Fetal Actocardiogram

Author:

Kazuo Maeda

Honorary professor, Department of Obstetrics and Gynecology,
Tottori University Medical School, Yonago, Japan

ABSTRACT

Aims: Fetal actocardiogram (ACG) recorded fetal heart rate (FHR) and movements, to diagnose fetal brain damage by FHR response to fetal movement, to solve controversial problems of cardiotokogram (CTG) and to correctly evaluate fetal states.

Methods: Fetal behavior was determined by 4 ACG parameters, fetal outcome was estimated by the ratio of FHR acceleration duration/fetal movement burst duration (A/B ratio), physiologic sinusoidal FHR was diagnosed by the ACG and FHR frequency spectrum analysis, and the developing mechanism of FHR acceleration and variability were studied to diagnose the brain damage in the loss of FHR variability.

Results: The ACG and frequency spectrum analysis separated physiologic sinusoidal FHR from the true one, and controversial problems in CTG were solved by the analysis of ACG. The loss of FHR variability was the sign of fetal brain damage.

Conclusion: The analyses of FHR and fetal movements in ACG were indispensable for correct fetal diagnosis. Since cerebral palsy (CP) will develop in the loss of FHR variability, C-section should be performed before the loss of FHR variability.

SHORT TITLE: Fetal actocardiogram

KEY WORDS: FHR; Fetal movement; Actocardiogram; A/B ratio; Fetal outcome; FHR variability; Acceleration; Fetal disorders

CONFLICT OF INTEREST: No conflict.

CORRESPONCENCE:

Kazuo Maeda,
Home postal address: 3-125, Nadamchi, Yonago, Tottoriken, 683-0835 Japan
Phone and FAX No.: 81-859-22-6856
E-mail address: maedak@mocha.ocn.ne.jp; j2p4e4@bma.biglobe.ne.jp

TERMINOLOGY

Actocardiogram (ACG): Simultaneous record of FHR curve and fetal movement signals.

Cardiotocogram (CTG): Simultaneous records of FHR and uterine contraction curves.

Actocardiotokogram (ACTG): Simultaneous records of FHR, fetal movement signal and uterine contraction curve. Commercial Actocardiogram belongs to this type. You can use it as a Cardiotocogram (CTG) or Actocardiogram (ACG) according to your situation or in your application of an ACTG.

Fetal movements: The ACG motion signal developed by the fetal chest movements, because the ultrasound targeted the fetal heart. The motion also recorded fetal respiratory movements, fetal hiccupping, and fetal chest motion caused by extremity and mouthing movements, which were conducted to fetal chest.

Fetal heart rate (FHR): More than 100 Hz Doppler signals developed by fetal heart beats were processed by an autocorrelation FHR meter, which detected the FHR up to 210 beats per minutes (bpm).

Uterine contraction: It is simultaneously recorded using external tocodynamometer for the diagnosis of FHR deceleration.

FHR baseline: Continuous FHR tracing without acceleration or deceleration. Normal baseline is 110 to 159 bpm.

Fetal bradycardia: The baseline FHR less than 110 bpm.

Fetal tachycardia: The baseline FHR of 160 or more bpm.

FHR variability: Minor variation of FHR baseline (long term variability, LTV) in the FHR recorded by ultrasonic Doppler autocorrelation FHR meter. The LTV amplitude is normal when it is from 5 to 24 bpm.

Acceleration: Transient FHR increase for 15 or more bpm and 15 or more sec is normal in 30 or more weeks of pregnancy, while 10 or more bpm and 10 or longer sec is normal in 30 or less weeks of pregnancy.

Deceleration: Fifteen or more bpm deep and 15 or more sec long transient FHR decrease. V-shaped slow deceleration is a periodic deceleration including early (ED) and late (LD) decelerations. The lag time is 20 or longer sec to uterine contraction in LD. U-shaped irregular deceleration with sudden decrease and recovery is variable deceleration, divided into mild, moderate and severe variable deceleration (MVD and SVD). The LD and SVD are pathological in the pattern classification.

A/B ratio: Total sum of acceleration duration ratio to the total sum of fetal movement duration in the study period.

1. INTRODUCTION

Although fetal movements were evaluated by maternal perception or the motion detected by mechanical actograph at maternal abdominal surface in old time, the results were intolerable to scientific study [1]. A direct detection of fetal movement at fetal surface was required, and the device was invented by Maeda in 1984 [2]. Although the CTG was diagnosed mainly by FHR pattern classification [3], several vague problems and diagnostic difficulty developed in the past. It was, therefore, necessary to analyze the CTG with quantified techniques and numeric evaluation, and the result was the creation of the FHR score [4]. Still, there was the difficulty of differentiation of resting fetal state from non-reactive FHR, the differentiation of physiologic sinusoidal FHR from true ominous sinusoidal FHR, and so on, which were related to the lack of direct fetal movement study. Although some problems were solved by the real-time B-mode ultrasound [5], most diagnostic difficulties were solved by the processing of ultrasonic Doppler fetal movement signals [2]. The prototype actocardiograph was hand-made by Maeda (Figure1 & 2) and reported after the confirmation of its basic properties [2].

2. METHODS

The fetal movements were studied by the ACG. There were various commercial ACG models because the author asked TOITU (Tokyo) to produce a commercial ACG after the success of the ACG prototype. Commercial ACGs are MT-320, MT-325, MT-332, MT-333U, MT-430, MT-516, MT-517, MT-522, MT-540 and MT-610. Most of the commercial models were ACTG, because they prepared uterine contraction record, enabling both studies on ACG or CTG, namely, daily fetal monitoring is performed by visual CTG observation, and problems found in the CTG are solved by ACG function.

Four parameters were quantitatively analyzed in the quantified studies on fetal behavior [2]. Physiologic sinusoidal FHR was differentiated from true ominous one by fetal periodic respiratory and swallowing movements [6]. The developmental mechanism of acceleration and variability was studied by ACG. Short and long fetal outcomes were predicted by the A/B ratio [7]. The prognostic value of the loss of variability was found to develop definite damage of the fetal brain, and it was suggested to perform early delivery before the loss of variability.

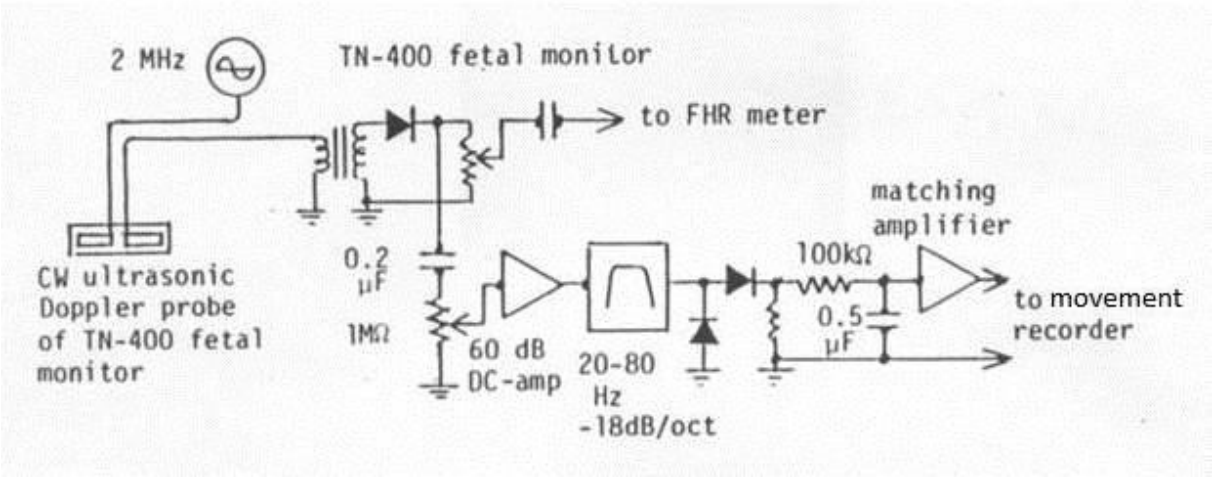

Figure 1. The circuit to record fetal movement in the first actocardiograph, which was planned and hand-made by Maeda in 1983 [1].

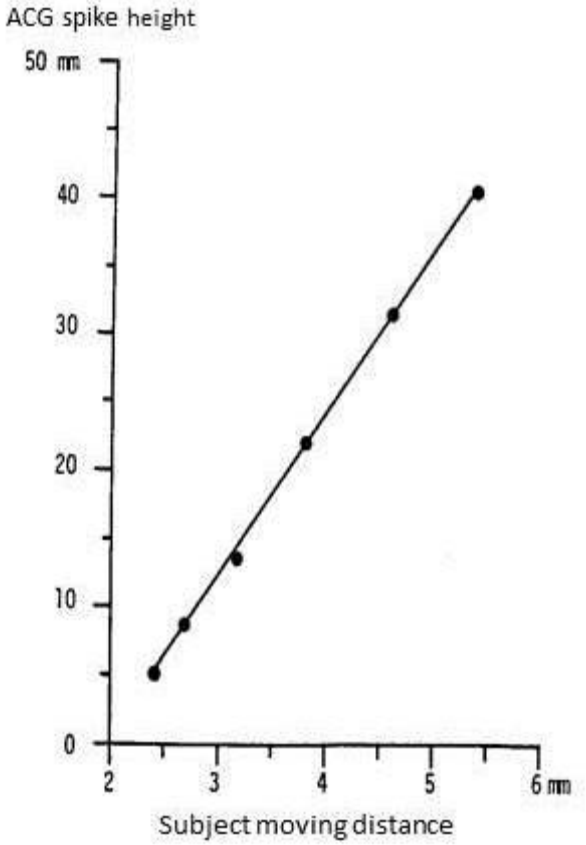

Figure 2. The amplitude of fetal movement spikes recorded at fetal ACG chart was linearly parallel to the moving distance of subject by a steel ball moved in the water. The ACG precisely records the movement of the subject [1].

3. RESULTS

Differential diagnosis of physiologic sinusoidal FHR

Frequency spectrum analysis of FHR traces

Since the FHR record is composed of various frequency signs, e.g. straight baseline, the acceleration, deceleration, sinusoidal change and long term variability, the fast Fourier transform (FFT) frequency analysis was suitable in the quantitative FHR analysis.

The main subject of the frequency analysis was the differentiation of true ominous sinusoidal FHR from physiological benign sinusoidal change. The true ominous sinusoidal FHR was diagnosed when the low frequency area to total spectral area (La/Ta) ratio was 39% or more and at the same time peak power spectral density (PPSD) was 300 or more bpm^2/Hz, while the values were lower in the physiologic benign sinusoidal FHR than true one [9]. In addition, the loss of LTV less than resting fetal state was diagnosed when the La/Ta was less than 15% and the PPSD was less than 60 bpm^2/Hz [10].

Physiologic sinusoidal FHR separated from true ominous one by the ACG

The CTG did not differentiate physiologic FHR from the truly ominous one. A physiologic one was easily diagnosed to be harmless in fetal ACG when the periodic fetal movements synchronized to the sine wave-like FHR, e.g. periodic fetal respiratory or mouth movements provoke physiologic sinusoidal FHR (Figure 3) [6], because the FHR increased when the fetus moved, and the FHR was parallel to the moving fetal body.

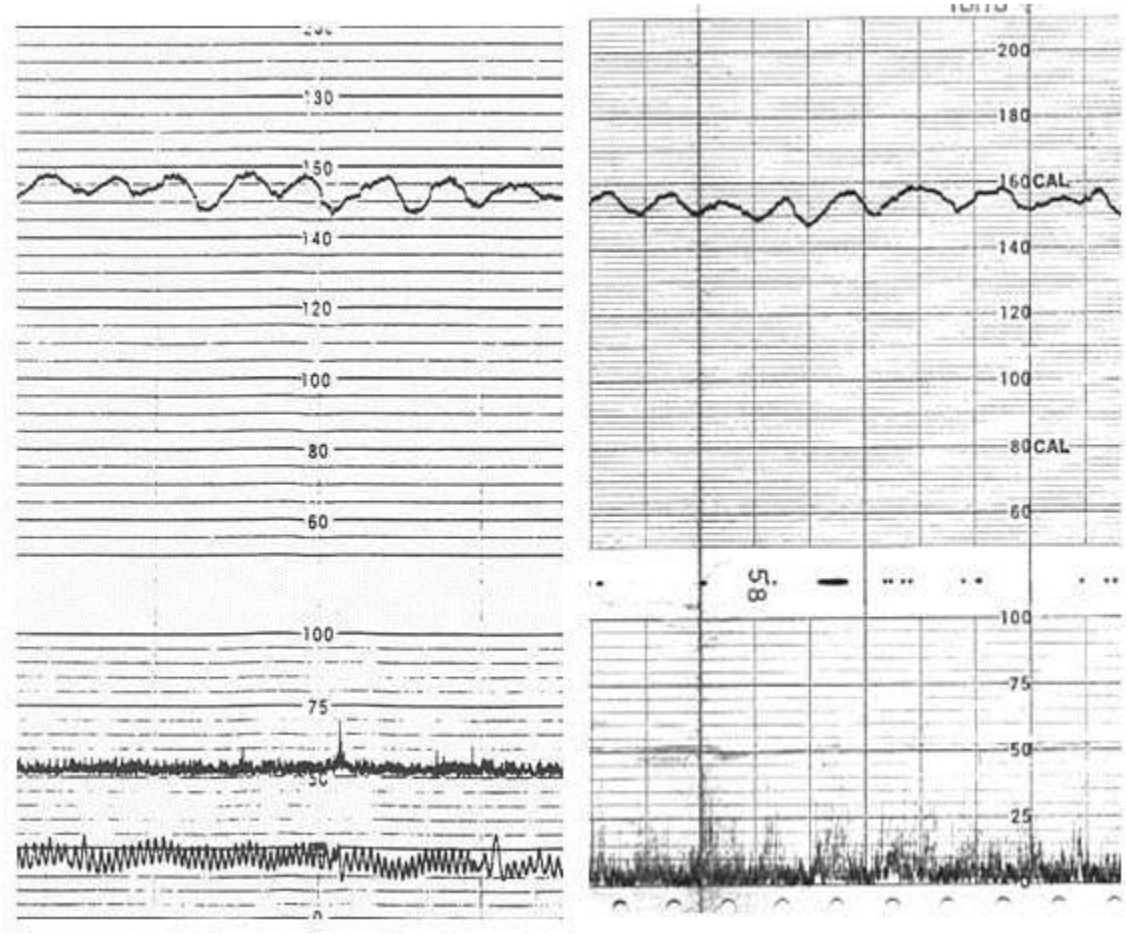

Figure 3. Differentiation of ominous sinusoidal and benign physiological one by ACG [6].
Left: True ominous sinusoidal heart rate. The case outcome was fetal death.
Right: Benign physiologic sinusoidal heart rate. It was separated from ominous one (left) by the synchronization of FHR curve to periodic fetal respiratory movements recorded on the ACG.

Quantified diagnosis of fetal behavior by the ACG

Visual diagnosis of fetal behavior with actocardiogram

Resting fetal state is visually diagnosed when there is no acceleration, and no fetal movement was registered, where the baseline variability was preserved (Figure 4).

Active fetal state is characterized by frequent FHR acceleration, which is synchronized to fetal movement bursts, and baseline variability amplitude is 5 to 24 bpm (Figure 5).

Highly active fetal state is diagnosed by long duration of acceleration synchronized to long lasting fetal movements. The longest acceleration lasted for about 5 minutes (Figure 6). Intermediate state showed rare FHR accelerations but not zero and accompanied rare fetal movement burst (Figure 7) [2].

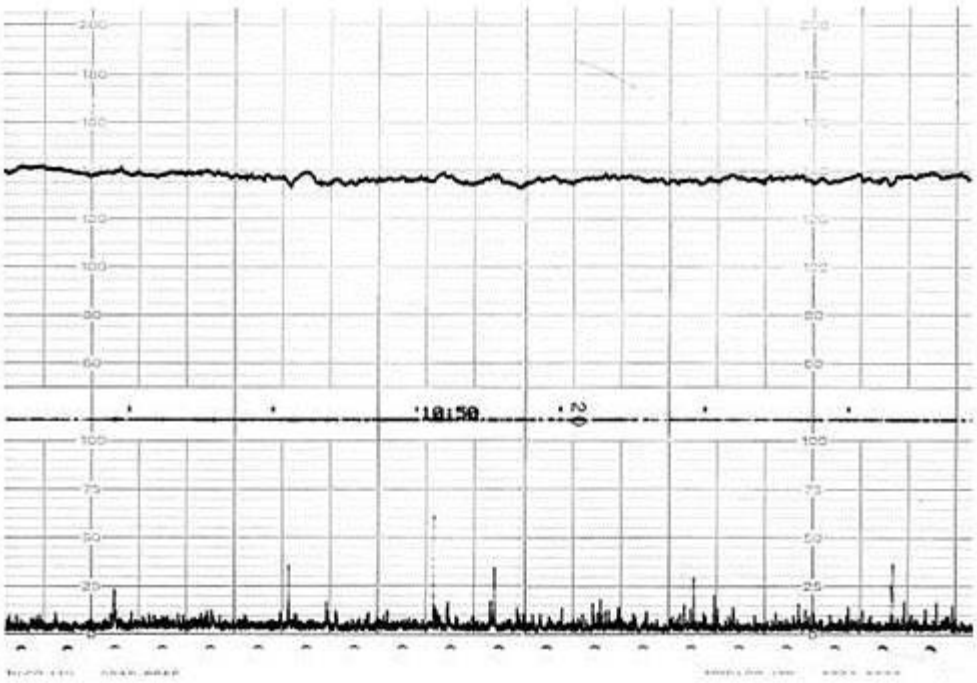

Figure 4. Resting fetal state ACG

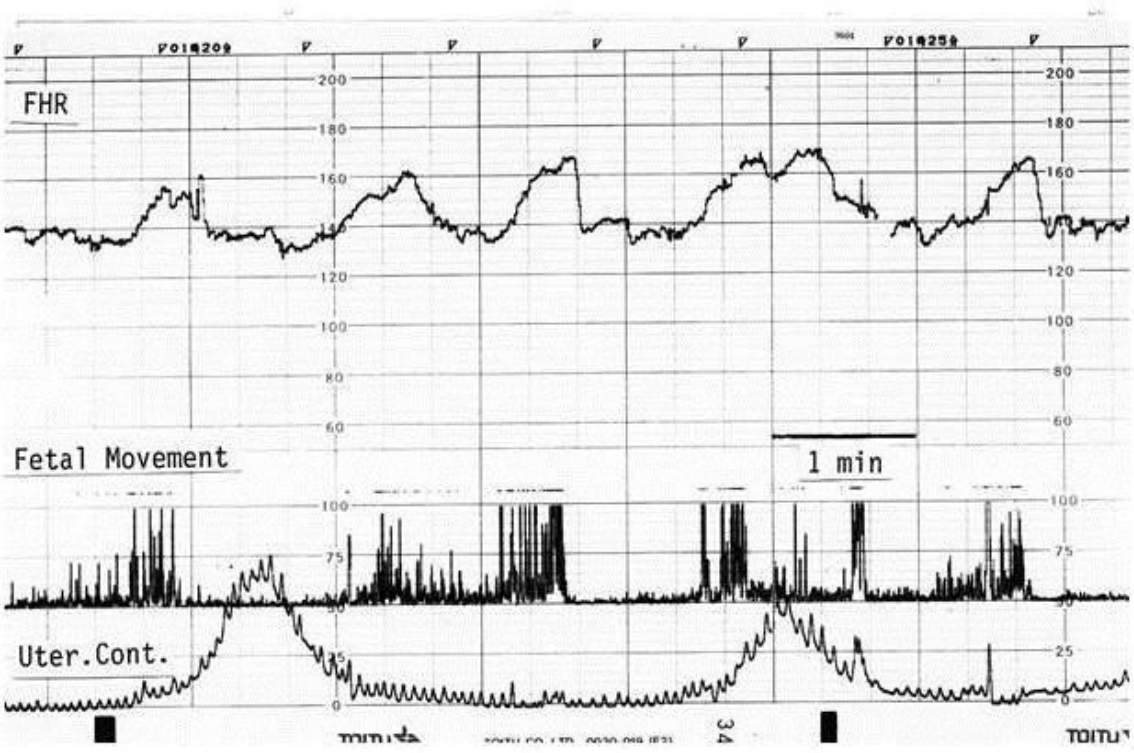

Figure 5. Active fetal state ACG

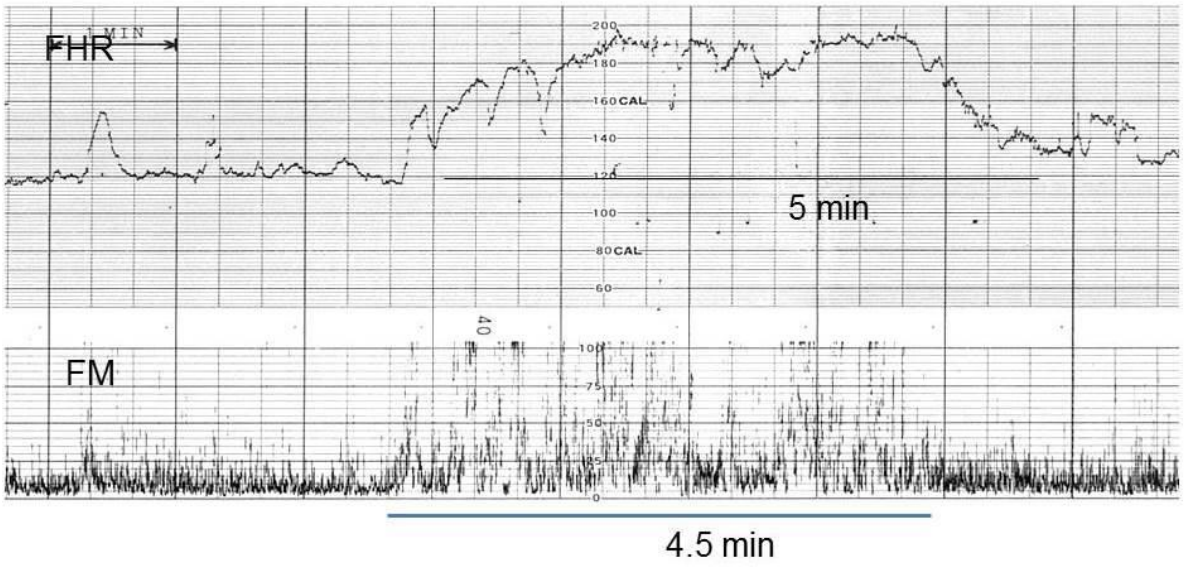

Figure 6. Highly active fetal state ACG

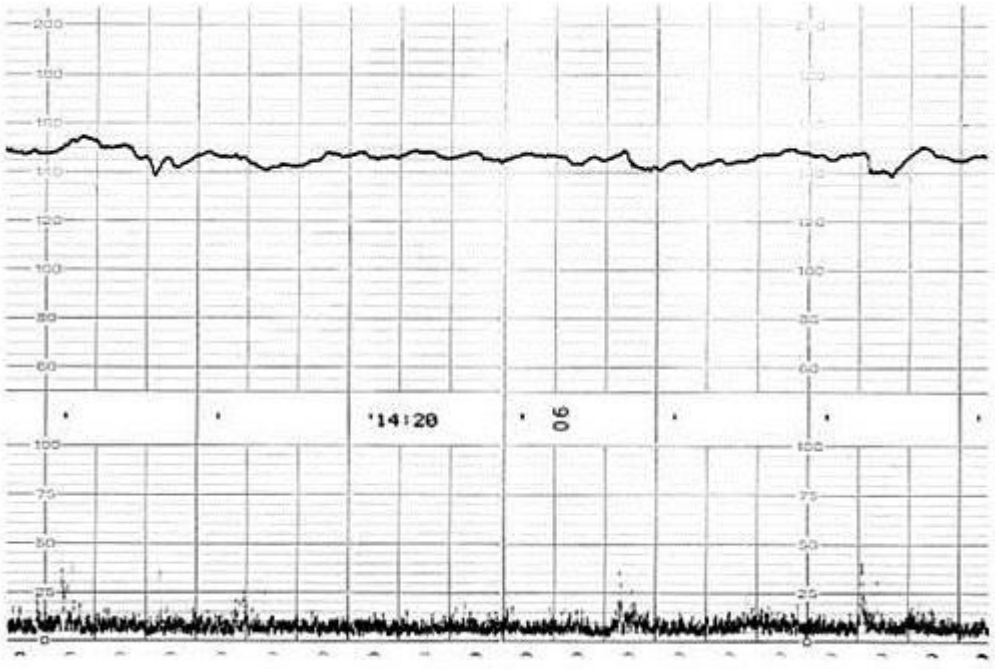

Figure 7. Intermediate fetal state ACG
Very small acceleration and movement were recorded.
It is intermediate between active and resting states.

Quantified analysis of fetal behavior [2]

The fetal ACG was analyzed by four parameters as follows:

Four fetal movement parameters:

1. Burst duration (sec): Mean duration of fetal movement burst.

2. Occupancy (%): Percentage of the sum of movement burst durations against the whole analysis time.

3. Frequency (cpm): The incidence of movement bursts in one min.

4. A/B ratio (%): The ratio of the sum of acceleration durations to the sum of movement burst durations.

Four parameter values were determined in the fetal ACGs of normal fetuses in the late stage of pregnancy (Table 1). Fetal behavior will be determined by 4 parameters in new cases. The importance of A/B ratio was confirmed, because it is not influenced by fetal behavior in the evaluation of fetal statuses.

Difference of ACG parameters in normal fetus and fetal hypoxia

There was a significant difference of quantified parameters between normal and hypoxic fetuses, i.e. occupancy was 32.67% in normal fetus and 10.00 % in hypoxia, frequency was 0.65 and 0.24 cpm, A/B ratio was 1.03 and 0, respectively [11].

Table 1. Quantitative values in four fetal behavioral states detected by fetal ACG. The behavioral state of a fetus will be determined by comparing the ACG data to this table. The A/B value was constant among the behavioral states of normal fetus, i.e. the A/B ratio will be feasible to evaluate the fetal states.

Behavioral States	Duration (sec)	Occupancy (%I	Frequency (cpm)	A/B ratio (%)	N
Resting	0	0	0	0/0	12
Active	29.7±10.3	32.7±14.8	0.7±0.2	1.4±0.4	14
Intermediate	17.7±3.7	6.4±1.8	0.2±0.03	1.2±0.3	5
Highly active	88.6±14.6	44.1±5.3	0.5±0.3	1.2±0.1	3

Fetal hiccupping movement

Movement spikes repeated with 2-3 sec regular intervals in fetal hiccupping (Figure 8). Hiccupping duration was 20-30 min, where some cases repeated it twice in a day. No FHR acceleration was recorded in the hiccupping; therefore, it should be differentiated from non-reactive heart rate.

The spikes are very regular and each spike is sharp in fetal hiccupping, ACG records hiccupping movements in the actogram, while the hiccupping spikes were not recorded in the CTG.

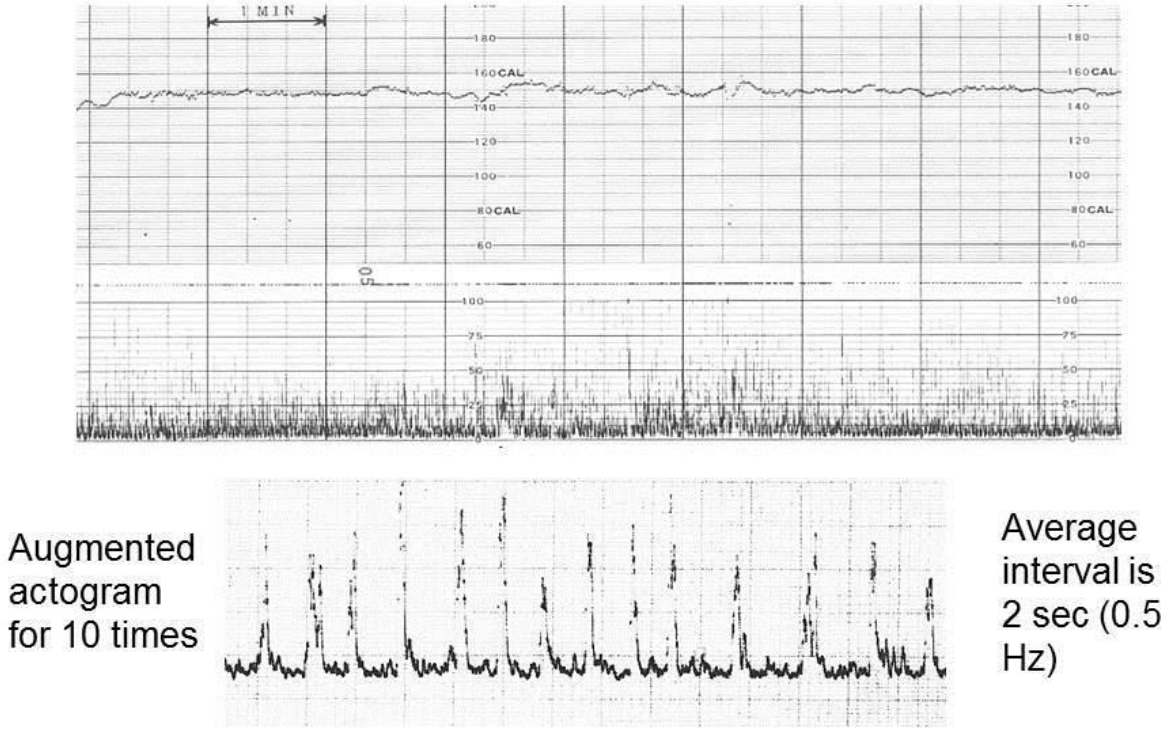

Figure 8. Fetal hiccupping movements.

Developmental mechanism of FHR acceleration and variability in the ACG

Acceleration

Fetal motion increases heart rate as has studied in human physiology. It was clear also in the fetus, i.e. a fetal movement burst accompanies triangular FHR acceleration (Figure 9), where the movement precedes acceleration for approx. 7 sec. The acceleration was lost in non-reactive FHR, which follows severely hypoxic FHR changes after some weeks in fetal growth restriction (FGR) [12], where fetal asphyxia was composed of fetal bradycardia, late deceleration and the loss of variability, and accompanied neonatal deaths [12].

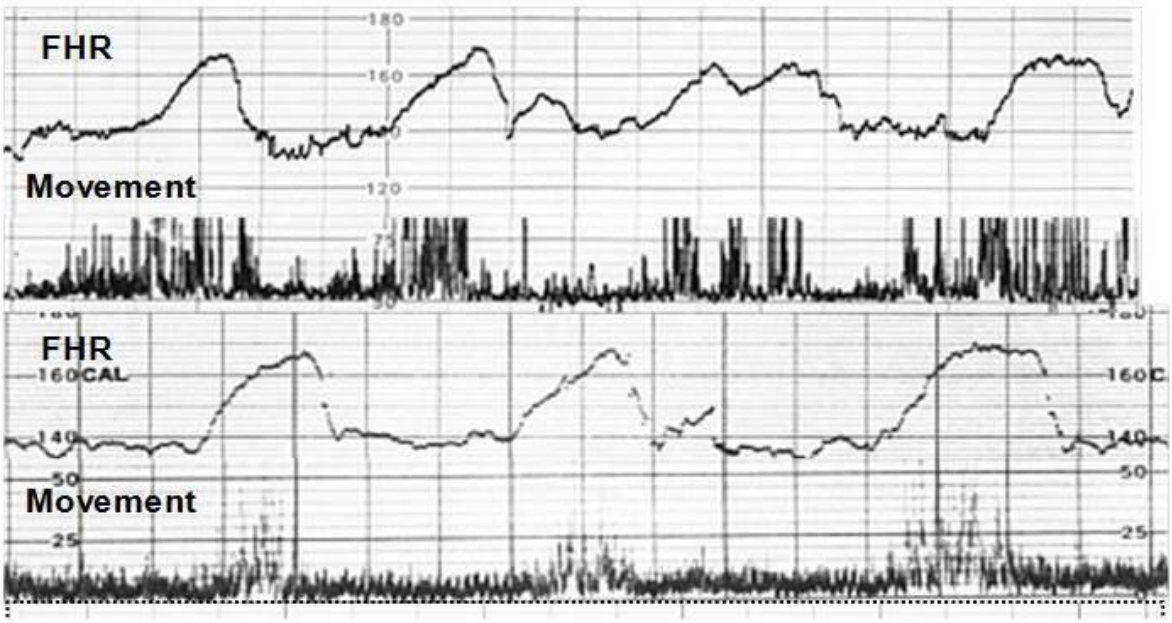

Figure 9. Two normal active fetal state cases characterized with triangular FHR accelerations, which synchronized to fetal movement bursts, that is grouped fetal movement spikes.

The mechanism to develop triangular acceleration:

A triangular curve developed from 10 Hz electric wave bursts after passing through an integral circuit with 7 sec time constant (Figure 10). Also triangular heart rate curves developed in the 1 min continuous leg motions in an adult man (Figure 11) [13]. The electric and physiologic simulations explained the developing mechanism of triangular FHR, which was the reaction of the fetal brain to the movements, in the mid brain [14], which is objectively detected non-reactive FHR caused by the initial fetal hypoxia, preserving FHR variability (Figure 12). Some weeks later after the loss of acceleration, severe fetal asphyxia appeared in FGR case, which was the same as the ACG of an anencephalic fetus (Figure 14). As minor fetal movement provoked FHR variability (Figure 13), the loss of variability will be severe fetal brain damage, which is the same as the loss of the brain in anencephalic fetus (Figure 14) The fact shows that the acceleration develops in the brain of a normal fetus, where triangular acceleration is the product of integral circuit of 7 sec time constant situated in the normal midbrain, then the acceleration is lost in the mildly damaged brain of the hypoxic fetus. The loss of variability will be severe fetal brain damage, as severe as anencephalic fetus.

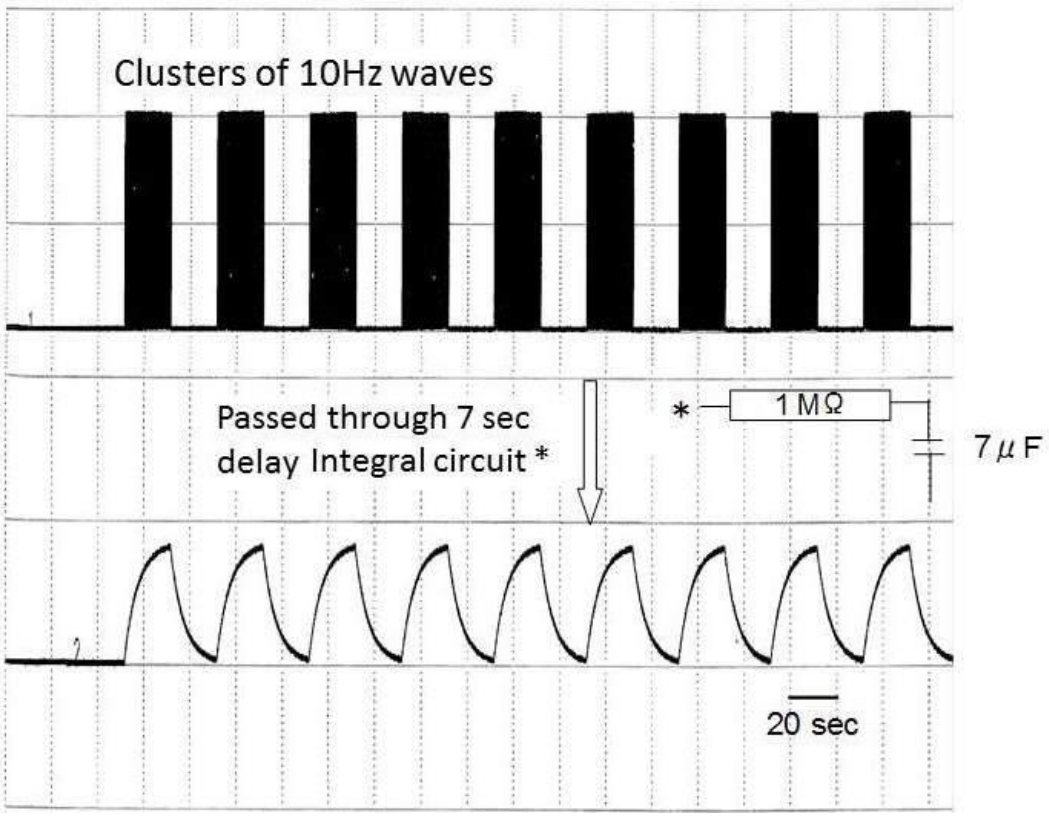

Figure 10. Electronic simulation to develop triangular acceleration from grouped spikes passing through an integral circuit of which time constant was 7 sec.

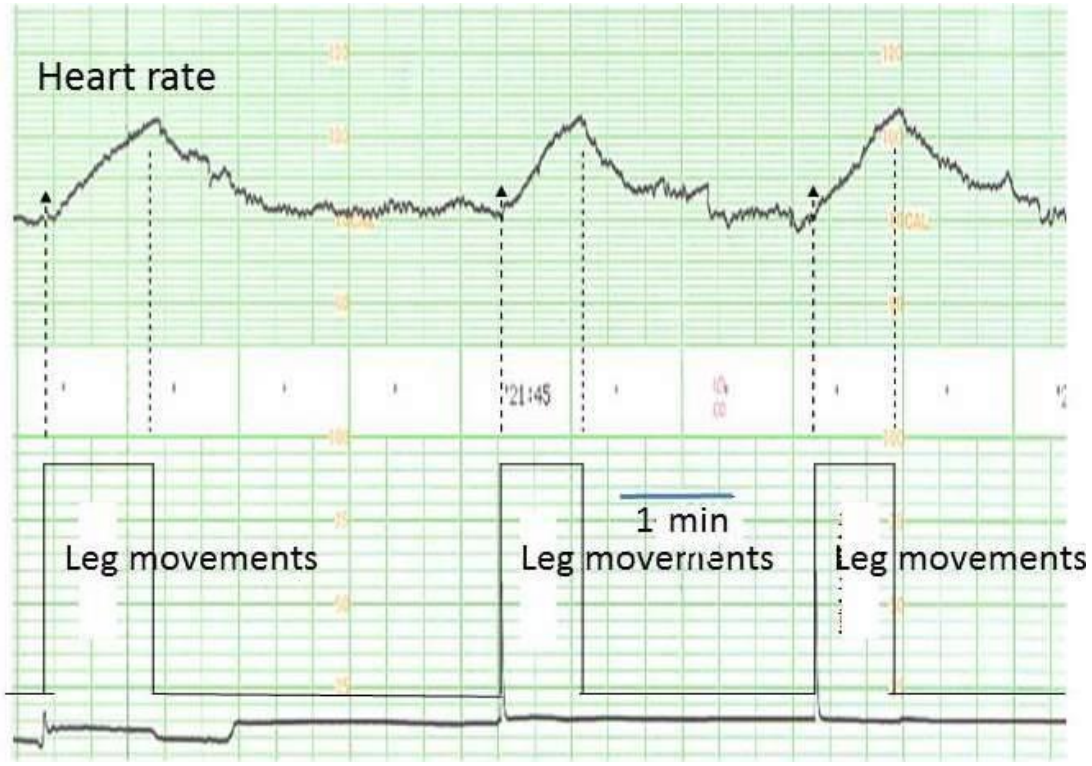

Figure 11. Physiologic simulation to develop triangular heart rate by moving an adult leg for 1 min repeatedly.

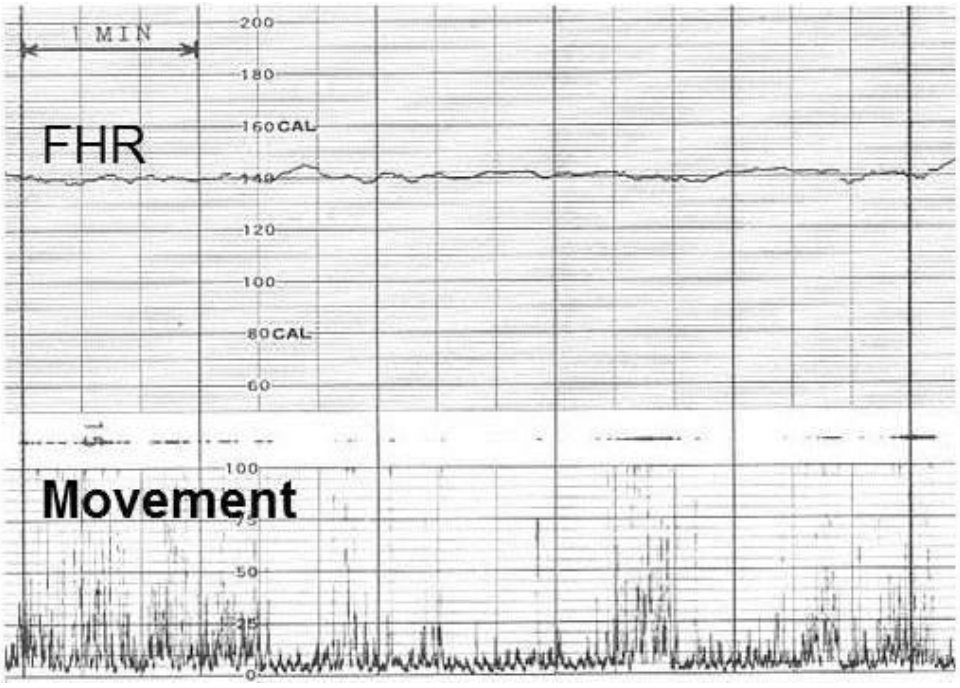

Figure 12. Loss of FHR acceleration （＝Non-reactive FHR）
No acceleration was recorded, preserving baseline variability.
Severe asphyxia appears within 2 weeks in FGR [12].

The mechanism to develop FHR variability in the brain:

Fetal movement group (movement burst) provokes FHR accelerations (Figure 9), and minor fetal movements provoke FHR baseline changes (Figure 13). Since the long term variability (LTV) develops as the response of fetal brain to minor fetal movements, the location to respond the movement is the same as FHR acceleration, i.e. it is the midbrain [14].

The loss of FHR baseline variability

Anencephalic fetus shows neither FHR acceleration nor variability due to the loss of brain (Figure 14). Severe fetal asphyxia, caused by hypoxia, develops the loss of variability, which was the same as the loss of variability in anencephalic fetus, namely fetal brain is totally damaged as anencephalic fetus (Figure 15), therefore, neurological sequel such as cerebral palsy will appear in the loss of variability. Therefore, the fetus should be delivered before the loss of variability. The timing of early delivery will be the loss of acceleration, decreased FHR variability, or very severe change of FHR pattern. The hypoxia index of 20-24 will indicate the C-section, because the hypoxia index of the loss of variability was 25-26 in Maedas collection. Hypoxia index is the duration of bradycardia (min) x 100 / bradycardia nadir (bpm), where bradycardia FHR is used instead of PaO_2, because the heart rate is parallel to the heart rate in the hypoxic rabbits (Figure 19).

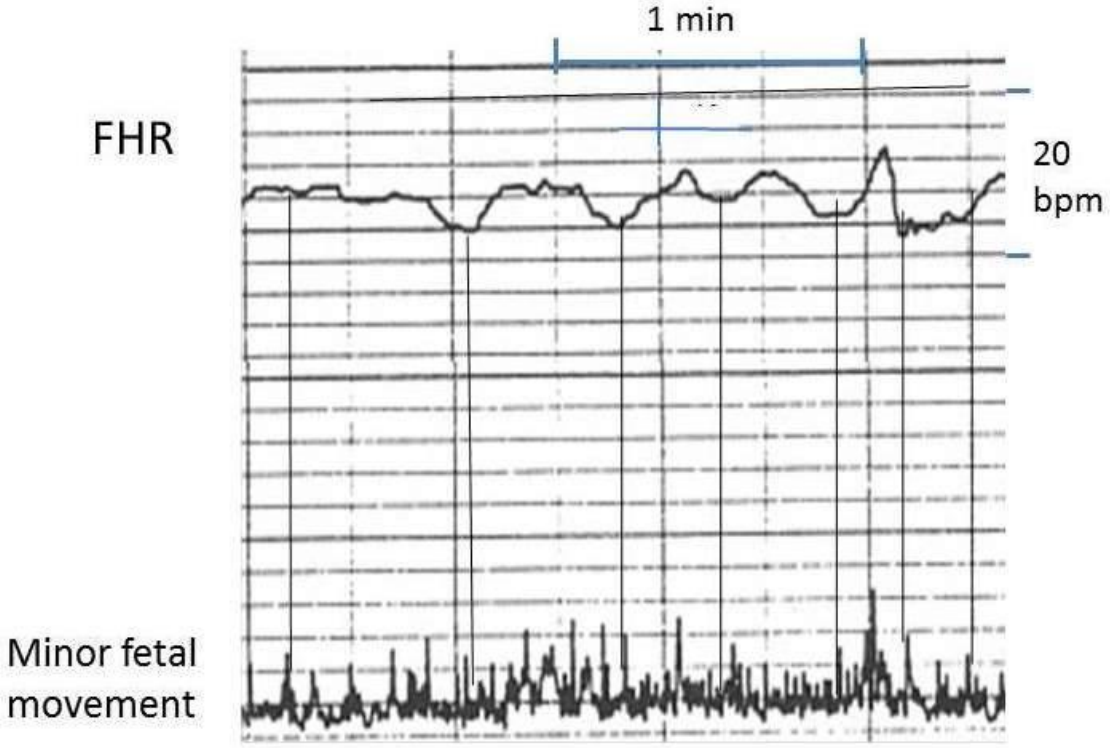

Figure 13. The FHR variability (upper line, LTV) forms variation with the minor fetal movements visualized in an augmented fetal ACG.

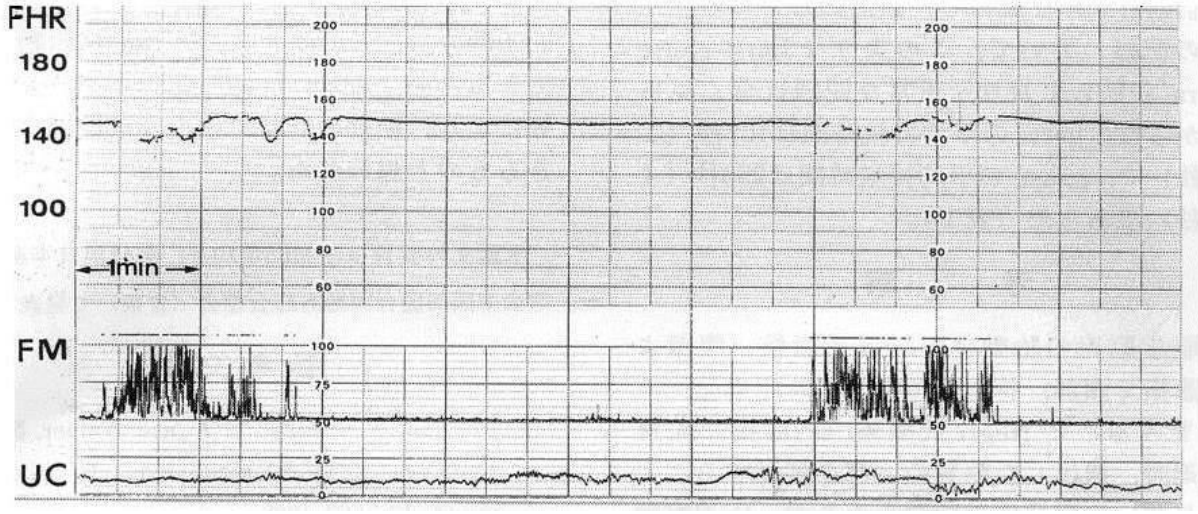

Figure 14. The fetal ACG of an anencephalic fetus. The variability (LTV) is lost. There was no acceleration against the frequent fetal movement.

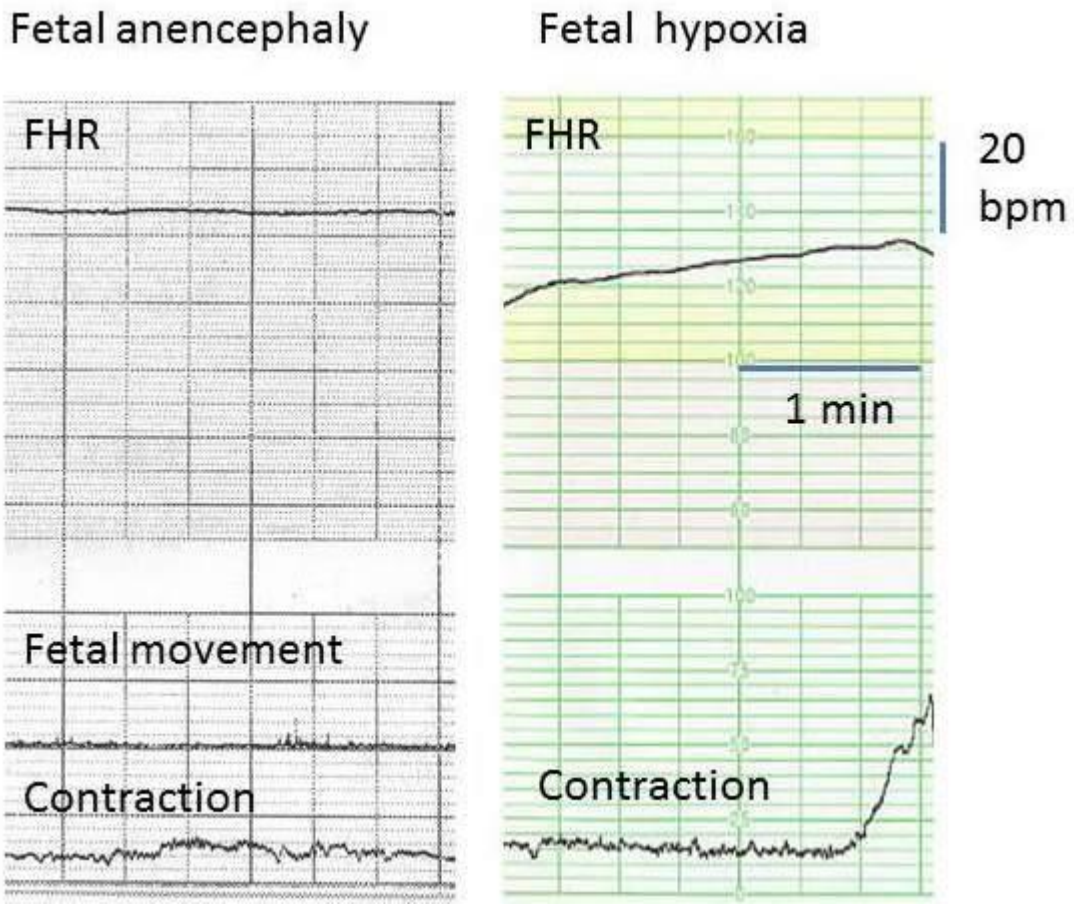

Figure 15. The loss of variability

Left: The loss of variability in a fetal anencephaly.

Right: The loss of variability in severe fetal asphyxia and brain damage. States of variability are the same in both FHRs.

Prenatal diagnosis of quantitative outcome using the A/B ratio of ACG

The A/B ratio was standardized dividing the total duration of acceleration by the total duration of movement bursts (Figure 16). ACGs of 15 common fetal disorders were quantitatively analysed with A/B ratios (Table 2), and compared to the Apgar scores and the numeric long term outcome after births (Figures 17 and 18) [7].

The short term outcome analysed by 1 and 5 min Apgar score closely correlated A/B ratios, i.e. Y (1 min Apgar) = 7.68X (A/B ratio)-1.75, R^2=0.85, p<0.001, Y (5 min Apgar) = 6.44X+ 0.58, R^2=0.68, p<0.001, and Y (numeric long term outcome) = 6.42X+0.05, R^2 = 0.71, p<0.001. Short and long term outcomes were abnormal, when the A/B ratio was less than 1. The fact was important, because a spastic quadriplegia case was found among cases of lower A/B ratio than 1. Therefore, A/B ratio of ACG is a useful parameter to expect fetal short and long term outcomes [7].

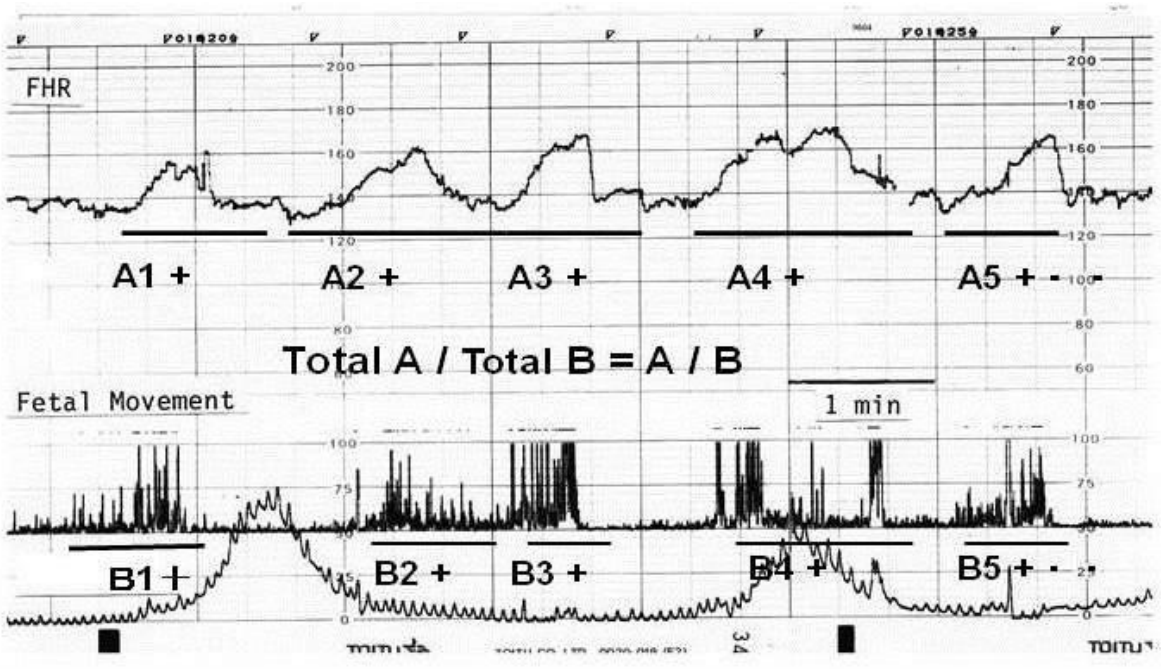

Figure 16. The calculation of A/B ratio in an ACG recorded in active fetal state.

Table 2. Common fetal disorders studied by the A/B ratios in prenatal ACGs

Fetal disorders	pregnancy weeks	A/B ratio	Apgar 1min	Apgar 5min	Long-term outcome
NRFS(LOV)	35	0	0	0	0
18-trisomy	29	0.15	0	0	0
PIH, IUGR, LD	35	0.34	2	9	8
hydrops foetalis	26	0.42	0	0	0
osteodysplasia	29	0.5	1	4	1
excencephaly, multiple anomalies	37	0.54	1	3	3
intestinal obstruction	36	0.96	2	4	6
polydactyly	37	1.01	8	9	9
cardiac sick sinus bradycardia	39	1.34	7	9	8
endocardiac cushion defect	37	1.2	9	9	8
megacystis	38	1.22	8	9	8
myotonic dystrophy	32	1.26	9	9	6
hydronephrosis	39	1.31	9	9	9
normal pregnancy	37	1.43	8	9	10
hydrocephaly	33	1.16	8	9	7
hydrocephaly	30	0.73	3	4	4
spina befida, ventriculomegaly	41	0.88	4	8	6
spina bifida, corp call.part.defect	38	1.11	8	8	8
interrup. ascend. aorta type A	40	1.34	9	9	8
placental abruption	38	1.32	9	9	9

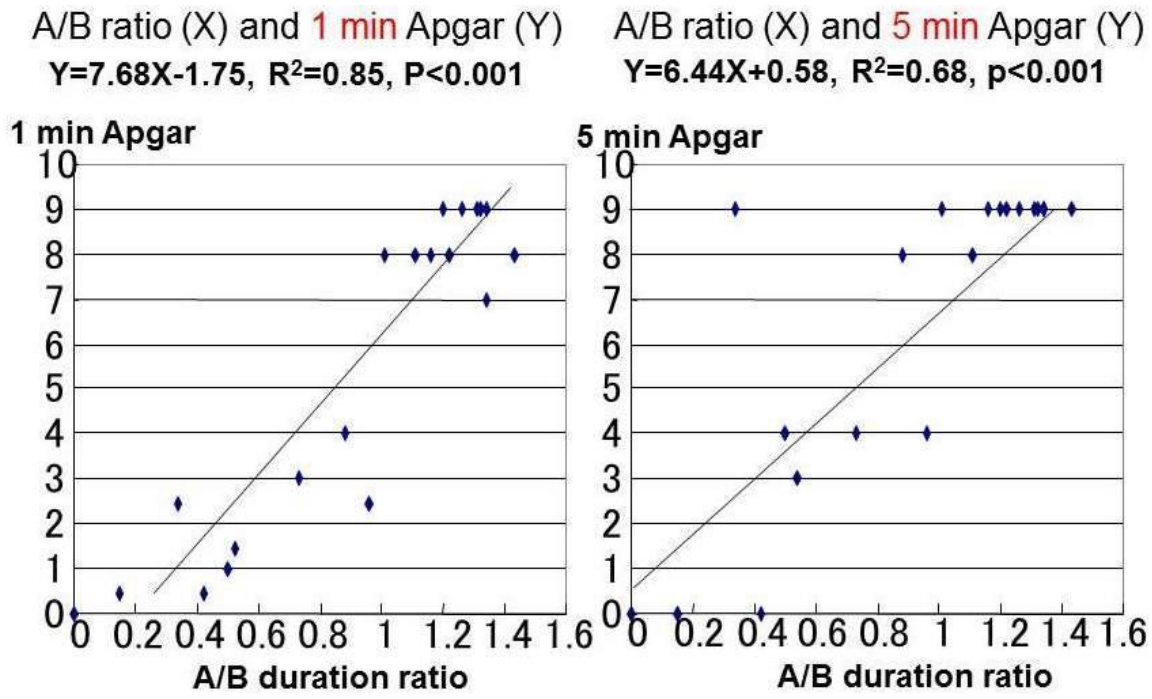

Figure 17. One and 5 min Apgar scores closely correlated the A/B ratio of ACG recorded during pregnancy in cases of fetal disorder (Table 2). Regression equations are listed in the text [7].

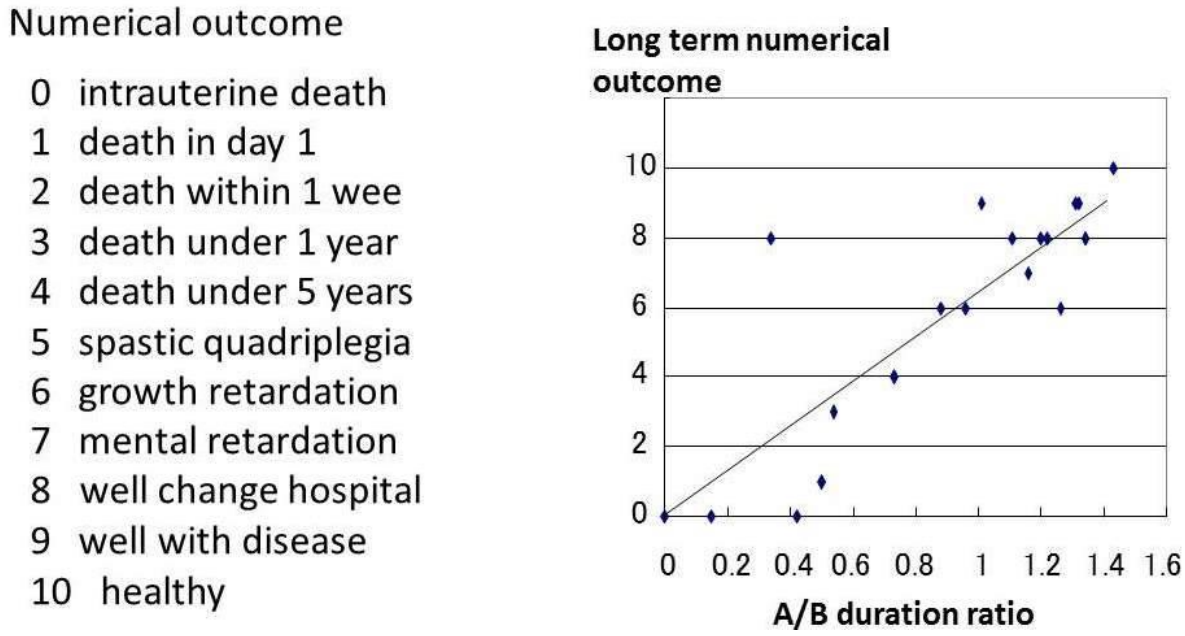

Figure 18. The long term numerical outcome (right) determined using voluntarily determined numerical outcome (left) closely correlated the A/B ratio of ACG recorded in pregnancy [7]. Regression equation is in the text.

Advantage of fetal movement record

Since the height of movement signal of ACG is parallel to those of fetal movement (Figure 2), the influence of fetal movement on FHR is precisely evaluated by the ACG, i.e. fetal movement precedes FHR change, e.g. periodic fetal movements provoked sine wave-like FHR changes (Figure 3), where the physiologic sinusoidal FHR is differentiated from truly ominous sinusoidal heart rate by the ACG. Resting state of the fetus is differentiated by the absence of fetal movement bursts from non-reactive FHR. The loss of acceleration against fetal movement burst shows hypoxic suppression of fetal brain predicting the severe hypoxia (Figure 12).

The role of fetal bradycardia

Although fetal bradycardia has been considered to be the sign of fetal damage, it develops by the excitation of parasympathetic nerve center located at medula oblongata. As no experimental rabbit bradycardia, which developed by nitrogen gas, appeared after uretan anesthesia [15], and apneic bradycardia in anencephalic neonate disappeared by the infusion of oxygenated blood [16], hypoxic bradycardia is a neurologic reaction to hypoxia, but it is no fetal brain damage, but reversible to recover. It was reported that bradycardia appears in the deep diving sea animal, where hypoxic bradycardia may be a damage preventing reaction, but not the sign of brain damage. It may show only fetal environmental hypoxia. In conclusion, the confirmed sign of severe fetal brain damage will be the loss of FHR variability.

Examples of CTG problem solved by ACG

A case received emergency C-section due to late decelerations in the CTG, and placental abruption image found in ultrasound B-mode where neonatal asphyxia was expected and resuscitation was prepared, while no neonatal depression was found after delivery. In a further study of ante-partum fetal ACG, FHR accelerations were found against fetal movement bursts (Figure 20), where the A/B ratio was 1.32 and larger than 1.0, which was the sign of normal outcome (Figure 17). The CTG problem was solved by ACG.

In another case of a sick sinus bradycardia, normal neonate was achieved after C-section, who showed responsive FHR accelerations against fetal movements on the prenatal fetal ACG, and the A/B ratio was larger than one, which was the sign of favorable outcome. The problem in bradycardia in CTG was solved by ACG.

In summary, a favorable outcome was shown by the reactive accelerations of ACG, in spite of abnormal late decelerations and bradycardia, namely, a paradoxical outcome suspected by CTG was solved by ACG in these cases.

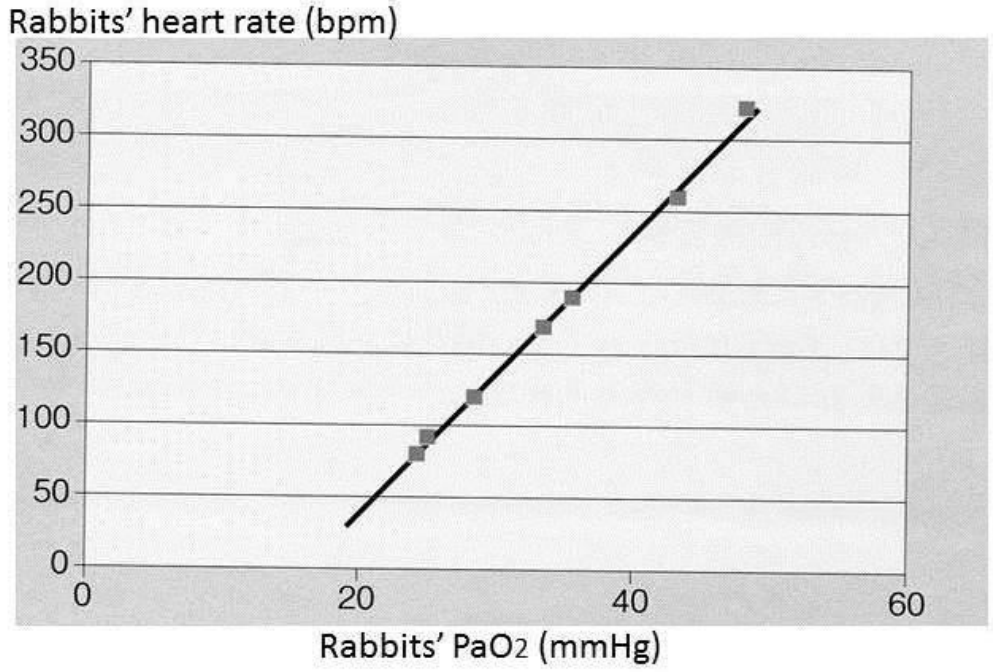

Figure 19. Close relation of heart rate and PaO2 in hypoxic female rabbit [15 & Maeda].

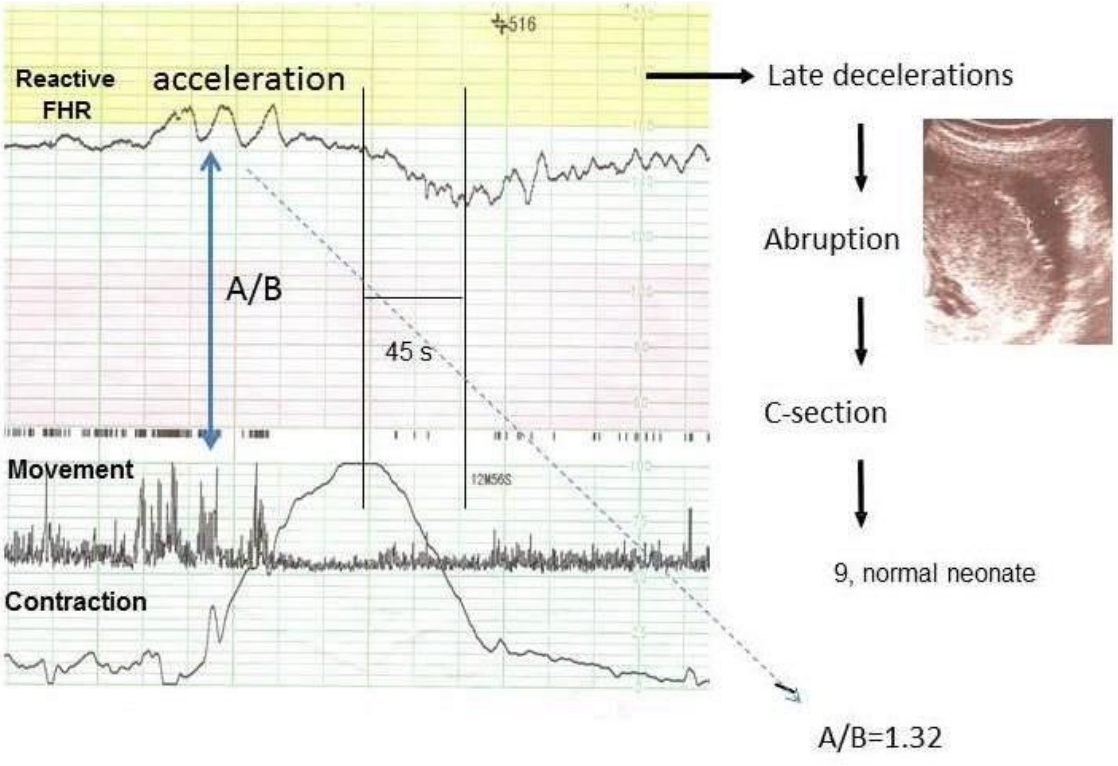

Figure 20. A solution of an unexpected favorable newborn.

Despite the diagnosis of late decelerations and placental abruption, Apgar score was 9, no asphyxia. Prenatal A/B ratio of ACG was 1.32>1, normal outcome could be expected. ACG A/ B ratio was a normal sign of fetal brain.

Computerization of fetal monitoring

The FHR score calculation, neural network analysis and frequency analysis of FHR achieved beneficial results in computerized analyses. Automated computerized classification achieved to estimate the development of fetal behavior [17]. The diagnosis of all components of ACG will be achieved in the future.

4. CONCLUSION

Since the ACG and its quantitative analysis created a new field of fetal evaluation, which is totally objective in quantitative as well as visual analysis of ACG as discussed in this report, the subjective visual FHR pattern classification, which was vague with big interobserver difference, is definitely improved, and the fetal management is greatly progressed by the introduction of quantified FHR analysis and fetal ACG, where C-section was recommended to be performed before the loss of FHR variability. In addition, computerized automatic fetal diagnosis, which utilizes various quantified analyses, will extensively improve obstetric statuses in the future.

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