The BASIC language program for computerized FHR diagnosis of the author was published in a Japanese book [10]. An automatic computerized system is provided by TOITU (Tokyo).

The other problems related to the origin of HR changes will be solved by the analysis of actocardiogram, which is simultaneous record of fetal movement and heart rate.

References

1. Maeda K (1979) Healthy baby and reduced fetal death by improved obstetric management.
2. In SMM Karim and KL Tan eds, Problems in Perinatology. Proceedings the 1st ASIA Oceania Congress of Perinatology, Univ of Singapore Kandang Kerbau Hospital, Singapore.
3. Tsuzaki T, Sekijima A, Maeda K, Morishita K, Takeuchi Y, et al. (1990) The survey on the perinatal variables and the incidence of cerebral palsy for 12 years before and after the application of the fetal monitoring system. Acta Obstet Gynecol Jpn 42: 99-105.
4. Takeshita K, Ando Y, Otani K, Takashima S (1989) Cerebral palsy in Tottori, Japan. Benefits and risk of progress in perinatal medicine. Neuroepidemiology 4: 184-192.
5. Hon EH (1968) An Atlas of Fetal Heart Rate Patterns. Harty Press New Haven.
6. Caldeyro-Barcia R, Poseiro JJ, Gulin LO (1967) Effects of abnormal uterine contractions on fetal heart rate during labor. Proc, The 5th World Cong Gynaecol Obstet, Sydney 9-29.
7. Chik L, Rosen MG, Hirsch VJ (1974) An analysis of lag time and variability of fetal heart rate measurements. Am J Obstet Gynecol 118: 237-242.
8. Hammacher K (1976) Einf Ührung in die Cardiotokographie. Die Scweizer Hebamme 74: 6-12.
9. Umezawa J (1976) Studies on the relation between heart rate and PaO_2 in hypoxic rabbit: a comparative studyfor fetal heart rate change during labor. Acta Obstet Gynecol Jpn 28: 1203-1212.
10. Maeda K, Kimura S, Nakano H, (1969) Pathophysiology of Fetus, Fukuoka printing, Fukuoka, 21st Annual Conv Japn Soc Obstet Gynecol, Kanazawa.
11. Maeda K (2013) Actocardiogram Tokyo Medical View.

12. Maeda K, Utsu M, Makio A, Seriawa M, Noguchi Y, et al. (1998) Neural network computer analysis of fetal heart rate. J Matern Fetal Fetal Invest 8: 163-171.

13. Liu Y, Wei S, Liu XN (2012) Nonlinear FHR baseline estimation using empirical mode decomposition and Kohonen neural network. IEEE biomedical circuits and systems conference (BioCAS) 368-371.

14. Cao H, Lake DE, Ferguson JE2nd, Chishlom CA (2006) Toward quantitative fetal heart rate monitoring. IEEE Trans on Biomedical Engineering 53: 111-118.

15. Maeda K, Murao F, Yoshiga T, Yamauchi C, Tsuzaki T (1986) Experimental studies on the suppression of cultured cell growth curves after irradiation with CW and pulsed ultrasound. IEEE Trans Ultrasonics. Ferroelectrics and Frequency Control 33: 186-193.

16. Maeda K, Ide M (1986) The limitation of the ultrasound intensity for diagnostic devices in the Japanese Industrial standards. IEEE Trans Ultrasonics Ferroelectrics and Frequency Control 33: 241-243.

17. Takeuchi Y, Hogaki M (1975) Auto correlation method for fetal heart rate measurement from ultrasonic Doppler fetal signal. In D. White, R Brown eds Ultrasound in Medicine Vol 3B Engineering aspects Plenum Press, New York.