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Cerebral Palsy caused by Intrapartum Damage is Prevented with Hypoxia Index

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Abstract

Aims: To prevent the cerebral palsy caused by intrapartum fetal brain damage. Methods: The late deceleration (LD) was controversy. The neonate of 3 connected LD was vigorous, but the neonate of 50 minutes repeated LD was severe asphyxia, loss of variability and expired by brain hemorrhage, i.e. repeated decelerations where summarized hypoxic effects damaged fetal brain, instead of the LD pattern, thus, the sum of deceleration durations (min)divided by the lowest FHR (bpm) and multiplied by 100 was Hypoxia Index. The FHR was used instead of PaO_2, because heart rate was parallel to PaO_2 when PaO_2 was lower than 50 mmHg and fetal PaO_2 was lower than 50mmHg. Results: Hypoxia index (HI) of 6 cases of cerebral palsy were 25 or more and the HI of cases of no cerebral palsy were 24 or less. There was significant difference between cases of 25 or more and 24 or less cases in the development of cerebral palsy. Discussion: As cerebral palsy developed when HI was 25 or more, and cerebral palsy did not develop when HI was 24 or less, the threshold to develop cerebral palsy exists between 24 and 25 of HI. Conclusion: Cerebral palsy caused by intrapartum brain damage will be prevented, if the hypoxia index is 24 or less. Fetal monitoring is recommended to calculate hypoxia index, and to perform early delivery when the hypoxia index is less than 24, to prevent cerebral palsy.

Keywords: Fetus heart rate, Deceleration, Bradycardia, Variability, Hypoxia Index, FHR score, Cerebral palsy, Early delivery.

Introduction

The cerebral palsy developed in 5 situations, including congenital fetal disease, intrapartum fetal damage, preterm delivery, fetal infectious diseases and postpartum event. Intrapartum fetal damage is the target of the present study, namely, the late deceleration (transient FHR bradycardia), which was characterized by the delay of its development to uterine contraction, and it was reported to be ominous in fetal outcome [1]. However, a neonate born after 3 connected fetal late decelerations (LDs) was vigorous, Apgar score was 9 (normal) without asphyxia and no resuscitation. It was contradictory to the reports on fetal outcome. In another case, however, 50 minutes' repetition of LDs resulted Apgar score 3 in severe asphyxia and the loss of FHR variability, which was the sign of severe fetal brain damage [2], followed by infantile demise from brain hemorrhage. The LD is defined when the deceleration pattern repeated 15 or more minutes in another LD definition. Thus, it was suspected that fetal outcome will be decided by the repetition of decelerations but not by the deceleration pattern, namely, a deceleration (bradycardia) means transient hypoxia, by which develops transient excitation of parasympathetic center and transient bradycardia. It will mean that a deceleration is transient hypoxia, and repeated deceleration means repeated hypoxia, not only in LD but in all decelerations including

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early, late, variable decelerations. Namely, hypoxic effect is weak to develop brain suppression or damage in 2-3 decelerations, while repeated decelerations for more than 5 times may suppress fetal brain, and highly repeated decelerations develop fetal brain damage.

Methods

Hypoxia index was calculated under the idea as above, and it is as follows; Hypoxia index = Sum of deceleration durations (min) divided by the lowest FHR(bpm) and multiplied by 100, "divided by the lowest FHR" means "multiplication with hypoxia amplitude", namely, the equation means the deceleration area (dip area). Namely, the HI is the summation of dip areas, which is hypoxic area. The100 multiplication is to keep the index integer. As the hypoxia index is the sum of all decelerations, it is necessary to calculate in every addition of new deceleration. Thus, a simple computerization will be convenient, where the HI value will be reported doctor in appearance of new deceleration. A new fetal monitoring was reported ethical committee of hospital, and it was performed getting informed consent of monitored pregnant woman.

Results

The relation of hypoxia index and cerebral palsy was studied in a case of LD in the department of Obstetrics and Gynecology, Tottori University hospital. Other cases were the deliveries in the department of Obstetrics and Gynecology, Seirei Mikatahara hospital. Maeda calculated the hypoxia index, six cases had cerebral palsy after delivery. FHR patterns were not diagnosed except for a severe late deceleration, which was discussed in the LD study. Hypoxia index were 25- 67, mean and SD were 36.7 ±1 6.48. Sixteen cases of FHR decelerations had no cerebral palsy, where the HI was 1-24, mean and SD were 15.52 ± 7.42. In Chi square test of 6 cases whose HI was 25 or more and 16 cases whose HI was 24 or less significantly differed to develop cerebral palsy, i.e. $p<0.05$ (Table 1).

Discussion

As you see, no cerebral palsy will develop due to intrapartum damage, if the hypoxia index is kept 24 or less at delivery.

Fetal deceleration will be treated by the change to lateral posture from supine, because late deceleration develops by the compression of maternal iliac artery with contracted uterus stopping placental circulation, and late decelerations disappeared by maternal lateral posture [3]. Other decelerations caused by supine hypotension and umbilical cord compression are also improved by maternal lateral posture. Other deceleration treatment will be maternal oxygen inhalation, and heparin solution of fibrin deposit in placental intervillous space, which is diagnosed by fetal growth restriction and high GLHW tissue characterization of placenta [4].

There are, however, further possibilities to develop cerebral palsy, thus, we have to be careful to preterm delivery and fetal infectious diseases (TORCHS) in Obstetrics, which will be discussed elsewhere. The treatment of genetic diseases would progress in the gene editing and chromosomal engineering, and the progress of postpartum event is expected in pediatrics.

Although fetal monitoring has been progressed, it should be more careful to the prevention of cerebral palsy as the author studied in the present report.

However, observation of FHR record on CTG or actocardiogram is rather limited in continuous fetal monitoring, thus, computerized fetal monitoring will be applied in fetal monitoring. It is the role of computer to detect risky change of FHR and automatically report it to the attending doctor, of which large computer for multiple simultaneous fetal monitoring was completed in TOITU MF-4000 system and utilized in Seirei Mikatahara hospital reporting significant decrease of perinatal mortality and zero cerebral palsy [5]. Thus, we are providing a simpler single birth monitoring computer, where the monitoring is done by FHR score to report expected Apgar score and UA pH, hypoxia index is effectively applied by fully continuous calculation, and frequency spectrum to report malignant sinusoidal pattern, which may cause fetal death, are incorporated. However, FHR pattern diagnosis is excluded, because the role of FHR pattern classification to diagnose fetal outcome is fully achieved by FHR score and hypoxia index, namely, FHR pattern is known simply by the observation of CTG record.

Table 1: Chi square test of high and low hypoxia index cases.

Hypoxia index (HI)	Develop cerebral palsy (p)	
	Yes	No
25 or more	6	0
24or less	0	16
P=0.000008 < 0.05, significant difference between both HI groups. Cerebral palsy: N=6, HI mean ± SD=36.7 ± 16.48 No cerebral palsy: N=16, HI mean ± SD=15.5 ± 7.42		

Conclusion

Cerebral palsy due to intrapartum fetal brain damage is prevented by early delivery when hypoxia index is close to 24 but not to be 25 or more.

References

1. Hon EH (1968) An Atlas of Fetal Heart Rate. Harty Press, New Haven.
2. Maeda K (2014) Modalities of fetal evaluation to detect fetal compromise prior to the development of significant neurological damage. J Obstet Gynaecol Res 40: 2089-2094.
3. Poseiro JJ, Mendex-Bauer SV, Caldeyro-Barcia R (1969) Effect of uterine contractions on maternal blood flow through the placenta. PAHO Advisary Committee on Medical Research.
4. Maeda K, Utsu M, Kihaile PE (1998) Quantification of sonographic echogenicity with gray-level histogram width: A clinical tissue characterization. Ultrasound Med Biol 24: 225-234.
5. Maeda K, Noguchi Y, Nagasawa T, Utsu M, Matsumoto F (2012) Central computerized automatic fetal heart rate diagnosis with a rapid and direct alarm system. The Open Medical Devices Journal 4: 28-33.

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