Analytic studies on fetal heart rate changes to prevent cerebral palsy with novel hypoxia index in fetal monitoring

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Abstract

Fetal heart rate (FHR) rises when the fetus moves, where FHR acceleration is reacted fetal brain to fetal movements (burst), it loses in early fetal hypoxia, while FHR variability is the reaction to minor fetal motions, which loses in severe hypoxia followed by cerebral palsy.

FHR falls in hypoxia when fetal PaO₂ is 50 or less mmHg, while fetal PaO₂ is lower than 50 mmHg, where fetal vagal nerve center is excited by hypoxia, forming FHR bradycardia and deceleration. The late deceleration was ominous in the past, while 3 connective typical late decelerations outcome was normal, while repeated late decelerations for 50 minutes developed heavy asphyxia and severe brain damage, thus, novel hypoxia index is the sum of deceleration durations (min) divided by the lowest FHR, and multiplied by 100, where no cerebral palsy developed when hypoxia index was 24 or less, while the index was 25 or more in cases of cerebral palsy. Visual FHR pattern classification will be changed to objective hypoxia index and FHR score which predicts Apgar score and UApH. FHR frequency spectrum the lowest FHR, and multiplied by 100, where no cerebral palsy developed when hypoxia index was 24 or less, while the index was 25 or more in cases of cerebral palsy. Visual FHR pattern classification will be changed to objective hypoxia index and FHR score which predicts Apgar score and UApH. FHR frequency spectrum the lowest FHR, and multiplied by 100, where no cerebral palsy developed when hypoxia index was 24 or less, while the index was 25 or more in cases of cerebral palsy. Visual FHR pattern classification will be changed to objective hypoxia index and FHR score which predicts Apgar score and UApH.

Introduction

The fetal heart rate (FHR) is the target of intrapartum fetal monitoring, where main subject was abnormal FHR pattern, including bradycardia, the loss of baseline variability followed by cerebral palsy, late and severe variable decelerations and pathologic sinusoidal FHR, mainly diagnosed by visual classification during delivery. Abnormal FHR was treated by oxygen inhalation, posture change of parturient woman to lateral one, and main therapy was caesarean early delivery. Perinatal mortality and cerebral palsy were reduced in Japan while Dublin trial did not show the reduction of cerebral palsy, thus, update target is the reduction of cerebral palsy.

Methods

Although main target was the detection of FHR pattern abnormality to perform emergency early caesarean delivery, particularly late and severe variable decelerations. Some abnormal FHR classification placed the loss of baseline variability at the worst FHR change, however, the loss of variability resulted unexpected cerebral palsy, namely, the loss of variability was the mostly severe hypoxic fetal brain damage. Although late deceleration was severely abnormal pattern in abnormal FHR pattern [1], the author experienced fully normal, Apgar 9 neonate was born by caesarean delivery, after 3 connected typical late decelerations in the past, while frequently repeated late decelerations in 50 minutes due to refusal of caesarean delivery, the results was severely asphyxiated Apgar 3 neonate with severe infantile brain damage. In addition, a late deceleration definition needed 15 or more minutes' repetition of the decelerations before definition of late deceleration, thus the author understood that late decelerations is ominous not due to the late appearance of deceleration, but frequent repetition of FHR decelerations may develop severe fetal hypoxia, namely, the hypoxia stimulate and excited vagal nerve center located in medulla oblongata, developing FHR bradycardia, where the heart rate was lineally parallel to PaO₂, if the PaO₂ is lower than 50 mmHg [2] and actual human fetal PaO₂ was 50 or less mmHg [3]. Thus, small number of FHR deceleration will not affect the fetus, while frequently repeated decelerations will damage the fetus. The author intended to observe the damage by the novel hypoxia index as follows; Novel hypoxia index the sum of all deceleration durations (min) in fetal monitoring, divided by the lowest FHR (bpm), and multiplied by 100 to keep the index an integer. The authors collected 6 cases of infatile cerebral palsy, and 16 cases of no cerebral palsy diagnosed in pediatric clinics and calculated their hypoxia indexes in FHR recorded in intrapartum monitoring preserved in Obstetric ward.

Results and discussion

The hypoxia index of cerebral palsy was 25 or more in all of 6 cerebral palsy cases, while the index was 24 or less in all of 16 cases of no cerebral palsy, and there was significant difference. As the probability to make wrong decision was 0.000008, almost zero in the Chi² test, we decided the case whose hypoxia index is 24 or less predicted no cerebral palsy, namely, infantile cerebral palsy is prevented when the novel hypoxia index is 24 or less.

Also, the case, whose hypoxia index was 25 or more, can receive the cerebral palsy treatment early, even in neonatal stage.

Furthermore, increasing hypoxia index in fetal monitoring can be prevented by the change to Lateral posture posture from supine...
posture, when FHR deceleration is recorded on the FHR curve in fetal monitoring, because it was reported that FHR deceleration disappeared when the parturient woman changed supine posture to lateral one [4], namely, hypoxia index does not increase if the deceleration is lost.

Although novel hypoxia index is able to be manually calculated, automatic calculation with computer will be more convenient in the future, where already Apgar score and UApH are predicted by the FHR score [5], and pathologic sinusoidal FHR which appears in severe fetal anemia and asphyxia imminent to fetal demise will also be predicted by computerized FHR curve frequency spectrum, thus, visual FHR chart diagnosis with FHR pattern classification will be replaced by computerized mathematical FHR diagnosis in the future.

We have to recognize that intrauterine fetal environment is hypoxic, namely fetal blood PaO$_2$ is lower than 50 mmHg, which is lower than half of adult arterial blood PaO$_2$ which will be 100 mmHg or more. Thus, a fetus is easily damaged by hypoxia. We have to recognize that FHR deceleration is the hypoxic sign lower than 50 mmHg. Unfortunately the relation of human fetal PaO$_2$ and fetal heart rate is unknown. It is an estimation based on rabbit experiment, namely, fetal deceleration hypoxia is estimated from rabbit's case [2].

**Disclosure**

None

**Conclusion**

It is lucky to know that there is no cerebral palsy if hypoxia index is 24 or less. We can prevent cerebral palsy keeping hypoxia index at 24 or less level. Lateral posture at appearance of deceleration will promote to keep hypoxia index at low level.

As Happier world will appear when handicapped children decrease by advanced fetal monitoring reducing cerebral palsy by novel hypoxia index. The index and similar mathematical fetal diagnosis will change conventional FHR pattern classification, where update FHR diagnosis is clearly preventing cerebral palsy to improve young happy lives in the world.

**References**