Fetal Heart Rate Changes are the Fetal Brain Response to Fetal Movement in Acto cardiogram: The Loss of Fhr Variability is the Sign of Fetal Brain Damage

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Abstract

Aims

To study fetal brain response to fetal movements with fetal heart rate (FHR) changes.

Methods

FHR changing mechanism was investigated by simultaneously recorded FHR and fetal movements detected directly at fetal thorax with Doppler ultrasound in act cardiogram (ACG). FHR changing process was confirmed by electronic and physiologic simulations.

Results and conclusion

As recorded movement spike height was parallel to fetal movement amplitude in ACG, FHR increased when the fetus moved, and triangular FHR acceleration developed at fetal movement burst by the integral function of midbrain. Moderate fetal movements developed moderate FHR increase, and periodically changing fetal movements developed physiologic sinusoidal FHR separating pathologic sinusoidal one. FHR variability developed by minor fetal movements. FHR acceleration was lost in early stage of hypoxia, and then the loss of variability comparable to anencephaly appeared in severe hypoxic fetal brain damage, followed by cerebral palsy. Thus, early delivery is recommended before the loss of variability, instead of C-section after the loss of variability.

Rabbit heart rate reduced along PaO$_2$ drop, where parasympathetic centre was excited in medulla oblongata by low PaO$_2$ developing fetal bradycardia, which showed environmental hypoxia, whereas the loss of variability was full brain damage due to severe hypoxia, where fetal brain could not respond fetal movements as anencephaly.

Keywords

Fetal heart rate; Fetal movement; Acceleration; Sinusoidal heart rate; Baseline variability; Hypoxia; Bradycardia; Brain damage; PaO$_2$

Introduction

The cardiotocogram (CTG) studied FHR and uterine contraction [1], but it was unable to show developing mechanism of FHR acceleration, physiologic sinusoidal FHR and baseline FHR variability. Although FHR pattern classification was commonly used in obstetrics, the pattern diagnosis was frequently controversial, possibly due to empirical and subjective FHR patterns, though perinatal outcome was improved by the rapid delivery after introduction of CTG monitoring. As the actocardiogram (ACG) records FHR and fetal movement spikes [2], various problems in CTG were solved, e.g. the separation of physiologic sinusoidal FHR from pathologic one, and the mechanism to change FHR were clarified.

Methods

Maeda [2] created the ACG, which was simultaneous records of FHR and fetal movement spikes detected directly at the surface of fetal thorax using ultrasonic Doppler method (Figure 1), because the transducer was set to detect fetal heartbeat. Fetal movements were fully detected by world researchers using the ACG [3-5], therefore, the relation between FHR change and fetal movement was objectively investigated in the off-line studies [6-13]. As the spike amplitude recorded on the chart was parallel to the moving width of the subject (Figure 2) [2], fetal moving size was correctly represented by the spike amplitude in ACG, i.e., the magnitude of fetal movement is able to be discussed measuring recorded movement spikes.

Umezawa [14] found that heart rate of adult rabbit closely correlated to rabbit's PaO$_2$, when it was lower than 50 mmHg, and Nakano [15] found that PaO$_2$ of the umbilical cord arterial blood was 50 or less mmHg Therefore, fetal hypoxia was diagnosed by fetal bradycardia.

Fetal anencephalic ACG and heart rate of anencephalic neonates were studied in the comparison to normal fetus [16].

Teshima [10] studied ACG of 20 fetal growth restriction (FGR) which lost FHR acceleration against fetal movement bursts preserving FHR variability (non-reactive FHR), comparing to the foetuses of 20
normal reactive FGR with positive acceleration against fetal movement burst.

**Figure 1:** A normal actocardiogram recorded in fetal active state. Triangular accelerations were accompanied with fetal movement bursts. The figure shows how to calculate the A/B ratio of the actocardiogram, where sum of acceleration duration (total A) divided by the sum of movement duration (total B) is A/B ratio [12].

FHR acceleration developed against fetal movement bursts. Fetal movement bursts repeatedly associated triangular FHR accelerations in fetal active state (Figure 3), while neither FHR acceleration nor fetal movement was recorded in fetal resting state, namely, FHR acceleration was formed by the fetal movement burst.

**Figure 3:** Two cases of active fetal sate ACGs, which show clear association of triangular FHR accelerations with fetal movement bursts. The reason why the acceleration is triangular is explained by simulation experiments listed in the text.

**Acceleration development was estimated by two simulations**

FHR acceleration was reported to develop in the midbrain in the studies on anencephalic fetal heart rate [6]. Fetal movement burst was usually continuous in fetal active state, where all accelerations were triangular. Therefore, electronic simulation was studied by the input of 10 Hz wave groups into the integral circuit with 7 sec time constant, because the correlation coefficient was largest when the movement signal delayed for 7 sec in the correlation study of FHR and fetal movement [7]. The output of integral circuit was invariably triangular in the electronic simulation [8].

Also in a physiologic simulation test, adult heart rate was recorded with a fetal heart rate meter in the repetition of leg motions for 1 min, where the heart rate repeatedly changed to triangular accelerations [8]. Since the experimental person did not recognize the heart rate change, the process of triangular heart rate increase would not be formed in the cerebrum, but in the midbrain.

**Moderate fetal movements developed moderate FHR variations**

Suitable example is fetal physiologic sinusoidal heart rate. The pathologic sinusoidal FHR developed in severe fatal anaemia, feto-maternal transfusion, Rh-incompatibility or Parbo-B 19 viral infection, while physiologic sinusoidal was healthy with favourable outcome, therefore, it should be differentiated from pathological one. The separation of physiologic sinusoidal from pathologic one was, however, unable with the CTG., while the differentiation was achieved in ACG, where moderate sine wave-like FHR variation was closely similar to the envelop of periodic fetal movements in the fetal cyclic respiration or mouthing movements (Figure 4) [9]. The FHR variation would develop also in the midbrain.

**Minor fetal movement develops FHR baseline long term variability**

Long term FHR variability amplitude is 5-24 beats per minutes (bpm) on the FHR baseline. The variability develops by minor fetal movement.
movements, which were recognized on enlarged ACG using computer processing (Figure 5). The variability would also develop in the midbrain.

The variability would also develop in the midbrain.

Figure 4: The most upper wavy line was a physiologic sinusoidal FHR, which should be differentiated from true pathologic sinusoidal one. The most low record was periodically changing fetal respiratory movements, of which envelop was manually traced and compared the sinusoidal heart rate after 7 s delay. The upper sinusoidal FHR was the same as the envelop, therefore, the FHR was diagnosed as physiologic reaction to fetal movement, and the fetal outcome was fully favourable.

Figure 5: Minor fetal movements revealed in augmented resting state actocardiogram. FHR variability was provoked in the augmented FHR (upper trace) by the minor fetal movements (lower trace).

Fetal brain damage is estimated by the loss of long term variability

FHR accelerations reduced in the early stage of fetal hypoxia, where the ratio of acceleration duration to fetal movement burst (A/B ratio) decreased, where short and long term outcomes closely correlated with the A/B ratio, and the outcomes were unfavourable when the A/B ratio was less than 1.0. Also the severity ranking of central nervous system lesions was determined by the A/B ratio [11-13].

The loss of variability suggests the severe fetal brain damage

The acceleration of fetal growth retardation (FGR) cases disappeared in early stage of hypoxic suppression in FGR, where the variability was preserved, while severe NRFS asphyxia including the loss of variability appeared after the loss of acceleration within 2 weeks, and its outcome was ominous in spite of all C-section comparing to normally reactive FHR acceleration of FGR [10].

The variability was preserved when the acceleration was lost, then it was lost finally at the most severe hypoxic fetal brain damage, which was experienced in a case of severe late decelerations, where the baseline was as smooth as an anencephalic fetus (Figure 6). Also the variability was lost in a case of severe FHR variable decelerations, later accompanied cerebral palsy (CP) after birth.

Figure 6: Augmented FHR in the loss of variability. A: The loss of variability in severe NRFS due to repeated late decelerations for long period. Apar score was 3 and the infant died at 3 months due to brain hemorrhage. B: The loss of variability in anencephalic FHR baseline, also showing the loss of other FHR changes. The FHR baseline of severe NRFS is the same as that of anencephaly.

Comparison of FGR who lost FHR acceleration and reactive FGR

Sixteen cases developed severe asphyxia showing the loss of variability, bradycardia, and severe variable and late decelerations among 20 non-reactive FGR cases within 2 weeks after the loss of FHR acceleration. Although all non-reactive FGR received C-section, 2 neonates died and 4 severely asphyxiated, while reactive FGR cases that normally developed acceleration against fetal movement had normal vaginal deliveries developing neither neonatal death nor asphyxia. Significant outcome differences were noted between reactive and non-reactive groups in outcomes [10]. Thus, it is recommended to perform C-section when the acceleration is lost in high-risk cases before the loss of variability.

Discussion

Fetal heart rate variations are caused by fetal movements

Fetal heart rate had been discussed with the uterine contraction in the CTG of fetal monitoring, where various problems remain unsolved
in the use of uterine contraction, and they were clearly solved studying simultaneously recorded fetal movements in ACG as seen in this article, particularly in the developmental mechanism of FHR acceleration, physiologic sinusoidial heart rate and baseline variability. The outcome of fetal disorder is predicted, ranking of fetal CNS lesions is determined, paradox in fetal monitoring was solved, namely, almost all of controversy problems in CTG were solved by the introduction of simultaneous fetal movement studies using ACG, e.g., healthy neonates were obtained in fetal bradycardia associated with FHR acceleration [17,18]. Fetal brain reaction to fetal movement is indispensable in the discussion of fetal pathophysiology.

Fetal bradycardia as the sign of low PaO₂

Adult rabbit heart rate did not respond the PaO₂ change when it is higher than 50 mmHg, while the heart rate highly correlated PaO₂ when the PaO₂ was lower than 50 mmHg, i.e. heart rate dropped showing bradycardia when PaO₂ decreased by the nitrogen gas inhalation [14]. Since human umbilical arterial blood PaO₂ was usually lower than 50 mmHg [15], the fetus shows bradycardia in low fetal PaO₂, i.e. fetal deceleration can be reversible from transiently low PaO₂, but bradycardia itself would not indicate brain damage in transiently mild to moderate decelerations. The parasympathetic centre located in the medulla oblongata is stimulated and excited by the hypoxia, producing bradycardia, actually, experimental hypoxia produced bradycardia, but it did not appear under general rabbit anaesthesia [14]. Also, remained medulla oblongata was excited by the hypoxia after the delivery of anencephalic apoene neonate, developing severe bradycardia which returned to normal heart rate after injection of oxygenated blood [19], namely, bradycardia is an nervous reaction to low PaO₂, but not fetal brain damage in short duration of hypoxia, as confirmed in moderate variable deceleration, showing fetal environmental hypoxia tolerating for some duration. Urgent C-section in prolonged irreversible bradycardia is valuable to prevent hypoxic damage due to prolonged hypoxia.

Severely asphyxic fetus should deliver before the loss of variability

Since the loss of variability appears in the most severe fetal brain damage similar to the loss of brain in anencephalic fetus [16], the highly asphyxiated fetus is recommended to deliver before the loss of variability. Since total CP is 0.2% of birth and the brain damage of mature fetus develops in 10% of CP, the CP developed in term fetus will be 0.02% of births that is 1 in 5,000 births. It is rare; however, it will be 200 cases in yearly one million deliveries in Japan. Therefore, the CP in the loss of variability would be prevented by the delivery before the loss of variability. The delivery timing would be severe bradycardia, severe variable or late decelerations, loss of acceleration, decreased variability, high FHR score, low A/B ratio, high hypoxia index etc. CP was reduced in old time when C-section was performed by above indications, where the fetus might deliver unexpectedly before the loss of variability [20-23].

Hypoxia index

Hypoxia index was studied to indicate the timing of C-section in severe FHR changes. The index is the duration of bradycardia (min) × 100/the lowest heart rate (bpm) of bradycardia. The author studied the index of the cases that showed the loss of variability, which was 25 to 26, while the FHR changes who did not accompany the loss of variability was 20 to 24. Therefore the timing of C-section would be indicated when the hypoxia index is more than 20 but lower than 24.

Is it possible to treat fetal hypoxia to avoid maternal morbidity due to C-section?

Although some one announced coming treatment of hypoxic damaged fetus by anti-glutamate and free radical scavenger, they had not succeeded. Maternal oxygen inhalation is always applied but the effect was insufficient. However, two strategies will be proposed, one of which was already successful to increase placental function, which was; transfer oxygen from maternal to fetal blood.

Solution of fibrin deposit of intravillous fibrin deposit

The treatment was successful in a case of placental fibrin deposit solute by heparin treatment [24]. Fibrin deposit was diagnosed to detect placental B-mode echogenicity by ultrasound tissue characterization, GLHW, which can be determined using common B-mode histogram, of which higher value than normal placenta showed the fibrin deposit. Dr. Utsu treated the mother with 5000 U heparin in a case of FGR in the 29th trimester, where fetal weight increased, showing normal fetal growth, and then normal neonate was obtained, instead of IUFD in previous pregnancy.

Anti-sympathiontic therapy in preeclampsia

Sympatico-tony is characteristic in preeclampsia [25], where uterine arterial constriction would be characteristic, showing the abnormal uterine arterial Doppler flow will be effective to administer antisympaticotonic medicine to the preeclampsia patient to treat the mother and fetus at the same time.

CP prevention in preterm labor

Since CP is definitely more in preterm babies than those in term delivery [22], strategy to prevent CP in Obstetrics will be preventing preterm birth, that is term delivery by various tocolyses, including pharmaceutical or uterus-brain nerve sedation [23], etc., and the treatment of periventricular echo density (PVE) in preterm neonatal brain [19], possibly by administration of growth factor, etc.. These strategies are problems to be solved in the future.

References


