## Acid accumulation during end-stage bradycardia in term fetuses: how long is too long?

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## **Abstract**

OBJECTIVE: To estimate the existence and degree of fetal accumulation of acid during endstage bradycardia as reflected by the base deficit. This may set a criterion for proper intervention during labour.

SETTING: Maternity unit of the Tel Aviv Medical Centre.

SUBJECTS: Forty-three consecutively born term infants whose mothers were delivered by vacuum extraction were analysed: 27 because of end-stage bradycardia and 16 controls whose mothers were delivered electively because of maternal indications.

MAIN OUTCOME MEASURES: Analysis of umbilical arterial cord blood for pH, PCO2 and base deficit. The base deficit was compared between the groups using the two-tailed Student's t test, and was correlated with variables of fetal heart rate monitoring using the Pearson correlation coefficient.

RESULTS: The base deficit was greater in newborns who had end-stage bradycardia than in controls (11.02 vs 5.01, P < 0.0001). The duration of loss of short term variability in fetal heart rate during end-stage bradycardia correlated positively with the base deficit (r = 0.8, P < 0.0005). Conversely, the time until the loss of short term variability during end-stage bradycardia correlated negatively with the base deficit. The length and the depth of the bradycardia and their product, had a weaker correlation with the base deficit.

CONCLUSIONS: End-stage bradycardia, which presumably reflects fetal hypoxia, is associated with acidaemia in the umbilical artery at birth in some fetuses. The fetuses who are predisposed to acidaemia, as reflected by an increased base deficit, are those who lost their fetal heart rate variability during end-stage bradycardia for more than 4 min or started to lose this in less than 3 min from the beginning of the end-stage bradycardia. Operative vaginal delivery should be reserved for these indications.