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Prevention of Intrapartum Brain Damage with Hypoxia Index and the Problem in Preterm Birth

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Introduction

It was big problem to prevent cerebral palsy caused by intrapartum fetal brain damage, and prevent neurological sequels of preterm delivery. The intrapartum fetal heart rate (FHR) monitoring was almost established to prevent fetal death and child cerebral palsy by the introduction of novel strategies, while it would be still future problem in preterm delivery.

Hypoxia Index in Fetal Heart Rate Monitoring

Fetal growth is restricted by several reasons, e.g. reduced active transfer of placental villi and maternal circulation in the placental intervillous space fibrin deposit [1]. The placental infarction in preeclampsia also reduced oxygen transfer developing fetal hypoxia. Fetal blood PaO_2 is 50 mm Hg or less [2], i.e., the fetus is rather hypoxic if compared to 100 mm Hg PaO_2 of adult. Thus, the fetus tends to be hypoxic, which can damage fetal brain if hypoxia is severe and persisted. As there was fetal death in severe fetal asphyxia, hypoxic fetus was diagnosed in fetal monitoring and cured by early caesarean delivery after the diagnosis of fetal asphyxia, however, cerebral palsy did not reduce in the Dublin RCT of fetal monitoring, which was dissapointed and the reduction of cerebral palsy has been the important problem in fetal monitoring.

In addition, the effect of fetal late deceleration (LD) was vague in fetal monitoring, namely, despite the outcome of LD was reported to be ominous [3], 3 connected typical late decelerations were followed by vigorous neonate, while highly repeated LD resulted the loss of fetal heart rate variability **(Figure 1)**, which was severe fetal brain damage experienced by the author. The unstable LD results were discussed. As Poseiro et al. [4] reported that LD was caused by the loss of placental maternal blood flow due to the compression of illiac artery with contracted pregnant uterus in supine posture; the LD was caused by external mechanical reason, where the hypoxia was caused by repeated decelerations. Actually, an LD is defined after the repetition in 15 min, which might include 6 decelerations, but it was not characterized by the lag time to uterine contraction. Thus, theoretically, fetal heart rate deceleration should be evaluated by its repetition defined by the sum of deceleration duration. The hypoxic state is the same in sudden continuous fetal bradycardia. Fetal hypoxia is discussed by fetal heart rate but not by fetal PaO_2 in this report, because rabbit heart rate was fully parallel to PaO_2 when PaO_2 was lower than 50 mm Hg [5] and human fetal PaO_2 was 50 mm Hg or less [2] and fetal blood sampling to measure fetal PaO_2 is unable during labor.

Thus, Hypoxia Index (HI)=the sum of fetal heart rate deceleration duration (min)/the lowest fetal heart rate (bpm) and multiplied by 100. The deceleration duration was divided by the lowest fetal heart rate, because the lowest heart rate shows hypoxic intensity, thus the shape of transient bradycardia (deceleration) was similar to area of transient hypoxia. HI is objective numeric index of fetal hypoxia. The author's intention to numerically measure fetal hypoxia was satisfied by the hypoxia index.

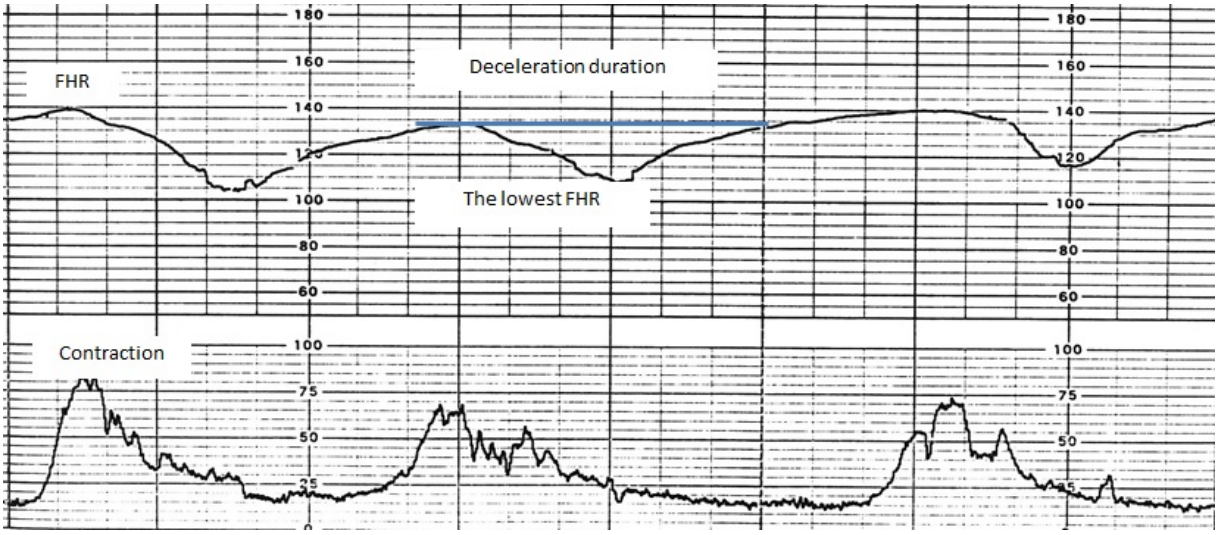

Figure 1 A typical late decelerations repeated in 50 min due to refusal caesarean delivery, where hypoxia index was 26, FHR variability was lost similar to anencephalic fetus. Neonatal Apgar score was 3, severe neonatal asphyxia and severe brain damage expiring 3 months after birth by cerebral hemorrhage.

The nature of hypoxia index was overlapping effects of hypoxia in repeated transient bradycardia (deceleration) not only in late deceleration, but also in all decelerations including early, late and variable decelerations; in addition sudden continuous fetal bradycardia is evaluated by the hypoxia index.

The hypoxia index (HI) was 24 or less before births in 16 cases of FHR changes but FHR variability was normally preserved, while the HIs of repeated decelerations which associated the loss of fetal heart rate variability were 25 or more in 6 cases of fetal brain damage and cerebral palsy.

As fetal heart rate variability developed by fetal brain reaction to minor fetal movement, and the loss of heat rate variability was the same as anencephalic fetus, the loss of varisbility was severe fetal brain damage followed by cerebral palsy. Two groups, where HI was 25 or more and 24 or less showed significant difference in the formation of severe brain damage and cerebral palsy, by the Chi square test. Thus, it is recommended to deliver the fetus before the loss of variability, where hypoxia index was 24 or less, because the cases who received caesarean delivery after the loss of variability developed cerebral palsy, whose HI were 25 or more [6].

Thus, the cerebral palsy caused by intrapartum hypoxia will be prevented by the calculation of hypoxia index and keeping it at 24 or less at delivery before the loss of variability [6]. The early delivery is recommended, when the FHR acceleration is lost and baseline variability amplitude reduced to 5 bpm assisting the hypoxia index.

Although maternal characters influenced neonatal aorta structure [7] the hypoxia index of fetal heart rate diagnosed ominous outcome in the present study with the definite threshold value of hypoxia index.

The Problems in Preterm Birth

The neurological sequels of preterm delivery are another cause of brain damage. Its incidence will be higher than intrapartum damages of full-term births, namely, 18% of periventricular echo-density (PVE) of preterm labor fetuses changed to periventricular leukomalacia (PVL) in neonatal period and followed by cerebral palsy **(Figure 2)**, if the PVE lasted until preterm delivery, thus it is better to prolong pregnancy until full-term delivery by suitable tocolysis, because there was no PVL in full-term deliveries [8] while the tocolysis failed to prolong pregnancy. It may be caused by the tocolytics administered in the stage of intense preterm uterine contraction, where the tocolysis was ineffective to fully suppress contraction. It will be needed to tocolyze in early stage of preterm labor detected by early contraction detection with continuous tocodynamometry, the observation of round uterine anterior wall deformation, which is protruded into bladder image with vaginal scan real-time B-mode [9] or the detection of shortened soft cervix with shear wave ultrasound in vaginal scan [10], by which the tocolysis may be effective to prolong pregnancy until full term birth.

It is also recommended to ultrasonically detect neonatal brain PVE immediately after birth, and to treat it before changing to PVL, applying growth factor, because of its possible fetal brain reparing effect, and it was rich in the fetus in the last trimester, while disappears within 3-4 days after birth, which might be maternal origin and may be effective to repair fetal brain abnormality. The results have to be confirmed by tremendous studies on the uterine contractions and on the fetus and neonates of preterm birth.

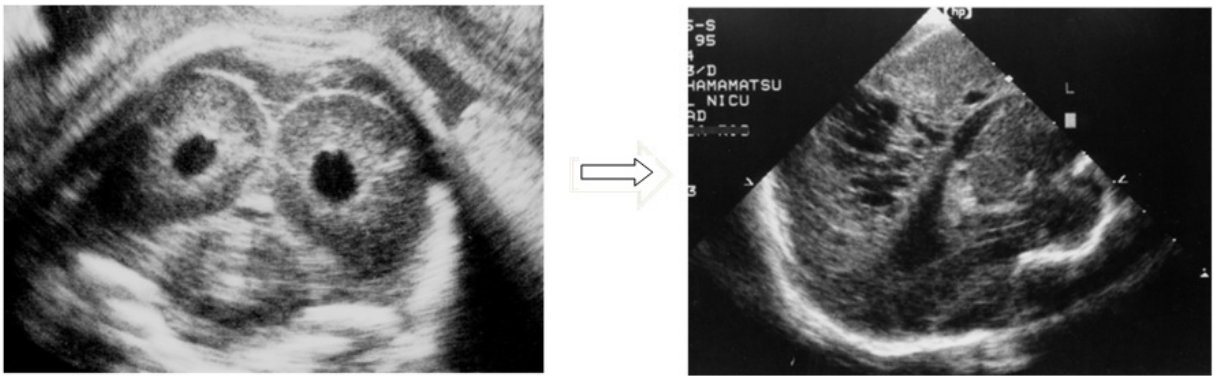

Figure 2 Fetal PVE in a preterm labor (left) changed to neonatal PVL (right) [7]. Courtesy of Yamamoto et al. [8].

In addition, congenital anomaly and fetal brain damage in infectious diseases (TORCHS) would be further discussed elsewhere.

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