Journal of
Health & Medical Informatics

Maeda et al., J Health Med Informat 2013, 4:4
DOI: 10.4172/2157-7420.1000137

Research Article Open Access

CP Prevention by Early Delivery before Fetal Brain Damage in the Loss of FHR Variability, even in Non-hypoxia

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Abstract

Aim: To assess the hypoxic fetal brain by the fetal heart rate (FHR) changes, particularly with the loss of long term variability (LTV) and acceleration, and to determine hypoxic threshold to prevent cerebral palsy (CP) using hypoxia index.

Methods: The fetal state was assessed by actocardiogram (ACG), the recording of FHR and fetal movement. Origin of FHR acceleration and LTV were assessed by ACG in physiologic sinusoidal FHR and augmented ACG. Hypoxia was determined by the bradycardia and hypoxia duration.

Results: The LTV and acceleration develop in midbrain as the reaction to fetal movement, thus their loss particularly that of LTV indicated such severe brain damage as an anencephalic fetus, which will cause CP. Thus, the threshold level of hypoxia was studied using hypoxia index.Since such non-hypoxic severe FHR changes as the loss of LTV indicate fetal brain damage, the fetus should be delivered also before the loss of LTV in non-hypoxic fetal disorders.

Conclusion: Since FHR acceleration and LTV are produced in the midbrain as the response of brain to fetal movements, the loss of LTV indicates hypoxic brain damage, thus the fetus should be delivered before the loss of LTV, of which development will be detected by the hypoxia index.Since non-hypoxic abnormal FHR can develop by the fetal brain damage, the fetus should be delivered before the loss of LTV to prevent the CP.

Keywords: Fetus; Brain; CP: FHR; LTV; Acceleration; Mid-brain; Hypoxia; Non-hypoxic FHR change; C-section

Introduction

Although it is usually recommended to perform Cesarean section (C-section), when the FHR baseline LTV disappears, an obstetrician experienced unexpected CPdeveloped after C-section due to severe variable decelerations accompanied by the loss of LTV. In addition, no reduction of CP has been reported. These facts suggest to study the developmental mechanism of LTV and acceleration, and their relation to fetal brain damage.

Fetal movements evoked FHR variability and acceleration

The benign physiologic sinusoidal FHR was synchronized to the periodic fetal respiratory movements and differentiated from the true sinusoidal FHR in ACG (Figure 1) [1]. Since movement spike amplitudes on ACG precisely reflect fetal movement amplitudes [2], the FHR changed along with fetal movements, i.e. large LTV changes along with moderate fetal movements, and small fetal movements provoked common LTV [3].

Triangular FHR accelerations were synchronized to large fetal movement bursts [3]. The electronic simulation confirmed that square shaped 10-Hz wave clusters were converted to single triangular wave after passing through the integral circuit with a 7-sec time constant [3], because the correlation of FHR and movement curves was largest when the movement signal was delayed for 7 sec [4]. In addition, continuous leg motion for 1 min induced a triangular heart rate curve in the adult exercise [3]. The acceleration center seems to be outside the brain cortex, because the exercised person did not recognize the own heart rate change [3], and the center of acceleration was reported to be in the mid-brain [5].

Electric signal group changed to single triangular wave after passing through a integral circuit with 7 sec time constant, and triangle heart rate was produced during adult exercise [3]. Thus, FHR LTV and acceleration would develop by the reaction of mid-brain to fetal movements, i.e. large acceleration is provoked by large fetal movements, and LTV derives from small movements[3].

Fetal brain paralysis and damage could be exhibited by the loss of FHR acceleration and LTV, i.e. the acceleration and LTV disappeared in the hypoxic fetal brain paralysis and damage, because both phenomena are produced in the mid-brain, a part of brain. Hypoxic brain damage started by the loss of acceleration in the non-reactive FHR, where acceleration does not appear against fetal movement bursts, but LTV is preserved. Some days later, due to further progress of hypoxia,the bradycardia, severe deceleration and the loss of FHR variability appear. Neonatal states who delivered by the C-section due to the severe NRFS after non-reactive FHR were worse than normal FHR cases. The loss of acceleration against fetal movement bursts and the loss of LTV are found in anencephalic fetus, which loses most parts of the brain. The same FHR changes as anencephalic fetus, found in a severe fetal asphyxia in severe late decelerations, loss of acceleration and LTV but the mother refused C-section, showed severe neonatal asphyxia and the brain damage, of which Apgar score was 3. Such severe asphyxia is rare in the fetus, whereas neuronal cell deaths may accompany to produce cerebral palsy.

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Received September 11, 2013; **Accepted** November 19, 2013; **Published** November 24, 2013

Citation: Maeda K (2013) CP Prevention by Early Delivery before Fetal Brain Damage in the Loss of FHR Variability, even in Non-hypoxia. J Health Med Informat 4: 137. doi:10.4172/2157-7420.1000137

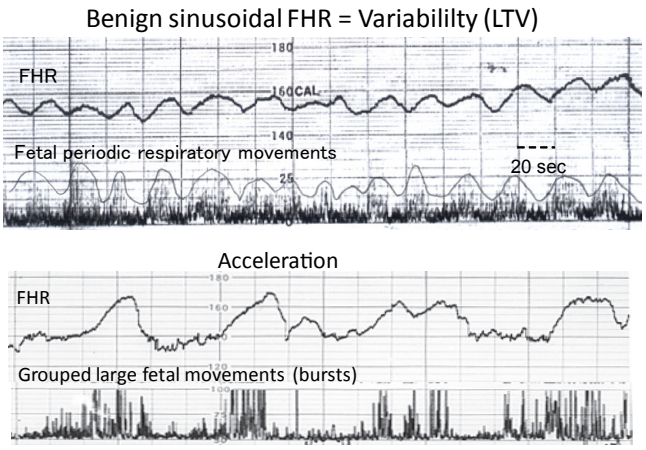

Figure 1: Upper figure shows a moderate long term variability (LTV) developed by the 7 sec delayed reaction of fetal mid-brain to fetal periodic respiratory movements. Lower records show triangular FHR accelerations developed by the delayed reaction of fetal mid-brain, after passing through an integral system of 7 sec delay.

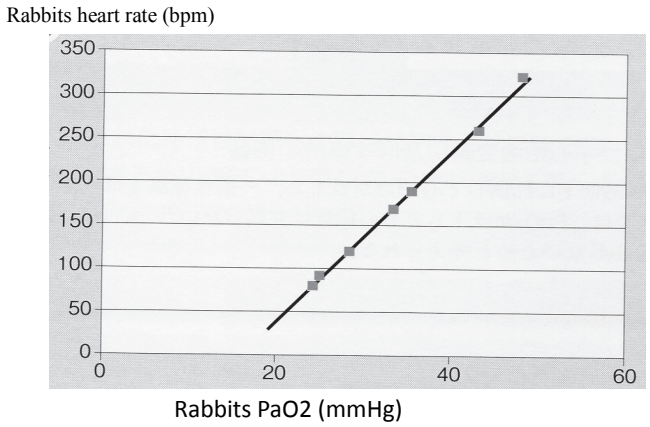

Figure 2: The heart rate and PaO_2 of a rabbit were closely correlated in hypoxia [6]. Therefore, the hypoxic index was determined by the lowest FHR instead of fetal PaO_2.

Experimental hypoxic bradycardia developed by PaO_2 lower than 50 mmHg

Bradycardia appeared in hypoxic rabbits when the PaO_2 dropped below 50 mmHg, where the heart rate decreased parallel to PaO_2 lowering (Figure 2), while the rabbit showed no hypoxic bradycardia after urethane anesthesia [6]. In addition, anencephalic neonates without brain but preserved medulla oblongata, showed severe bradycardia in the postnatal apnea, where the heart rate recovered to normal by an oxygenated blood infusion [7]. These findings indicate that the parasympathetic center of medulla oblongata was excited by hypoxia to produce bradycardia. The PaO_2 level in hypoxia could be determined by fetal bradycardia or deep deceleration (Figure 2). A human fetus undergoes bradycardia in response to hypoxia, because the fetal umbilical blood PaO_2 is lower than 50 mmHg [8]. The medulla oblongata is stimulated, while other parts of the fetal brain are paralyzed or damaged by hypoxia, i.e. the severity of hypoxia could be determined by FHR in the labor and the possible damage of fetal brain is estimated by the fetal bradycardia.

Possible prevention of cerebral palsy in severe fetal asphyxia

The loss of FHR variability indicates no response of fetal brain to fetal movements in hypoxia. The sign will show that it is a partial sign of general brain damage, and it can be possible to produce neuronal cell deaths, which causes cerebral palsy (CP) after the birth. Therefore the CP will be prevented, if the hypoxic fetus is delivered by C-section before the severe brain damage in the loss of variability (LTV). The non-reactive FHR is the loss of FHR acceleration against fetal movements preceding severe hypoxic signs including the loss of variability, severe bradycardia and deceleration some days after the loss of FHR acceleration [9]. Thus, the C-section in non-reactive FHR will be a case to deliver the fetus before the loss of variability. Another way to predict the loss of variability is to estimate fetal PaO_2 in the labor, and the threshold of PaO_2 level will be determined to cause the loss of variability. However, it is very difficult to determine fetal PaO_2 during the labor. Since the rabbit PaO_2 was indicated by the heart rate (Figure 2), the PaO_2 will be estimated by FHR in the human fetus. An index to determine the threshold to cause the loss of FHR variability will be the Hypoxia Index (HI) to estimate the hypoxic impact on the fetal brain. The HI is shown by the following equation;

Hypoxia Index=Duration (min) of bradycardia (D)×100/the lowest FHR (R)

A HI of V-shaped deceleration is half of U-shaped one, because the area of V-shaped deceleration is half of U-shaped one.

In repeated decelerations, HI=(D1/R1+D2/R2+- - - -+Dn/Rn)×100.

The Hypoxia indices of various FHR samples

Case 1 (Placental separation): The lowest FHR was 60 bpm and duration 15 min.

The HI was 15×100/60=25 (Figure 3).

Case 2 (Severe late decelerations and the loss of variability, Apgar 3, brain damage):

Two min V-shaped late decelerations with the nadir of 100 bpm repeated. Total durationwas 50 min. Severe loss of variability was accompanied. The HI was 50×100/100/2=25 (connected V-shaped

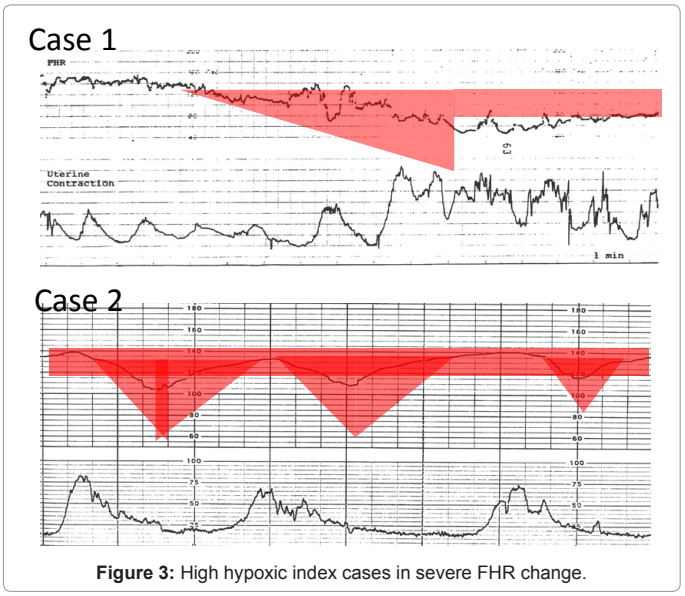

Figure 3: High hypoxic index cases in severe FHR change.

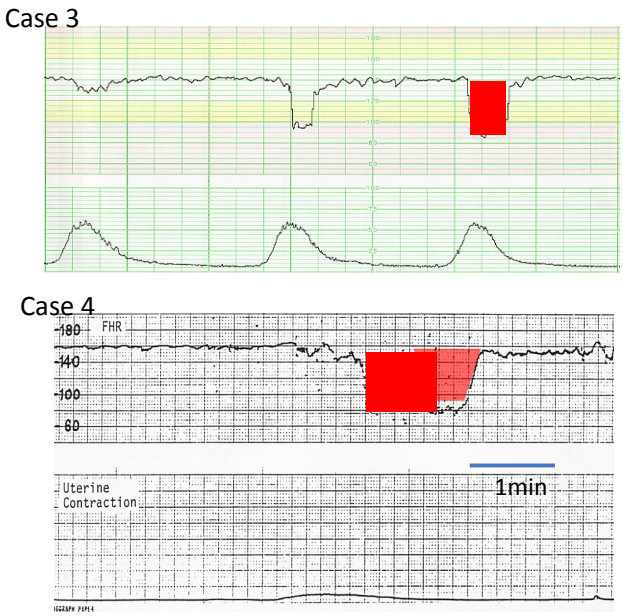

Figure 4: Mild and moderate variable decelerations.

decelerations were lasted for 50 min due to the C-section refusal). Despite nadir FHR was 100 bpm, long duration would result the loss of variability (Figure 3).

In another severe variable decelerations accompanied the loss of variability, who was born by C-section, HI was 26 and unexpected CP developed.

Case 1 was a placental separation. Bradycardia lasted 15 minutes before the emergency C-section. The HI was 25.

Case 2 was 50 min duration of severe late deceleration and severe loss of variability due to the refusal of C-section. Apgar score was 3 and brain damage was diagnosed. The HI was 25, where the loss of variability was associated.

Low HI was determined in mild and moderate variable decelerations. It was lower than 10 and variability was preserved in case 3. A severe variable deceleration case 4 was 21 in 10 decelerations, showed normal variability.

Case 3: Mild variable U-shaped deceleration, normal outcome (Figure 4):

HI=0.5×100/95=0.6. Total HI<6 even in 10times repetition, No impact to the brain.

Case 4: Moderate variable U-shaped deceleration(Figure 4):

HI=1.7×100/80=2.1 Total HI=21, in 10 decelerations. Normal variability and normal outcome.

The HI threshold not causing the loss of variability would be less than 25, but more casesshould be studied in the future.

Case 3: Low hypoxia index was determined in mild deceleration.

Case 4: Moderate hypoxia index in moderate deceleration. Total HI is 21, when the deceleration is repeated for 10 times.

Comments

The hypoxia affects fetal brain in the loss of FHR variability (LTV), and severe loss of LTV less than 1 bpm amplitude could develop such severe brain damage as an anencephaly [3]. Since it is possible to produce necrotic change of neuronal cells in severe brain damage, which is the cause of a CP, the fetus should be delivered by the C-section before the loss of LTV, which indicates the presence of general brain damage.

Since the actocardiographic non-reactive FHR, where FHR acceleration disappears in the presence of fetal movements, and differentiated from fetal resting state, which shows disappeared acceleration but also no fetal movement, will follow severe NRFS including the loss of LTV within some days, it will be reasonable to perform a C-section before the loss of LTV.

Another way to perform C-section before the loss of LTV is to know the PaO_2 level to produce the loss of LTV and fetal brain damage. The hypoxic index (HI) is proposed in this article to know the PaO_2 threshold, where the lowest FHR is used instead of PaO_2, a critical HI will indicate the time to perform C-section before the loss of LTV.

Non-hypoxic FHR changes were reported [10,11], but the abnormality would indicate the brain damage, the principle would be the same as hypoxic change, i.e. the fetus of abnormal FHR should be delivered before the loss of LTV in the FHR.

Conclusion

Since the loss of LTV (long term variability of FHR) is the sign of fetal brain damage, the fetus of abnormal FHR should be delivered before the loss of LTV to reduce the CP. A C-section will be indicated in the case of actocardiographic non-reactive FHR or in the case of hypoxia index below the threshold for the loss of LTV. Since the severely abnormal FHR could indicate the fetal brain damage also in non-hypoxic fetal insults, C-section could be indicated before the loss of LTV in the FHR.

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