

Importance of Hypoxia Index in FHR Monitoring

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Abstract

Aim: Numeric objective evaluation of FHR deceleration.

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 FHR decelerations followed by the loss of variability and cerebral palsy (CP), and the HI was 26 in a case of repeated late decelerations for 50 min with the loss of variability, Apgar 3, and brain damage, the HI below 25 at delivery would be safe, showing neither brain damage nor CP, namely, the HI of 3 connected late decelerations was 6 and Apgar was 9, without CP. Also HI values of repeated abnormalities were 20-24 preserving the FHR variability, without brain damage.

Conclusion: The late, early, mild and severe decelerations and sudden acute FHR bradycardia would not be followed neither by brain damage nor CP, when the variability is preserved, and the HI is lower than 25. Thus, it is recommended to decide early caesarean delivery, when the HI is 20 or less, considering the time to prepare the surgery, while no normal neonate may be guaranteed by the caesarean delivery performed after the higher hypoxia index of 25 or more with the loss of FHR variability. The other sign of ominous outcome will be pathologic sinusoidal FHR, which mean severe fetal anemia

Keywords: Fetus; Hypoxia index; FHR deceleration; Late deceleration; Variable deceleration; Acceleration; Variability; Cerebral palsy; Apgar score; Long term FHR variability

Introduction

Fetal outcome of late deceleration (LD) is uncertain in fetal monitoring, namely, the outcome of its 2-3 repetition was favorable, however, LD repetition resulted severe neonatal asphyxia, associating the loss of variability and infantile brain damage, where Apgar was 3, while the 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 [1], and apneic bradycardia disappeared and heart rate returned normal after infusion of oxygenated blood to anencephalic neonate [2], thus, fetal bradycardia is only the sign of environmental hypoxia but not the sign of fetal brain damage, which is the loss of FHR variability. As short duration of low PaO₂ does not affect the brain, but the brain is damaged by long hypoxic exposure as shown in hypoxic-ischemic encephalopathy. Therefore, hypoxic effect was found in long duration of hypoxia, which was determined by the sum of durations of decelerations, where the heart rate (bpm) is used instead of PaO₂, because FHR is highly correlated PaO₂ when PaO₂ was lower than 50mmHg [1], and fetal PaO₂ was less than 50mmHg [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 of FHR, and multiplied by 100.

Results

The Apgar score of three LDs of 45 sec lag time was 6, where the outcome was normal, while HI was 25 and 26 in two fetuses of severe asphyxia followed by the loss of variability and cerebral palsy in a case, and in another case who repeated LD for 50 min associated by the loss of variability followed by 3 months infant death due to brain hemorrhage, while HI was 20-24 in cases of abnormal FHR but neither followed by the loss of variability nor cerebral palsy [3]. Thus the threshold neither to develop the loss of variability nor cerebral palsy is 20-24, and the HI to decide to perform early caesarean delivery will be the HI of 20 or less, after the loss of acceleration and decreased amplitude of variability to the level of 5 bpm or less.

Discussion

As the theory of hypoxia index is adopted not only late deceleration but also to all decelerations including variable decelerations, because the HI does not evaluate the lag time but the FHR score does, and also adopted to the sudden and acute



continuous FHR bradycardia. Therefore, fetal heart rate will be evaluated with FHR score in short period for 5min, while by long period is evaluated with hypoxia index in computerized FHR monitoring, where the diagnosis of pathologic sinusoidal heart rate should be added.

Conclusion

The novel hypoxia index is useful not only to evaluate late deceleration, but also early and variable decelerations without the classification of deceleration patterns, excluding the controversy FHR pattern. Continuous fetal bradycardia is also evaluated by HI, excluding hypoxic ischemic encephalopathy. Additional necessary

diagnoses will be the short term FHR score and pathologic sinusoidal FHR.

References

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