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# **Opinion**

# The hypoxia index covers roles of all decelerations and will reduce cerebral palsy

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It was strange that fetal outcome was vigorous in 3 late decelerations, but ominous when it repeated. It was thought that FHR decelerations expresses hypoxia, because the heart rate was fully parallel to rabbit PaO2, if it is below 50mmHg, and human fetal PaO2 is 50mmHg or less. Thus, a hypoxia index (HI), that is hypoxic nature, was sum of deceleration duration (min) divided by the lowest nadir FHR (bpm), and multiplied by 100. Its calculation is easy by computer, though possible by manual calculation. HI has regression equation with Apgar score, and Apgar score was high in 2 to 3 late decelerations (LD), while HI was high and Apgar score was low in repeared LDs, solving the LD discrepancy. Although no particular role is expected by LD pattern, hypoxic effect is estimated in repeated large decelerations and deep bradycardia, namely, numerical analysis is possible in the deceleration and continuous bradycardia. More important role of HI is the prediction of fetal brain damage followed by cerebral palsy, i.e. HI was26,

followed by the loss of FHR variability like as anencephalic fetus, very low Apgar score and very severe infantile brain damage. Also in another severe case, who received caesarean delivery, intrapartum HI was 25 and followed by cerebral palsy. However, cases who had FR changes but preserved FHR variability, and followed by no cerebral palsy, the HI were 20 to 24, just below 25.

Thus, it was estimated that the threshold (the lowest level) of HI, which preceded the loss of variability, fetal brain damage and cerebral palsy, was 25. The fact means, the fetus of severe FHR changes must deliver before the loss of variability, where the HI is 24 or less. Therefore, we must study HI in fetal monitoring, and the fetus must deliver before the loss of variability, where the HI is 24 or less [1].

## References

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