Fetal Movement in Actocardiogram and Prevention of Cerebral Palsy with Hypoxia Index

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Abstract

Aim: To analyze the relation of fetal movement and heart rate.

Method: Fetal movement and heart rate (FHR) was analysed by actocardiogram. Novel hypoxia index was calculated by the sum of FHR deceleration duration and the lowest FHR.

Results: As FHR increased when the fetus moved, FHR was studied in the relation to fetal movements, e.g., benign sinusoidal FHR was evoked by periodic fetal respiratory movement. No FHR acceleration was found in fetal hiccuping due to no formation of fetal movement burst. Hypoxic loss of FHR acceleration and variability were caused by no response of damaged fetal brain to fetal movement.

Discussion: The fetus should be cured from cerebral palsy by early delivery before the loss of variability, where the hypoxia index was 25 or more in the loss of variability followed by cerebral palsy, while normal variability and no cerebral palsy was preceded by 24 or less hypoxia index. Thus, the HI should be 24 or less to prevent cerebral palsy.

Conclusion: FHR changes, which had not solved by CTG, was solved by the application of fetal movement. Novel hypoxia index is useful for the prevention of cerebral palsy by the 24 or less of hypoxia index.

Keywords: Fetus; Heart Rate; Brain Damage; Cerebral Palsy; Hypoxia Index; Ultrasound; Doppler Signal

Introduction

Although pregnant woman feels fetal movement as kicking, that is subjective and unsuitable to scientific study. Thus, there had been several technical methods to detect fetal movement, e.g., piezo-electric element, microphone, external uterine contractin detector, photo-diode technique, etc [1]. However, they detected the movement at maternal abdominal surface, but it was not direct detection of fetal movement in the amniotic fluid. The author intended to record fetal motion directly in the amniotic fluid with ultrasonic Doppler signal developed by fetal chest motion in amniotic fluid.

Methods

It was usual to detect ultrasonic Doppler vibration developed by fetal heart beat to record fetal heart rate (FHR) with ultrasonic probe in commercial ultrasonic Doppler fetal heart rate monitor, where the direction of ultrasound beam was fixed at fetal chest, therefore, the Doppler signals contained various movements including maternal and fetal movements and fetal heart beats. The author extracted the fetal movement Doppler signals using various frequency filters in the remodeling of TOITU TN-400 fetal monitor (Tokyo) by himself, detecting the ultrasonic Doppler signal at fetal chest, which contained various movement Doppler signals. The author sit at the bed side observing oscillographic images measuring their frequencies, where maternal motion was 2 Hz or low, fetal heart beat was 100 Hz or more, then fetal movement Doppler frequency was 20-50 Hz, checking the monitor sound of the ultrasonic fetal monitor and measuring Doppler signal frequency with real-time frequency analyzer.

The author cut off maternal motion Doppler signal by 8Hz high-pass filter observing the oscilloscope, then cut-off the Doppler signal of fetal heart beat by 100 Hz high-pass filter, then obtained pure fetal movement Doppler signal, which was 20-50 Hz when ultrasound was 2 MHz and it was extracted using available 20-80 Hz band-pass filter, which was adjusted to fetal motion spikes to record in the fetal monitor recorder, where the prototype actocardiograph was completed [2,3] (Figure 1).

Results

Fetal movement spike amplitude

As the movement spike amplitude was parallel to the movement of the steel ball in the water set against the ultrasonic probe, fetal motion amplitude was able to be estimated with the height of actographic spike, namely, fetal chest movement width was estimated by the amplitude of recorded spike [2,3] (Figure 2).

Fetal movement spikes were coincided with the fetal motion detected by real-time ultrasound B-mode.

Commercial models

The author asked TOITU (Tokyo) to provide commercial model of

| 2 | 20 | 50 | 100 | ~ | 2 | 20 | 50 | 100 Hz ~ |
| Maternal | - - - - | Fetal | - - - - | - - - - | - - - - | - - - - | - - - - | - - - - |

Figure 1: Fetal movement Doppler signal frequency was 20-50 Hz, if the ultrasound was 2 MHz and it could be extracted using 20-80 Hz band-pass filter.

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actocardiogram, then MT-320, the first commercial model, was tested by perinatology experts in the world, where all fetal movements were recorded by actocardiogram, namely movements of extremities and even fetal mouthing were conducted to fetal chest. Afterwards, many actocardiographs were provided and update model is MT-630 (Figure 3).

Since TOITU models recorded fetal heart rate (FHR), fetal movement spikes and uterine contraction simultaneously, it was an actocardiograph (ACG) when FHR and fetal movement were analyzed and it was cardiotocograph (CTG), when FHR and uterine contraction were analyzed. Thus, the machine could be called as acto-cardio-toco-graph (ACTG), but usually called as actocardiogram from its main function.

**Fetal behavior**

Fetal behavioral states were classified into active, highly active, resting and intermediate states classified by the active to resting states [2,3] (Figures 4-7).

**Fetal behavioural states**

They are divided into active, highly active, resting and intermediate states, of which details are shown in Tables 1-3 (Figures 4-7).

**Fetal hiccupping movements**

Fetal hiccupping was clearly shown by sudden and large stretching motion in the real-time B-mode ultrasound image, which is continuous

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**Figure 2:** The fetal movement width is estimated by the amplitude of movement spikes.

**Figure 3:** Update MT-630 Actocardiograph.

**Figure 4:** Active fetal state. Triangular FHR accelerations appear against fetal movement bursts. FHR changes delay 7 sec to fetal movement [2,3].

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**Table 1:** Actocardiographic fetal behavioral states were compared to that reported by Nijhuis [4].

<table>
<thead>
<tr>
<th>Nijhuis's States</th>
<th>Fetal Movements</th>
<th>Fetal Heart Rate</th>
<th>Actocardiogram States</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body</td>
<td>Eye</td>
<td>Acceleration</td>
<td>Bandwidth</td>
</tr>
<tr>
<td>1F</td>
<td>– (or brief)</td>
<td>–</td>
<td>narrow</td>
</tr>
<tr>
<td>2F</td>
<td>++</td>
<td>+</td>
<td>&gt;1F</td>
</tr>
<tr>
<td>3F</td>
<td>–</td>
<td>+</td>
<td>The regularity and frequency of variability are more than 1F</td>
</tr>
<tr>
<td>4F</td>
<td>+++</td>
<td>+</td>
<td>unstable or tachycardia</td>
</tr>
</tbody>
</table>

**Table 2:** Three quantitative parameters of fetal movement.

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Mean duration of fetal movement bursts</th>
<th>Summarized fetal movement durations divided by the recording duration</th>
<th>The frequency of movement bursts in a minute.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burst duration (sec)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Occupancy (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frequency (cycles per minute, cpm)</td>
<td></td>
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</table>
regular repetition of sharp spikes appearing suddenly and repeating 10 or more min. Twice appearances were experienced in day time in late pregnancy.

Fetal hiccupping is a problem in fetal heart rate diagnosis, because the continuous fetal hiccuppings did not accompany fetal heart rate acceleration and the loss of acceleration is the sign of fetal hypoxic brain suppression in actocardiogram, however the outcome is favorable and there was no hypoxic asphyxia in fetal hiccupping (Figures 8 and 9).

As the hiccupping does not accompany FHR acceleration, hiccupping should be separated from non-reactive FHR by the loss of movement burst formation.

**How to separate fetal hiccupping from the non-reactive loss of acceleration?**

It is possible because fetal hiccupping does not form grouped movement burst, but it is single and very sharp single repeating continuous spikes, which suddenly starts and finishes, while it makes no triangular grouped fetal movement bursts, which is found in active fetal state (Figure 4).

Real-time ultrasound B-mode more clearly shows that hiccupping,

<table>
<thead>
<tr>
<th>Normal active state after 22 W</th>
<th>Intermediate States</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>N=14</td>
<td>N=5</td>
<td></td>
</tr>
<tr>
<td>Burst duration (Sec)</td>
<td>29.69 ± 10.27 (18.9-67.8)</td>
<td>17.78 ± 3.68 (12.1-21.1)</td>
</tr>
<tr>
<td>Occupancy (%)</td>
<td>32.7 ± 14.76 (12.9-58.8)</td>
<td>6.44 ± 1.75 (3.5-7.9)</td>
</tr>
<tr>
<td>Frequency (cpm)</td>
<td>0.7 ± 0.22 (0.23-0.99)</td>
<td>0.17 ± 0.03 (0.13-0.2)</td>
</tr>
<tr>
<td>state duration (min)</td>
<td>&lt;50</td>
<td>28.4 ± 7.09 (20-35)</td>
</tr>
</tbody>
</table>

**Table 3:** Parameters of active and intermediate states [2,3].

**Figure 5:** Highly active fetal state. Fetal tachycardia lasted for 5 min against 4.5 min fetal movement bursts [2,3].

**Figure 6:** Resting fetal state. There is neither FHR acceleration nor fetal movement burst. However, FHR baseline variability is preserved, which is the response to minor movements in resting state [2,3].
Figure 7: Intermediate fetal state, small movement bursts and small acceleration [2].

Figure 8: Continuous fetal hiccupping movements did not accompany FHR acceleration.

Figure 9: Ominous non-reactive FHR, which was the loss of FHR acceleration against fetal movement burst, which was triangular fetal movement signal groups.
which is strong convulsive stretching, which is regular solid actions, forming solid and very sharp actographic spikes.

In electronic simulation, 10 Hz grouped pulses for 30 sec formed triangular wave after passing through an integral circuit, while continuous signals for some minutes formed no acceleration after passing through the integral circuit, namely, fetal hiccupping, which is continuous sharp spikes form no acceleration after passing through integral circuit. These facts explains the no acceleration in the fetal hiccupping, namely, short duration of spikes make acceleration, however, 10 min continuously repeated regular spikes form no acceleration [4].

**How to separate physiologic sinusoidal FHR from the pathologic sinusoidal?**

Answer is to use actocardiogram which records fetal movement simultaneously with fetal heart rate, which changes according to fetal movement amplitudes, due to fetal brain function. Thus, the CTG did not separate physiologic sinusoidal from pathologic one because it did not record fetal movement. There are periodic fetal movements, e.g. periodic fetal respiration and fetal mouthing movements, of which motion was detected by fetal brain to change fetal heart rate periodically with 7 sec delay, that is the time constant of integral function of fetal brain, of which presence was confirmed in adult brain too, namely, regular leg motion of an adult changed its heart rate and formed triangular heart rate in a physiologic experiment. The envelope of fetal respiratory movements formed physiologic sinusoidal FHR [5] (Figure 10).

**Fetal response to sound and light**

*Intrauterine fetus responded external sound and light.*

1. **Fetal response to sound**

   200, 500 and 1,000 Hz sine waves were generated by HP signal generator and amplified with power amplifier and the sound was radiated by 10 inch dynamic loud speaker on maternal abdomen for 2 sec, 2-3 times repeatedly, Fetal behavioral state was resting state, because false fetal reaction in active state was avoided. Loud speaker output was measured with an audiometer at 1 m apart from speaker. Fetal heart rate and movement were recorded by a TOITU actocardiogram (Tokyo).

   Fetal response to the sound was fetal movement followed by FHR acceleration in positive reaction. All of 200, 500 and 1,000 Hz sound stimulation in 2 sec achieved positive fetal reaction, but significantly positive reaction was achieved only by 1,000 Hz sound stimulation [6-10]. Positive reaction was obtained by 80 dB intensity sound in a case of 28 weeks of pregnancy, while the same fetus responded to 60 dB ultrasound in 40 weeks of pregnancy, namely, fetal hearing function increased about 10 times in 2 weeks in late pregnancy (Figure 11).

   Is fetal education is possible by maternal voice? Answer is negative because maternal voice is lower than 1,000 Hz, to which the fetus responded [11-13].

2. **Fetal response to light**

   Photographic strobolight of 20 guide-number was flushed on maternal abdomen around at fetal head. Positive reactions were fetal movement then FHR acceleration. The first positive reaction was achieved at 23 weeks of pregnancy, when fetal retina was formed. Positive reaction rate to the light was increased to 77% in 40 weeks of pregnancy [13] (Figure 11).

**Discussion**

**Intrapartum fetal monitoring**

As stated above, continuous recording of fetal heart rate and
movement with actocardiogram was convenient to assess the cause of fetal heart rate change, fetal behavior, detection of fetal hypoxia by the loss of heart rate acceleration and the loss of baseline variability, where it is able to diagnose more fetal states than cardiotocogram, which is the recording of fetal heart rate and uterine contraction, while actocardiogram records also fetal movements, namely, actocardiogram also prepares the function of cardiotocogram too, if the user hopes to monitor heart rate and uterine contraction simultaneously. As fetal heart rate is recorded with ultrasonic Doppler autocorrelation heart rate meter, its heart rate tracing is external through pregnancy and labor and it is as clear as fetal scalp lead ECG with needle electrode, of which use is limited only after the rupture of membrane and in case of no maternal viral disease [6]. As ultrasound intensity of autocorrelation is only 1 mW/cm², which is very low and safe.

Novel hypoxia index to numerically prevent cerebral palsy

Since 3 connective late decelerations (LD) achieved normal neonate with Apgar score 9, though ominous outcome was expected, when particular pattern of LD was recorded, namely, fetal abnormality should be expected in all of LD in FHR record, while, 50 min continuation of LD patterns resulted Apgar 3, severe neonatal asphyxia with severe brain damage, characterized by the loss of FHR variability, where repeated decelerations, which is characterized by overlapping transient hypoxia will be the target, namely, novel hypoxia index was the sum of deceleration duration (min) divided by the lowest FHR (bpm) and multiplied by 100. Heart rate was used instead of PaO₂, because heart rate was completely linearly correlated to PaO₂ if it is lower than 50 mmHg and cord blood PaO₂ is lower than 50 mmHg. The hypoxia index is recommended to apply to all decelerations, including so-called early, late and variable decelerations and also fetal continuous bradycardia because the equation does not include the lag time. Also the author recommend to try to change maternal posture to lateral one, because various fetal bradycardia is caused by the compression of large blood vessels by contracted pregnant uterus to limit the blood flow through the placenta at supine posture. Typical disappearance of late decelerations after lateral posture was reported by Poseiro 1969. You do not need to calculate the index, if the deceleration disappears after taking lateral posture. In a retrospective study, the author collected 6 cerebral palsy cases and 16 normal cases without cerebral palsy with intrapartum FHR chart and calculated the Hypoxia Index by the author, where Hypoxia Index was 25 or more in cases of cerebral palsy and 24 or less in cases of no cerebral palsy. Thus, the numeric threshold exists between positive and negative cerebral palsy, it is located between 24 and 25 of hypoxia index (Table 4).

The author would like to recommend to try lateral posture when there is fetal bradycardia. Hypoxia Index is calculated, when the bradycardia does not disappear after taking lateral posture, then the Index should be kept to be less than 24 until delivery. It will be the way to prevent cerebral palsy. The author is planning a prospective study to reduce cerebral palsy by numeric hypoxia index.

Computerized fetal monitoring

The computerized intrapartum fetal monitoring is convenient and useful by introduction of FHR score with its regression equations to predict Apgar score and UApH [8]. Novel hypoxia index prevents cerebral palsy if the index is less than 24. Normal Apgar score is predicted by the

![Figure 11: Fetal response to sound and light stimulations. Left: Sound stimulation, 1,000 hz, 80 db, 2 sec (arrow), Right: Light stimulation with flush-light, twice (arrows), fetal response was movement and FHR acceleration in each stimulations.](image)

<table>
<thead>
<tr>
<th>Hypoxia Index</th>
<th>Cerebral Palsy</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Yes</td>
</tr>
<tr>
<td>25 or more</td>
<td>6</td>
</tr>
<tr>
<td>24 or less</td>
<td>0</td>
</tr>
</tbody>
</table>

* Chi square test: p=0.000008<0.05, significant difference

Table 4: The relation of cerebral palsy and Hypoxia Index.
duration ratio of FHR acceleration to movement burst (A/B ratio) and artificial neural network computer can predict the probabilities to be normal or pathologic outcome [9,11,14-17]. The author is planning a new fetal computer with FHR score, hypoxia index and frequency spectrum to detect pathologic sinusoidal heart rate [7]. Clinical results were already improved by a centralized computer system to significantly reduce periartum mortality and no cerebral palsy [10].

Conclusion

Fetal movement was studied to objectively and numerically diagnose intrapartum fetus and to reduce cerebral palsy due to intrapartum damage.

References