

Commentary on “Fetal Movement in Actocardiogram and Prevention of Cerebral Palsy with Hypoxia Index”

Kazuo Maeda*

Department of Obstetrics and Gynaecology, Tottori University Medical School, Japan

DESCRIPTION

Fetal movement and heart rate acceleration which is termed to be transient FHR rise developed at fetal movement group (movement burst) recorded in actocardiogram [1]. The Adult heart rate rises at leg motions. Hypoxia, which reduced PaO₂, stimulates vagal nerve centre in medulla oblongata, Hypoxia in anencephalic neonatal bradycardia, which recovered by oxygenated blood infusion [2]. Anesthesia of vagal nerve center did not develop bradycardia at hypoxia. The Excitation of vagal nerve developed fetal bradycardia which is commonly known to everyone.

Rabbit heart rate decreased parallel to the reduction of PaO₂ when PaO₂ is less than 50 mmHg [3]. Umbilical cord arterial blood PaO₂ is less than 50mmHg, which is hypoxic [4]. Three typical late FHR decelerations were not ominous [5]. While, repeated decelerations in 50 min, which were repeated hypoxia, were ominous due to overlapped hypoxia in repeated FHR decelerations. Late decelerations were ominous if the decelerations repeated 15 or more times (repeated hypoxia) in experts definition of late deceleration [6].

Hypoxic effect appears after repeated deceleration for 15 or more times, but not by 3 late decelerations. Thus, Hypoxia index (threshold to develop cerebral palsy)=The sum of all deceleration durations (min) in labor, divided by the lowest FHR, multiplied by 100 keeping index as integer, which was 25 or more in cerebral palsy diagnosed in Pediatrics, while no cerebral palsy hypoxia index was 24 or less in FHR records in the labor kept in Obstetrics of no outcome data [7], as Chi square $p=0.000008$, cerebral palsy is less than 24, to prevent cerebral palsy caused in the labor.

Continues minor fetal movements of fetal resting state developed FHR variability, which disappeared in severe fetal hypoxic brain damage similar to the loss of variability in anencephalic fetus, thus, severe hypoxic fetal brain damage was shown by the loss of variability where it is irreversible sign of cerebral palsy [8]. As numeric criteria to detect fetal brain damage in the labor preceding cerebral palsy is hypoxia index of 25 or more, it is the most easily diagnosed even by new comer, it was applied to prevent cerebral palsy to keep hypoxia index at 24 or less level, which is calculated by a computer, where the time to be 24 of index will be also indicated by a computer [9], and the pregnant

woman who showed positive hypoxia index is recommended to change to lateral posture to prevent deceleration and increase hypoxia index [10].

OBSERVATIONS

We collected 22 cases of infantile motions diagnosed by pediatric clinic to exclude false diagnosis of cerebral palsy, where cerebral palsy were 6 cases and no cerebral palsy 16 cases, whose intrapartum FHR records were preserved in obstetric wards and hypoxia indices were calculated by Maeda without knowledge of outcomes after births. All 6 cerebral palsy cases hypoxia index were 25 or more but no case was 24 or less, while all 16 no cerebral palsy cases hypoxia index was 24 or less in all cases but no case was 25 or more. In Chi square test $p=0.000008$, significant difference and there was no diagnostic error, as p is almost zero (Table 1).

Table 1: Chi square test of hypoxia index values.

Hypoxia Index	Cerebral palsy	
	Yes	No
25 or more	6	0
24 or less	0	16
Total	6	16

CONCLUSION

Thus, cases of hypoxia index of 24 or less is not cerebral palsy according to statistical analysis. It is cerebral palsy when hypoxic index is 25 or more, who can receive early treatment of cerebral palsy even in new-born age, namely, the effect of oxygen inhalation anti-glutamate drug, free radical scavenger or other drugs are effective in early treatments.

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Correspondence to: Kazuo Maeda, Department of Obstetrics and Gynaecology, Tottori University Medical School, Yonago, Japan, Tel: +81859226856; Email: maedak@mocha.ocn.ne.jp

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