Research Article

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Novel Hypoxia Index in Fetal Heart Rate Monitoring

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ABSTRACT

Aims: *Creation of novel objective & numeric fetal hypoxia index, solving controversy pattern classification, preventing cerebral palsy caused by intrapatum fetal brain damage before definite threshold.*

Methods and Results: *Hypoxia index (HI) is the sum of deceleration duration (min) divided by the lowest FHR (bpm), and multiplied by 100. As the HI was 25 in a case of abnormal FHR followed by the cerebral palsy (CP), and 26 in a case of repeated late decelerations (LD) for 50 min with the loss of variability, Apgar 3, and brain damage, while abnormal FHR whose HI was 20-24 developed neither loss of variability nor CP. The HI was 6, Apgar score was 9 and no CP in a case of 3 connected LDs. Apgar score was predicted by a regression equation.*

Discussion: *LD is ominous because of its high repetition producing high HI, while 2-3 LD repetition develops no ominous outcome. The principle can be adapted to the other decelerations and sudden continuous fetal bradycarda. Computerized FHR diagnosis is simplified introducing hypoxia index, rejecting subjective pattern classification and early delivery before reaching 25 of HI will prevent the brain damage and CP.*

Conclusion: *The late, early and variable decelerations and sudden FHR bradycardia will be followed by neither fetal brain damage nor CP, if the HI was lower than 25 during delivery, while normal neonate without CP will not be expected if fetal HI was 25 or more, despite fetal death was prevented. HI is the most effectively applied in computerized FHR monitoring, at the same time with the FHR score, FHR curve frequency spectrum and A/B ratio.*

Keywords

Fetus, FHR, Decelerations, Variability, Hypoxia index, FHR Score, Apgar score, Cereblal palsy, Early delivery.

Introduction

Fetal outcome of late deceleration (LD) is uncertain, namely, the outcome of its 2-3 repetition was favorable, however, LD repetition resulted severe neonatal asphyxia, Apgar was very low associating the loss of variability, infantile brain damage, where definite threshold to be ominous outcome was unknown. Thus, the author needed new LD evaluation.

Methods

As fetal bradycardia is caused by the excited parasympathetic nerve center with hypoxia, while there was no FHR change when the animal was anesthetized with Urethan [1], and apneic bradycardia disappeared and heart rate returned normal after infusion of oxygenated blood to anencephalic neonate [2], fetal bradycardia is only the sign of hypoxia in fetal environment, but it is not immediately the sign of fetal brain damage, while fetal brain damage is shown by the loss of FHR variability. Fetal brain reacts minor fetal movements showing FHR variability, thus the variability is lost after fetal brain damage followed by cerebral palsy due to prolonged hypoxia. As short duration of low PaO_2 does not affect fetal brain, but it is damaged by prolonged hypoxic exposure as shown in the development of hypoxic-ischemic encephalopathy. Therefore, hypoxic damage apears in hypoxia, which was detected by the sum of durations of repeated decelerations, where the heart rate (bpm) is used instead of PaO_2, because heart rate is highly correlated to PaO_2 when PaO_2 was lower than 50 mmHg [1], and fetal PaO_2 was less than 50 mmHg [2]. Thus, the duration of hypoxic exposure was determined by the sum of deceleration

duration, while hypoxic intensity was estimated by the inversion of nadir FHR, thus, the sum of deceleration duration (min) was divided by the lowest FHR (bpm), and multiplied by 100 to calculate the hypoxia index, namely, hypoxia index (HI) is the sum of deceleration duration divided by the nadir FHR, and multiplied by 100 [3]. The hypoxic exposure in HI is similar to the summated large dip area.

Results

The Apgar score after three connected LDs of 45 sec lag time was 9, where the HI was 6, while HI was 25 in a case of severe decelerations followed by the loss of variability and cerebral palsy, and in another case who repeated LD for 50 min associated by the loss of variability followed by severe brain damage, where the HI was 26, while HI was 20-24 in cases of abnormal FHR but neither followed by the loss of variability nor cerebral palsy [4]. Thus, upper limit to develop no loss of variability, no fetal brain damage followed by no cerebral palsy will be 24, and the lowest HI to develop the loss of variability and fetal brain damage followed by cerebral palsy will be 25 (Table 1).

Loss of variability, fetal brain damage and cerebral palsy	Yes	No
Hypoxia index = 25, 26	2	0
Hypoxia index = 20-24	0	5

Table 1: Chi square test of two HI groups. p= 0.048, significant difference.

Discussion

As the sum of durations in repeated LDs in HI, the theory of hypoxia index can be adopted not only LD, but also to all decelerations including early and variable decelerations, as well as the acute sudden continuous FHR bradycardia. Early deceleration was benign but it is caused not by the short lag time but short period of appearance passing through the birth canal. Therefore, even early deceleration can be evaluated by HI, if the time passing through birth canal is prolonged, where HI can be adapted.

Of course variable decelerations can be evaluated by the HI. Thus, all of FHR decelerations are evaluated by the HI, namely, HI covers all of pattern classification and in addition continuous sudden bradycardia can be evaluated by the HI. Therefore, the HI is applied in the FHR monitoring, not disturbing by the pattern classification, guided by the threshold not to be damaged by hypoxic effect. However, the HI should be continuously calculated evaluating continuously to avoid the summarized hypoxic effect, while application of a simple computer will solve the problem, then, the FHR score, FHR curve frequency spectrum analysis and A/B ratio [4] are simultaneously applied with the HI. Those are big advantage, as FHR score evaluates short 5 min FHR score, while long period is evaluated with the HI, FHR curve shape is analyzed by frequency spectrum [5,6] and A/B ratio [4]. Also, pathologic result obtained by computer is alarmed by expected Apgar score & UApH by cell phone or LAN, to perform the mostly correct treatment in fetal monitoring; early caesarean delivery will be performed at 24 or less of HI. The timely C-delivery will be planned, if the loss of FHR acceleration and decreased amplitude of variability to 5 bpm in the actocardiogram, because these changes preceed severe fetal asphyxia in FGR cases [7,8].

Conclusion

The novel hypoxia index is useful not only to evaluate late deceleration, but also all decelerations without the vague classification of deceleration patterns, in addition continuous fetal bradycardia is evaluated by HI, predicting hypoxic ischemic encephalopathy. Additional necessary diagnoses will be the short term FHR score, pathologic sinusoidal FHR and A/B ratio in computerized fetal monitoring.

References

1. 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.
2. Maeda K, Kimura S, Nakano H, et al. Studies on fetal pathophysilogy. Acta Obstet Gynecol. 1969; 8: 877-886.
3. Maeda K. Modalities of fetal evaluation to detect fetal compromise prior to the development of significant neurological damage. JOGR. 2014; 40: 2089-2094.
4. Maeda K, Iwabe T, Ito T, et al. Detailed multigrade evaluation of fetal disorders with the quantified actocardiogram. J Perinat Med. 2009; 37: 392-396.
5. Maeda K, Nagasawa T. Automatic computerized diagnosis of fetal sinusoidal heart rate. Fetal Diag Ther. 2005; 20: 3328-3233.
6. Maeda K, Nagasawa T. Loss of FHR variability diagnosed by frequency analysis. J Perinat Med. 2010; 38: 197-201.
7. Poseiro JJ, Mendex-Bauer SV, Caldeyro-Barcia R. Effect of uterine contractions on maternaty blood flow through the placenta. PAHO Advisary Committee on Medical Research. 1969; 161-171.
8. Teshima N. Non-reactive pattern diagnosed by ultrasonic Doppler fetal actocardiogram and outcome of the fetuses with non-reactive pattern. Acta Obstet Gynecol Jpn. 1993; 45: 423-430.