

FHR Problems Solved by the Actocardiogram

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Abstract

Aims: To clarify the problem related fetal heart rate.

Methods: Actocardiogram was used to analyze the relation of fetal heart rate acceleration, physiological sinusoidal heart rate, long term variability and fetal movement burst, periodic fetal respiratory movements as well as minor fetal movement. The correlation of actocardiographic acceleration duration ratio to movement burst duration (A/B ratio) and fetal outcome was analyzed.

Results and conclusion: Fetal heart rate acceleration was evoked by fetal movement burst, moderate physiological sinusoidal heart rate was evoked by moderate fetal respiratory movement, and the long term variability was evoked by minor fetal movements. Since the loss of variability possibly caused by hypoxic fetal brain damage, Cesarean section is recommended to be performed prior to the loss of fetal heart rate variability. Fetal short term and long term outcome were predicted by the A/B ratio.

Keywords: Fetal heart rate, Fetal large, Medium and minor movements, Acceleration, Moderate and minor heart rate change, Fetal brain damage, Early C-section, Prevention of fetal brain damage, Prediction of fetal outcome, A/B ratio of actocardiogram

Method

Actocardiogram

Although fetal heart rate (FHR) was discussed in the relation to uterine contraction in the EMS or CTG, no fetal movement was studied in EFM or CTG, while the FHR was principally controlled by fetal movement, i.e. adult heart rate increases in any exercise to increase cardiac output by increasing the beating frequency. Also FHR increased by fetal motion, which was recognized by fetal kicking by maternal perception, where the mother was afraid of fetal abnormality when fetal kicking reduced. However, fetal movement signal was not directly compared to fetal heart rate, because there was no precise method to directly record fetal body movement, i.e. maternal perception depends on maternal abdominal skin sensation, and a mechanical fetal motion detector at maternal abdomen was indirect partial record of fetal movements [1]. The direct detection of fetal motion was intended by the author listening noises of the monitor sound of ultrasonic Doppler autocorrelation fetal heart rate monitor, which were listened at fetal motion observed at maternal abdomen. The author had studied fetal heart valve beating signals in the records ultrasonic Doppler fetal heart detector, and he noted the possibility to record fetal motion directly at fetal chest like as fetal heart beat detection, then he analyzed the Doppler frequency of fetal motion by a real-time FFT frequency analyzer to find fetal motion Doppler frequency to be 20 to 50 Hz when the ultrasound was 2 MHz. Already he studied that fetal heart beat Doppler frequency was higher than 100 Hz including the motion of cardiac valve, fetal heart wall and cardiac blood flow.

The facts promoted him to separate fetal motion Doppler signals from fetal heart beat Doppler signal using a band-pass filter with the -18 dB/Oct attenuation at 20 and 80 Hz frequency, where single ultrasound could simultaneously detect fetal movements and fetal heart beats using ultrasound Doppler technique. The author did it by himself with hand-made remodeling TN-400 ultrasonic Doppler autocorrelation fetal monitor cardiocograph (TOITU, Tokyo) of which ultrasound frequency was 2 MHz, and a commercial -18 dB/Oct 20-80 Hz band-pass filter 1983. The results were successful, he studied basic technical

character and clinical studies on fetal movement detection at fetal chest, and confirmed the linear relation of recorded signal amplitude and that of the movement, then reported the creation of new ultrasonic Doppler fetal movement record to the Journal of Japan Society of Obstetrics and Gynecology in 1984 [2]. World perinatal researchers tested the new machine and guaranteed its function to record every fetal movement [3-5]. The author asked TOITU (Tokyo) to produce commercial new ultrasonic Doppler fetal movement recorder (actocardiogram, named by the author) on 1984, and the first commercial model MT-320 actocardiogram was provided, and followed by MT-325, MT-332, MT-333U, MT-430, MT-516, MT-517, MT-522 and MT-540 (TOITU, Tokyo). The price of MT-516 is about 6,000 USD at present.

Terminology

Actocardiogram: 'Acto' is fetal movement and 'Cardio' fetal heart rate, namely 'Actocardiogram' means simultaneous record of fetal movement and heart rate. 'ACG' is abbreviation of actocardiogram. Uterine contraction is also simultaneously recorded in actual ACG machine to prepare the function of 'Cardiotocogram (CTG)'.

FHR: Fetal Heart Rate.

FHR acceleration: Triangular transient FHR increase. Duration is 15 or more second amplitude 15 or more bpm after 30 weeks, while 10 sec and 10 bpm before 30 weeks.

The loss of acceleration is 'non-reactive FHR' before ACG.

Sinusoidal FHR: Two to 4 cycles of min sine wave-like smooth FHR waves of very ominous outcome. A physiological sinusoidal FHR resembles true sinusoidal FHR, while its outcome is benign without

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fetal obstacle. The true and physiological sinusoidal resembles each other, hard to distinguish by CTG, while physiological one is evoked by periodic fetal respiratory movements in ACG.

Deceleration: transient decreases of FHR. 'Nadir' is the lowest FHR in a deceleration. Periodic V-shaped decelerations are divided into early (ED) and late (LD) decelerations. The LD delays from uterine contraction and considered ominous sign. Variable U-shaped decelerations are divided into benign mild variable and ominous severe variable decelerations. Prolonged deceleration longer than 2 min is ominous. FHR baseline variability: The minor 5 to 24 bpm variation of FHR baseline. The loss of variability is a very ominous sign.

Bradycardia: Decreased FHR baseline less than 110 bpm. An ominous hypoxic sign, particularly sudden and prolonged bradycardia. Two non-hypoxic fetal bradycardia are fetal atrio-ventricular block and the sick sinus bradycardia.

Tachycardia: FHR baseline higher than 160 bpm. More than 200 bpm fetal tachycardia is fetal cardiac pathology which needs pharmacologic treatment via placenta.

NRFS: non-reassuring fetal status, fetal asphyxia, fetal distress.

A/B ratio: Acceleration duration ratio to associated fetal movement burst duration ratio. It was the sum of acceleration duration divided by the sum of movement burst duration in a observation period.

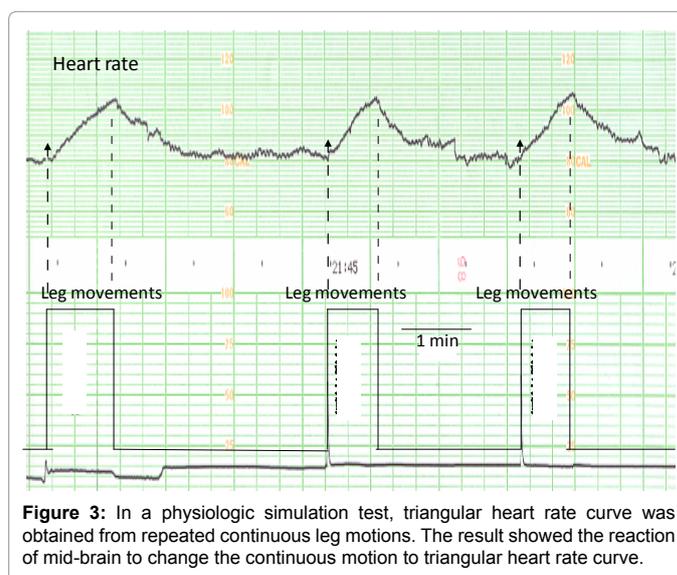
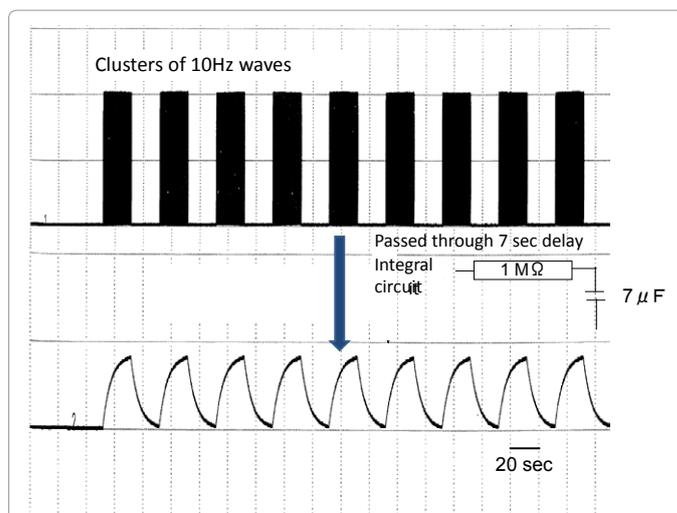
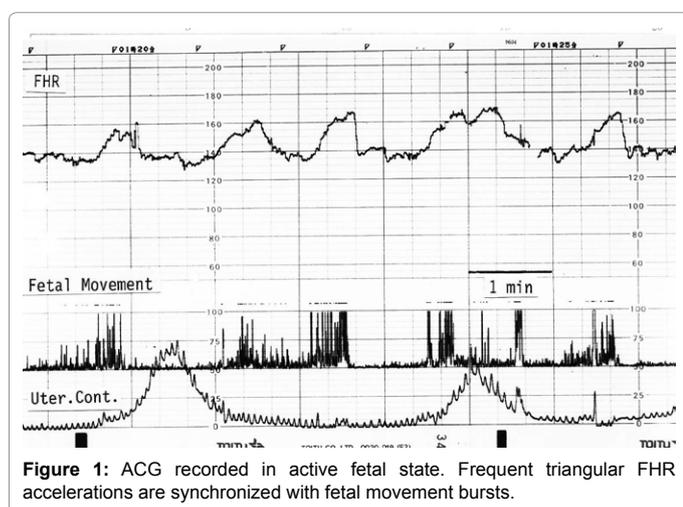
Results

Developmental mechanism of FHR acceleration

Triangular FHR accelerations are accompanied, rather synchronized, with fetal movement bursts in fetal active state (Figure 1). Terao [6] found that the acceleration is produced in the mid-brain in the studies on anencephalic fetus.

Why the acceleration was triangular had not been clarified. Since the movement signal would stimulate the mid-brain to form the triangular acceleration, the correlation of acceleration and fetal movement should be studied, i.e. in the cross-correlation study of FHR and fetal movement signals, the correlation coefficient was the largest, when the movement signal was delayed for 7 sec [7].

Since the 7 sec will mean the processing time of movement burst, an electronic simulation test was studied, where 10Hz wave groups (because fetal movement Doppler of 1MHz ultrasound is 10 Hz) were



passed through an integral circuit, of which time constant was 7 sec (the signal delay is 7 sec). The result obtained was not 10 Hz cluster, but triangular waves similar to FHR acceleration (Figure 2).

Another physiologic simulation test was the record of adult heart rate during continuous constant leg exercise. The heart rate curve was such triangular shape as FHR acceleration (Figure 3). Since the experimental person did not recognize the heart rate change, the transformation of movement to triangular heart rate would be conducted in the mid-brain but not in cortical brain. Therefore, the author confirmed that fetal brain reacted the stimulation of fetal movement with 7 sec delay.

Physiologic sinusoidal FHR (Moderate baseline variation)

Physiologic benign sinusoidal FHR was differentiated from true ominous sinusoidal one by the presence of synchronized periodic fetal

respiratory movements [8]. Maeda traced the envelope of periodic fetal movements, and delayed it 7 sec, where the envelope coincided the physiologic sinusoidal fetal heart rate (Figure 4). The result clearly showed that fetal brain reacted fetal movements.

Long term baseline variability

Furthermore, the actogram of fetal resting state was augmented for 3 times, where minor movement signals appeared even in resting fetal state preserving FHR variability. It may suggest that minor movement stimulate the brain and minor FHR variability developed. Maeda further compared augmented FHR baseline and actogram, where minor fetal movement resembled the small variation of FHR (Figure 5).

Discussion

In summary, large FHR acceleration was evoked by large fetal movement burst, moderate size FHR variation was evoked by moderate

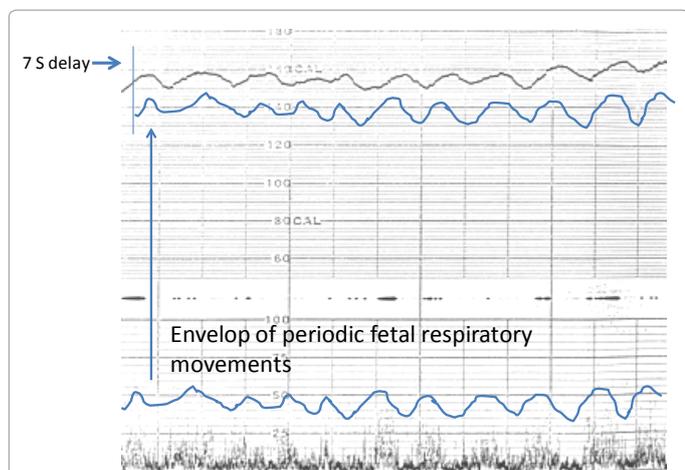


Figure 4: The envelope of periodic fetal respiratory movements was coincided physiologic sinusoidal FHR after the delay of envelope for 7 sec. The result shows the reaction of mid-brain to fetal movements to form moderate amplitude long term FHR variability.

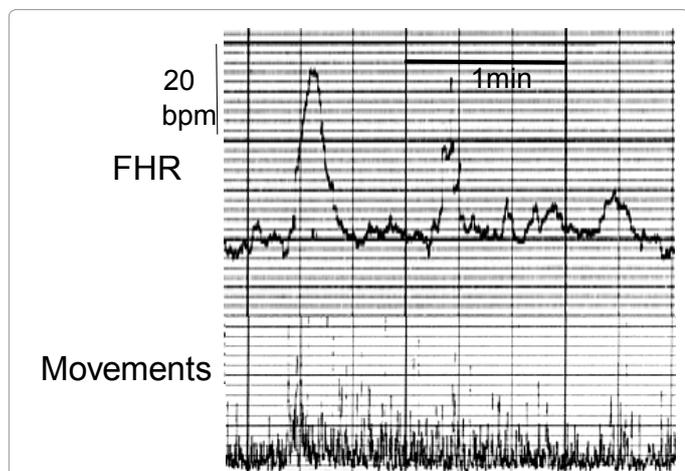


Figure 5: An augmented minor fetal movements provoked small long term FHR variability. Therefore, in summary, large fetal movement burst develop triangular FHR acceleration, moderate fetal movement develop moderate FHR variation, and minor fetal movement develop long term FHR variability by the reaction of fetal brain.

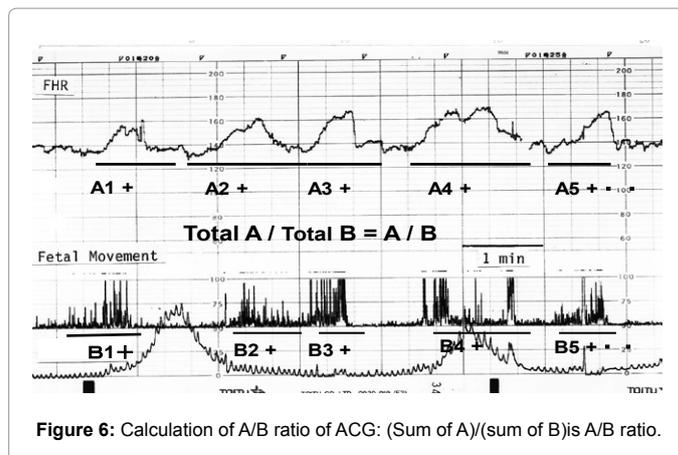


Figure 6: Calculation of A/B ratio of ACG: (Sum of A)/(sum of B) is A/B ratio.

fetal movements, and small FHR variability developed by the minor fetal movements. All transient FHR changes would be evoked by the reaction of fetal brain, probably in mid-brain.

As mentioned above, fetal movements evoke fetal heart rate increase by the reaction of fetal mid-brain. IUGR cases frequently suffer hypoxia, i.e. they frequently develop non-reactive FHR against fetal movement bursts, where FHR baseline variability is preserved. Some days or a week after the non-reactive FHR, severe NRFS appears with bradycardia or severe late deceleration and the loss of variability. Despite emergency C-section was performed, the outcome NTFS was more ominous including neonatal death than reactive FHR of positive acceleration without NRFS [9]. Since the loss of variability is partial phenomenon of fetal brain damage without reaction to fetal movements, there may be general brain damage possibly followed by neurological sequels or cerebral palsy. Therefore, initial loss of acceleration preserving variability will be early stage of hypoxia, and the loss of variability will be advanced severe hypoxia with fetal brain damage. Therefore, early C-section before the loss of variability would cure the fetus from severe brain damages (Figure 6).

Clinical recommendation on the loss of FHR variability

Since the loss of long term FHR variability (LTV) will possibly mean general brain damage due to hypoxia as discussed above, preventive C-section against neurological sequels is recommended to be performed before the loss of LTV, instead of the C-section after the loss of LTV, in general NRFS cases in fetal monitoring. Since the C-section is performed generally in severe NRFS including sudden continuous bradycardia, severe late or variable decelerations, prolonged decelerations, sinusoidal FHR, and so on, please confirm the presence of LTV before the C-section due to NRFS, or confirm that the hypoxic index (bradycardia duration (min) × 100 divided by the nadir FHR) is lower than 24 at the same time as positive LTV, because in severe NRFS, of which LTV may disappear, the hypoxia index was 25-26. The hypoxia index, however, should be further studied in cases of the loss of LTV.

Complicated FHR changes in the hypoxia

As discussed above, FHR shows complicated changes, i.e. the suppression or disappearance of acceleration but preserving FHR baseline variability in early stage of asphyxia, while the variability disappears in advanced hypoxia, according to the severe suppression or damage of fetal brain, while the suppression patterns complicated by the decrease of FHR in continuous bradycardia or deceleration, because

Fetal disorder	Week of pregnancy	A/B ratio	Apgar		Long term outcome
			1 min	5 min	
NFRS(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

Table 1: Twenty Fetal disorders of which outcome was studied by A/B ratios of ACG.

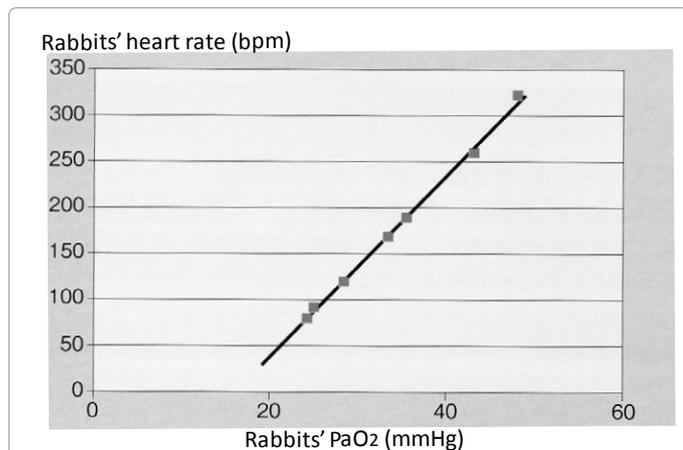


Figure 7: (A) Correlation of A/B ratio (X) and 1min Apgar score (Y) in 20 fetal disorders (Table 1): $Y=7.68X-1.75$, $R^2=0.85$, $p<0.001$, (B) Correlation of A/B ratio (X) and 5 min Agar score (Y): $Y=6.44X+0.58$, $R^2=0.68$, $p<0.001$.

of the excitation of parasympathetic center of the medulla oblongata, which will be a protective reaction of autonomic nerve system activated by low PaO₂ [10], i.e. rabbit heart rate decreases parallel to lowering of PaO₂ (Figure 7). In the most severe hypoxic fetal brain damage, all brain functions will belost,therefore FHR baseline is flat and constant due to the automaty of fetal heart, which may be calledfetal brain death.

Fetal outcome predicted by the A/B ratio of actocardiogram

The acceleration duration ratio to movement burst duration (A/B ratio) was parallel to the severity of fetal central nervous system lesions [11,12], furthermore in common fetal disorders the A/B ratio closely correlated Apgar score and long-term outcome of the infants [13].

One and 5 min Apgar scores were closely correlated to A/B ratios of ACG recorded in prenatal stages (Figure 8). The long term outcome after birth was voluntarily determined into numeric values, which

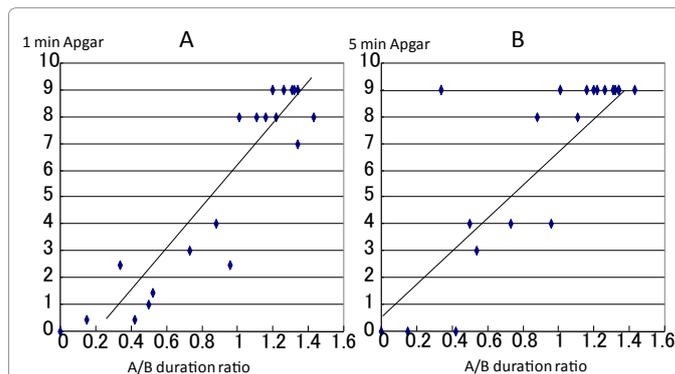


Figure 8: (A) Correlation of A/B ratio (X) and long term numerical outcome (Y). (B) Numerical long term outcome. $Y=6.42X+0.05$, $R^2=0.71$, $p<0.001$.

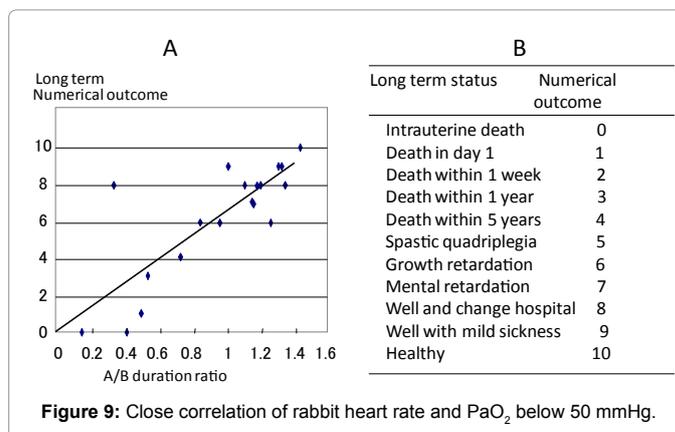


Figure 9: Close correlation of rabbit heart rate and PaO₂ below 50 mmHg.

was closely correlated fetal A/B ratio of ACG (Figure 9). These facts indicate that the A/B ratio of ACG, which was the FHR acceleration durationstandardized by fetal movement, is capable to predict short and long term fetal outcomes, i.e. the A/B ratio would be valuable to

be an important parameter in the fetalB monitoring. Although 'non-reactive FHR' was already reported to be an ominous sign of FHR, more detailed quantitative acceleration duration data, standardized by fetal movement, generally indicate fetal outcomes.

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