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Prevention of cerebral palsy with hypoxia index in fetal monitoring

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Abstract
Although fetal deaths was decreased by intrapartum fetal heart rate (FHR) monitoring, infantile cerebral palsy was not decreased in Dublin trials of electric fetal monitor, thus, an analysis to prevent cerebral palsy was studied, where cerebral palsy is prevented by setting the threshold of hypoxia index at 24 or less, in the analysis of FHR deceleration.

Keywords: fetal heart rate monitoring, late deceleration, hypoxia index, cerebral palsy, numeric threshold

Introduction

Electronic fetal monitors (EFM) were internal method using fetal scalp ECG electrode and intrauterine pressure tracing, where fetal heart rate (FHR) monitoring started limitedly after the rupture of membrane, while external fetal heart rate (FHR) monitor with fetal heart sound was performed by Hammacher and Maeda, and now mainly by ultrasonic Doppler autocorrelation fetal heart rate meter is distributed in the world, and the uterine contraction by tocodynamo meter. Although hypoxic FHR decelerations (transient bradycardia) were divided
classified into early, late and variable decelerations with visual observations, where visually diagnosed fetal late deceleration of FHR were ominous by Hon [1], and the loss of baseline variability was ominous in Hon & Hammacher's FHR diagnosis [1, 3], where it was reported to be irreversible fetal brain damage followed by cerebral palsy [2]. The separation of pathologic and physiologic sinusoidal FHR was unable by CTG, while it was possible by actocadiogram [4] and FHR frequency spectrum analysis by Ito *et al* [9]. FHR pattern classification was vague in several occasions, e.g. a variable deceleration was diagnosed as "variable and early deceleration" by Dr. Hon himself. Thus, objective numeric analysis is required in the intrapartum fetal monitoring, including the artificial neural network [6] or FHR score [5] in the update trend to introduce quantitative FHR analysis. The hypoxia index showed clear differentiation in the present report.

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Methods

Stable FHR

The FHR of resting fetus is an example of stable FHR, which is composed of around 120 bpm heart rate, normally overlapped by small irregular FHR variability, of which amplitude is 2-5 bpm. There are transient Tachycardia, that is FHR rise more than 200 bpm for 5 min, which was supposed to be fetal intraputerine cry. Mild continuous FHR rise is found in continuous fetal respiration and fetal hiccupping motion. Fetal respiratory movement is 1 cps spikes. Continuous 2 sec interval spikes are fetal hiccupping movements which also continuous 3 bpm FHR spikes, but not accompany FHR acceleration due to absence of fetal movement burst.

FHR decrease

Common FHR patterns were early, late, mild & severe variable decelerations, in fetal outcome discussion in visual analysis, where no numeric criteria was shown even in the delay time of late deceleration [1], which was more than 20 sec by Check *et al* later. Thus, the diagnosis with FHR pattern classifications was controversial even in the memory of the author. Thus, the author firstly created objective FHR score in 1969 [5], by which predicted Apgar score and UApH [6], then fetal outcome diagnosis with artificial neural network analysis [6]. The author created Actocardiogram, which analyses fetal heart rate with fetal movement, and clarified the relation between fetal heart rate and movement [8], where triangular FHR acceleration is originated in fetal motion created in the midbrain [7], which changed square shape fetal movement burst to triangular FHR with the integral function of mid brain with 7 sec delay time. The simulation of adult also developed triangular heart rate from square shape leg movements [8], Thus, it was clear that FHR increases by the fetal brain is excited by fetal movements. The function clarified developing mechanism of physiologic sinusoidal fetal heart rate, where the physiologic sinusoidal is the synchronization of fetal brain to fetal cyclic mouthing or respiratory movements, which separates physiologic sinusoidal FHR from pathologic sinusoidal one, which accompanied no fetal movement [10].

As fetal brain function to rise heart rate is suppressed by the hypoxia, where firstly FHR acceleration is lost in "non-reactive heart rate", while FHR baseline variability is present in mild hypoxia, thus, the baseline variability was the final response of fetal brain to minor fetal movements, then the variability is lost in the most severe irreversible hypoxic fetal brain damage. Thus, fetal damage before the loss of FHR baseline variability, thus, the delivery after the loss of variability cures fetal life but unable to early caesarean delivery is performed with the purpose to prevent early caesarean delivery is performed with the purpose to prevent severe hypoxia [11], while the threshold was unknown in the past, but it will be discussed in the present report.

FHR Fall

FHR falls in fetal hypoxia, where the vagal nerve center located in medulla oblongata is excited by the hypoxic stimulation to reduce FHR, then develop fetal bradycardia in a long hypoxia, thus, FHR deceleration develops in transient hypoxia, namely, hypoxic fetal bradycardia is a vagal nerve excitation, but not immediate fetal damage, namely, frequently repeated decelerations or prolonged fetal bradycardia develops fetal brain damage, which is shown by hypoxia index as follows.

Composition of Hypoxia Index

FHR was parallel to lower PaO_2 than 50 mmHg in experimental hypoxia produced in female rabbit by inhallation of N_2 gas by female rabbit, and fetal PaO_2 was lower than 50mmHg [10], The heart rate was estimated to be bradycardia in hypoxia, of which duration (min) was determined in intrapartum CTG, and the sum of hypoxic bradycardia durations were divided by the lowest FHR to add the intensity of hypoxia, then multiplied by 100 to keep the index to be integer, namely,

Hypoxia index=the sum of deceleration durations (min) / the lowest FHR and multiplied by 100

Threshold of Hypoxia Index to Damage Fetal Brain

It was mandatory to set quantitative numeric threshold to prevent fetal brain damage, which was shown by the loss of baseline variability, while there was no threshold in fetal monitoring in the past, thus, the author tried to study late FHR deceleration, looking for its hypoxic effect. The author experienced a case of 3 connected typical late decelerations with 45 sec lag time, who was born as fully normal neonate after caesarean delivery, where the Apgar score was 9, while a case of late decelerations repeated for 50 minutes due to refusal of caesarean delivery developed severe neonatal asphyxia of which Apgar score was 3, associated severe brain damage, which ended by the death in brain hemorrhage, namely, fetal typically characteristic late deceleration repeated 3 times dose not develop fetal damage, while frequently repeated decelerations for 50 minutes developed Apgar 3 brain damaged neonate. In addition, there was another definition of experts group, that was a late deceleration was diagnosed limitedly after 15 minutes repetition of late decelerations, Thus, it is confirmed that frequent repetition of decelerations are effective to develop hypoxic damage, due to frequent hypoxic fetal damage, but not by the characteristic late deceleration pattern, Thus, the author decided to clinically confirm the threshold level by the hypoxia index, which was defined by the repetition of decelerations.

Namely, the novel hypoxia index = The sum of all deceleration durations (min) in full course of fetal monitoring, divided by the lowest FHR (bpm),which indicates the intensity of hypoxia, then multiplied by 100 to keep the index to be integer (Figure 1). It was believed that fetal brain damaging threshold exists between 3 FHR decelerations and 15 minutes repetition of decelerations, which is detected by Hypoxia Index.

Results and Discussion

We collected retrospectively 22 cases of infants who were diagnosed by pediatric clinic, and their intrapartum FHR records were obtained in obstetric ward, where 6 cases were cerebral palsy and 16 cases were not cerebral palsy. Hypoxia index of the two groups showed significant difference with $p<0.000008$, and no cerebral palsy was followed, if Hypoxia Index is 24 or less (Table 1).

Hypoxia index (HI) of all 6 cerebral palsy cases was 25 or more, but no cerebral palsy developed in 24 or less HI, namely, the index of all 16 no cerebral palsy cases was 24 or less but 0 in 25 or more HI. The numeric threshold existed between 24 and 25 of hypoxia index (Table 1). The chi square test p was 0.000008, almost zero, and there was significant difference between the HI 25 or more and 24 or less, so that we concluded there is no cerebral palsy, if HI is less than 24.

As there was no cerebral palsy in 16 cases, whose hypoxia index was 24 or less, and diagnostic error was almost zero, it would true that cerebral palsy is prevented, if the HI is kept at the level of 24 or less in fetal monitoring.

The cases whose hypoxia index is 25 or more can receive early treatment of cerebral palsy even in neonatal period, as it is possible to develop cerebral palsy, if the index is 25 or more. Early treatment may improve the therapeutic effect.

As late deceleration develops to stop placental maternal blood flow by the compression of pelvic artery by contracted pregnant uterus in supine posture, and late deceleration disappeared by changing maternal posture to lateral one from supine [11, 12], the author recommends to change maternal posture to lateral one to reduce hypoxia index, when any deceleration appears during labor.

As the hypoxia index covers the area of deceleration pattern classification, including early, late and variable decelerations, thus, FHR diagnosis is changing to quantitative logical ones from visual FHR deceleration pattern classification.

As Cahill *et al* [13] also reported to predict UApH with FHR deceleration area, novel quantitative logical diagnosis of clinical outcome by fetal monitoring will change fetal monitoring to computerized system instead of human visual analysis of FHR records.

FHR Score

FHR score is numeric score to evaluate fetal deceleration, against FHR pattern classification method, published by Maeda in 1969 [5], where FHR decelerations in 5 min are analyzed and 9 deceleration parameters were evaluated its abnormality by evaluation scores, which were determined according the percentage of lower Apgar score than 7.

Evaluation scores were summarized in 5 min, that was FHR score, which was abnormal when it is 10, and 20 is heavily abnormal. Apgar score is 0 (died) when FHR score is 24, in regression equation. Also the fetus will die if Hypoxia Index is 37.

Pathologic sinusoidal FHR

It is caused by severe fetal anemia due to fetal hemorrhage to the mother, thus, fetal death is imminent. Experimentally, heavy rabbit hemorrhage showed sinusoidal heart rate (Hidaka).Physiologic benign sinusoidal FHR is FHR synchronization to periodic fetal respirations or sucking actions. As no therapy is needed, the fetus is separated from pathologic sinusoidal by actocardiogram (Figures 2 & 3) but not by CTG. FHR frequency spectrum diagnosed pathologic sinusoidal FHR. Fetus is delivered by early caesarean and receives transfusion. Fetal transfusion is performed, if possible.

Conclusion

As intrapartum FHR rise is caused by fetal brain response to fetal movements, and FHR fall is caused by fetal hypoxia, thus, it is mandatory to study FHR rise due to fetal movements and its fall due to hypoxia, do not only rely to 4 FHR patterns, but rely on numeric FHR rise including FHR baseline variability, which is fetal brain response to fetal movements, and hypoxia, which means low PaO_2, low oxygen content, which is shown by transient deceleration or continuous bradycardia, which damages fetal brain if it is repeated or continuous, thus numeric FHR analysis is important, where the rise and fall of FHR should be correctly analyzed according to facts but not by subjective FHR pattern, The revolution started by author's hypoxia index and Cahill's deceleration area [13]. They are examples of update modern FHR diagnosis, as FHR is diagnosed by numeric threshold, for example, no cerebral palsy will befollwed, if Hypoxia Index is 24 or less, where it does not need visual training of FHR decelerations, but possibly needs computerized index calculation.

Table 1: Correlation of FHR & neonatal scores

	Hypoxia	FHR	Apgar	Fetal arterial
	Index	score	score	pH (UApH)
	16	10	6	7.27
R>0.8	19	12	5	7.20
P<0.05	23	15	4	7.06
	24	20	3	6.08
	37	24	0 (death)	

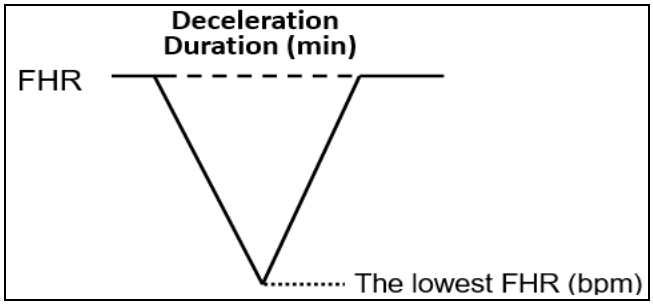

Fig 1: How to measure deceleration duration and the lowest FHR

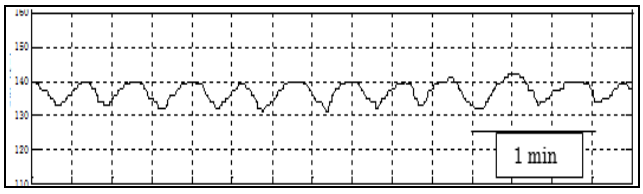

Fig 2: Pathologic sinusoidal FHR, which did not accompany fetal movement, but followed by fetal death.

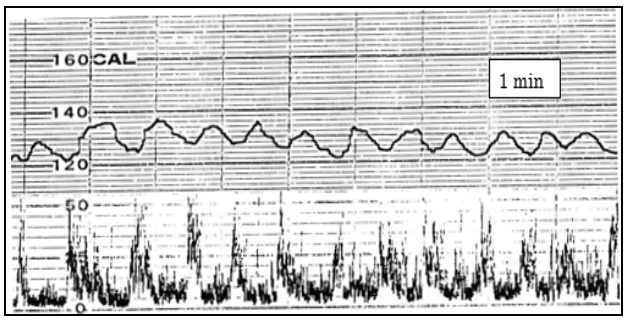

Fig 3: A physiologic sinuaidal FHR (upper line), which synchronized the lower line, which is repeated cyclic fetal respiratory movements. Recorded by Actocardiogram

Table 2: Frequency of cerebral palsy was 6 / 6 (100%) in 6 cases of 25 or more of hypoxia index, while it was 0 / 16 (0%) in 16 cases whose hypoxia index was 24 or less. There was significant difference of the number of cerebral palsy in two groups (p=0.000008<0.05).

Hypoxia index	Total NN of no cerebral palsy	N of cerebral palsy	
25 or more	6	0 (0%)	6 (100%)
24 or less	16	16 (100%)	0 (0%)

Chi square test error p=0.000008 (1/1000.00), there is no error, if it is diagnosed no cerebral palsy, when Hypoxia Index is less than 24.

References

1. Hon EH. An Atlas of Fetal Heart Rate Patterns. Harty Press, New Haeven, 1968.
2. Maeda K. Modalities of fetal evaluation to detect fetal compromise prior to the development of significant neurological damage. JOGR. 2014; 40:2089-2094.
3. Hammacher K. Einfuehrung in die Cardiotokograph, 2.Die Schweizer Hebamme74 Jahrgang, Nr. 1076; 6:6-8.
4. Maeda K. Actocardiogram, Analysis of Fetal Motion and Heart Rate Jaypee, New Delhi, 2016.
5. Maeda K, Kimura S, Nakano H *et al*. Pathophysiology of Fetus, Fukuoka Printing, Fukuoka, 1969.
6. Maeda K, Noguchi Y, Matsumoto F, Nagasawa T. Quantitative fetal heart rate evaluation without pattern classification: FHR score and artificial neural network analysis. In Kurjak, Chervenak Eds. Textboook of Perinatal Medicine, 2nd ed. London, Informa, 2006, 1487-1495.
7. Terao T, Kawashima Y, Noto H, Inamoto Y, Lin TY *et al*. Neurological control of fetal heart rate in 20 cases of anencephalic fetuses. Am J Obstet Gynecol. 1984; 140:201-208.
8. Maeda K. Actocardiographic analysis of fetal hypoxia detected by the bradycardia, loss of fetal heart rate acceleration and long term variability. Health & Med Inform. 2013; 4:118-122.
9. Ito T, Maeda K, Takahashi H *et al* 1994. Differentiation between physiologic and pathologic sinusoidal FHR pattern by fetal actocardiogram. J Perinat Med. 1994; 22:39-48.
10. Umezawa J. Studies on the relation between heart rate and PaO_2 in hypoxic rabbit: a comparative study for fetal heart rate change in labor. Acta Obstet Gynecol Jpn. 1975; 28:1203-1212.
11. Caldeyro-Barcia R, Poseiro JJ, Mendez-Bauer C, Gulin LO. Effects of abnormal uterine contraction on fetal Heat rate during labor. Proc. 5th World Cong Gynecol Obst, Sydney, 1967.
12. Poseiro JJ, Mendex-Bauer SV, Caldeyro-Barcia R. Effect of Uterine contractions on maternal blood flow through the placenta. 8 PAHO Advisary Committee on Medical Research, 1969, 161-171.
13. Cahill AG, Tuuli MD, Stout MJ, *et al*. A prospective cohort study of fetal heart rate monitoring: deceleration area is predictive of fetal academia, 2018.