

Computerised Analysis of Foetal Heart Rate Variation: Prediction of Adverse Perinatal Outcome in Patients Undergoing Prostaglandin Induction of Labour at Term

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Abstract

The aim of this study was to determine the value of antenatal numerical assessment of foetal heart rate variation in the prediction of adverse perinatal outcome in patients undergoing prostaglandin induction of labour at term. Two hundred and seven patients who underwent prostaglandin cervical ripening after 37 weeks gestation for the indications of pregnancy-induced hypertension, foetal growth retardation or post-dates pregnancy were included in this study. Prior to commencement of cervical ripening, a 30-minute cardiotocography tracing was recorded on the System 8000 machine and the long-term and short-term variations were calculated. Forty-three patients (20.8%) had a long-term variation of less than 30 ms; 9 (4.3%) had a short-term variation of less than 3 ms. The sensitivity and positive predictive values of long-term variation in the prediction of admission to neonatal special care unit were 25.6% and 23.2%, respectively. Corresponding values for short-term variation were 2.6% and 11.1%, respectively. The sensitivity and positive predictive values of long-term variation in the prediction of caesarean section for foetal distress were 33.3% and 9.3%, respectively. Corresponding values for short-term variation were 8.3% and 11.1%, respectively. Long-term and short-term variations appeared to be both poor predictors of adverse perinatal outcome. However, of 4 foetuses with both reduced antenatal heart rate variation and who were subsequently delivered by caesarean section for foetal distress in labour, all 4 were admitted to neonatal special care unit (NSCU). Foetuses with intrapartum evidence of foetal distress were more likely to be admitted to NSCU when antenatal foetal heart rate variation was reduced.

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Introduction

The induction of labour in term pregnancies complicated by pre-eclampsia, foetal growth retardation and post-dates usually requires prostaglandin cervical priming when the cervical score is unfavourable.^{1,2} The antenatal monitoring of patients with these obstetric complications is important in view of the associated increase in perinatal morbidity and mortality.^{3,4} Whilst a variety of non-invasive tests of foetal well-being have been used to predict subsequent foetal compromise, non-stress cardiotocography (CTG) remains one of the most important antenatal tools used to monitor these pregnancies.⁵

Computerised CTG analysis has been used recently to describe the foetal heart tracing, in particular foetal heart rate variation. The major disadvantage with visual analysis of reduced foetal heart variation is that it is

inaccurate,^{6,7} a problem which may be improved by computerised analysis.⁸ Other potential advantages with computerised CTG analysis include better quality records, better use of time spent recording and adjustment for changing foetal behavioural states. Many studies have reported that pre-labour numerical CTG assessment predicts adverse perinatal outcome.^{4,9-15}

The aim of this study was to determine the value of antenatal numerical assessment of foetal heart rate variation in the prediction of adverse perinatal outcome in patients undergoing prostaglandin induction of labour at term.

Materials and Methods

Two hundred and seven patients who underwent prostaglandin cervical ripening after 37 weeks gestation for the indications of pregnancy-induced hypertension

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(PIH) (blood pressure of 140/90 or greater after a period of rest of at least 20 minutes, with or without proteinuria), foetal growth retardation (foetal abdominal circumference less than the third centile for gestational age or amniotic fluid index less than 4.0) or post-dates pregnancy (gestational age greater than 280 days) between October 1994 and March 1995 were included in this prospective study. Prior to commencement of cervical ripening, a 30-minute CTG tracing was recorded on the System 8000 machine (Oxford Sonicaid, Oxford, United Kingdom) and the entire 30-minute strip was used for subsequent analysis. The characteristics of the foetal heart were analysed by the instrument computer. This foetal monitor was designed to measure the foetal pulse interval, a measurement controlled by error algorithms. Valid pulse intervals were averaged over an epoch of 1/16 minute (3.75 seconds) and the baseline fitted. Two measures of foetal heart rate variation were evaluated in this study. Long-term variation was expressed as the mean one-minute range of pulse intervals. A long-term variation of more than 30 ms was defined as normal.^{11,14} Short-term variation was calculated as the mean of successive epochal (1/16 minute) pulse interval differences. A short-term variation of less than 3 ms was regarded as abnormal.^{9,13} Decelerations were not included in calculating the foetal heart rate variation. The clinicians were blinded to the results of the computerised CTG analysis, and clinical management was based on visual interpretation of the CTG strip.

After completion of 30 minutes of CTG tracing, a 3 mg prostaglandin pessary (PGE₂, Upjohn) was inserted into the posterior fornix for cervical ripening. Foetal heart rate monitoring was continued for a further 2 hours after prostaglandin insertion. If the cervical score continued to be unfavourable, a prostaglandin pessary would be inserted daily until an amniotomy could be performed. During labour, continuous electronic foetal heart rate monitoring was instituted. The last 59 consecutive patients recruited into the study had umbilical artery and vein pH measurements performed at delivery. Data on delivery and neonatal outcome included caesarean section rate for foetal distress in labour, umbilical artery and umbilical vein pH at delivery, Apgar score at 5 minutes and admission to the neonatal special care unit (NSCU). Foetal distress in labour was defined as an ominous CTG trace which warranted immediate operative delivery.

Regression analysis was used to assess the relationship between foetal heart rate variation and umbilical arterial and venous pH at delivery. Significance was assumed if $P < 0.05$. The sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) of reduced foetal heart rate variation in the prediction of the various adverse outcome criteria were also calculated.

Results

The demographic and delivery details of the 207 patients who were included in the study are shown in Table I. Of these, 96 (46.4%), 64 (30.9%) and 47 (22.7%) had prostaglandin cervical ripening because of PIH, foetal growth retardation and post-dates, respectively. The mean long-term and short-term variations prior to prostaglandin insertion were 41.0 ms (SD 13.3 ms) and 7.0 ms (SD 2.1 ms), respectively. Forty-three patients (20.8%) had a long-term variation of less than 30 ms; 9 (4.3%) had a short-term variation of less than 3 ms. The median interval between prostaglandin insertion and delivery was 23 hours (range 1.5 to 100 hours). The median duration of labour was 3.5 hours (1 to 23 hours). Of the 207 patients, 59 (28.5%) had umbilical artery and vein pH values assessed at delivery. Twelve neonates were delivered by caesarean section because of CTG evidence of foetal distress during labour. Three neonates had an Apgar at 5 minutes of less than 7. Thirty-nine neonates were admitted to the NSCU. There was no significant relationship between long-term variation and umbilical artery pH (variation = 151 to 15.2pH, $R^2 = 0.01$, NS) or venous pH (variation = 249 to 28.4pH, $R^2 = 0.02$, NS) at

TABLE I: DEMOGRAPHIC DETAILS

Demographic and Delivery Details		
Mean age (year) (SD)		27.7 (4.9)
Parity		
	0	94
	1	57
	2	32
	3	8
Mean gestation at delivery (days) (SD)		279 (7.9)
No. of prostaglandin pessaries		
	1	148
	2	42
	3	14
Indication	Pre-eclampsia	47
	Foetal growth retardation	64
	Post-dates	96
Mode of delivery		
	Normal vaginal delivery	141
	Forceps/Ventouse	32
	Caesarean section	32
Indication for caesarean section		
	Foetal distress	12
	Fetopelvic disproportion	17
	Failed induction	2
	Others	1
Mean birthweight (g) (SD)		3 195 (408)
Mean arterial pH (SD)		7.2 1 (0.08)
Mean arterial base excess (SD)		-4.7 (2.7)
Mean venous pH (SD)		7.30 (0.07)
Mean venous base excess (SD)		-3.8 (2.3)

delivery. The sensitivity, specificity, PPV and NPV of the measures of foetal heart rate variation in the prediction of adverse pregnancy outcome are shown in Table II. It is evident that long-term and short-term variations were both poor predictors of adverse perinatal outcome. Of the 3 neonates who had a low Apgar score at 5 minutes, none had reduced long-term variation or short-term variation. Reduction in the long-term variation predicted 33.3% and 25.6% of patients requiring caesarean section for foetal distress and admission to NSCU, respectively; corresponding values for short-term variation were 8.3% and 2.6%, respectively. However, it is noteworthy that the 4 fetuses with both reduced antenatal heart rate variation and who were subsequently delivered by caesarean section for foetal distress in labour were admitted to NSCU.

Discussion

Recent studies have reported data to suggest that computerised assessment of foetal heart variation in pregnancies complicated by PIH,¹³ foetal growth retardation^{11,12,15} and post-dates⁴ predict adverse perinatal outcome. This study is unique in that the usefulness of antenatal numerical assessment of foetal heart rate variation was assessed in patients who subsequently underwent prostaglandin cervical ripening at term for the above obstetric indications. The results of our study suggest that numerical analysis of foetal heart variation appeared to be a poor predictor of adverse perinatal outcome in term pregnancies with the above obstetric complications. Nevertheless, the results also suggest that normal foetal heart rate variation was highly predictive of a good perinatal outcome.

The patients included in this study were strictly confined to those who underwent prostaglandin cervical

ripening at term for specific obstetric complications of PIH, foetal growth retardation and post-dates. The results of the numerical CTG analysis were blinded to the referring obstetricians and subsequent management was therefore based on visual interpretation of the CTG. In this study, long-term variation was used to quantify foetal heart rate variation as it has been reported to be predictive of acidosis at cordocentesis¹¹ and at delivery.¹⁴ Recent studies on numerical analysis of the CTG have also reported data using the short-term variation^{9,13} as this has been suggested to quantify foetal heart variation more accurately in the presence of low frequency sinusoidal rhythms. As there was no perinatal mortality in the patients we studied, adverse outcome measures of acidaemia at birth, low Apgar score at 5 minutes, caesarean section for foetal distress and admission to NSCU were used to define morbidity. However, it has to be acknowledged that these outcome measures, in particular admission to NSCU, are also likely to be affected by intrapartum events which are unrelated to the pathology of foetal hypoxia and acidosis (such as prolonged labour, use of drugs in labour and instrumental applications).

In our study, antenatal evidence of reduced long-term variation detected 33.3% and 25.6% of patients who had caesarean section for foetal distress in labour and admissions to NSCU, respectively. The poor performance of long-term variation in the prediction of adverse outcome may be due to several factors. Firstly, the population we studied comprised of a heterogeneous group of patients with differing obstetric complications. Nonetheless, the underlying pathophysiology of hypoxia and/or acidosis is believed to be common to these obstetric complications. Secondly, this study was confined to patients who were more than 37 weeks gestation at the time of antenatal assessment; consequently perinatal morbidity in the group as a whole was low. The limitations of numerical analysis of foetal heart variation in the prediction of morbidity in high-risk pregnancies have also been noted by Soothill and colleagues.¹⁶ In that study of 191 high-risk pregnancies, reduced foetal heart variation detected 5.1% of patients with perinatal morbidity with a positive predictive value of 28.6%. The perinatal morbidity reported by Soothill and colleagues¹⁶ (caesarean section rate of 15.2%, low Apgar score at 5 minutes of 3.1% and admission rate to special care unit of 7.8%) were fairly similar to those in the present study. In addition, there were no perinatal deaths reported in their study.

The results of our study and those of Soothill and colleagues¹⁶ contrast with others which report a significant relationship between reduced long-term variation and hypoxia and/or acidosis at delivery. In a study which compared antenatal foetal heart rate variation and umbilical artery pO₂ and pH at elective caesarean

TABLE II: PREDICTION OF ADVERSE PERINATAL OUTCOME

Test	Outcome	Se	SP	PPV	NPV
LTV <30 ms	Apgar 5 min <7	0/3 (0%)	161/204 (78.9%)	0/43 (0%)	161/164 (98.1%)
STV <3 ms		0/3 (0%)	195/204 (95.6%)	0/9 (0%)	195/198 (98.5%)
LTV <30 ms	NSCU	10/39 (25.6%)	135/168 (80.3%)	10/43 (23.2%)	135/164 (82.3%)
STV <3 ms		1/39 (2.6%)	160/168 (95.2%)	1/9 (11.1%)	160/198 (80.8%)
LTV <30 ms	CSFD	4/12 (33.3%)	155/195 (79.5%)	4/43 (9.3%)	155/164 (94.5%)
STV <3 ms		1/12 (8.3%)	186/195 (95.4%)	1/9 (11.1%)	186/198 (94.4%)

Se: sensitivity; Sp: specificity; PPV: positive predictive value; NPV: negative predictive value; LTV: long-term variation; STV: short-term variation; NSCU: neonatal special care unit; CSFD: caesarean section for foetal distress

section in 71 patients, fetuses with reduced foetal heart rate variation had lower pO_2 and pH values than those at a corresponding gestational age but with an optimal trace.¹⁴ In a retrospective study of 2582 patients, Street et al¹³ reviewed the perinatal outcome in 78 pregnancies with a long-term variation of less than 20 ms. Most of these pregnancies had proteinuric hypertension (73%) or foetal growth retardation (19%). The perinatal mortality rate in that study was 11.5% and all deaths occurred before 32 weeks of gestation. Of the 78 fetuses in that study, 44 also had a short-term variation less than 3 ms. Four of these fetuses died *in utero* and all 4 had short-term variation less than 2.6 ms. Of the remaining 40 fetuses, umbilical artery pH was reduced and base excess increased when the short-term variation was less than 2.6 ms, suggesting that the fetuses were becoming acidaemic. In a study of 89 patients with a short-term variation less than 3 ms, Dawes et al⁹ also reported a significant relationship between reduced short-term variation and metabolic acidaemia and intra-uterine death. The perinatal mortality rate in that study was 9.0%. In each of these 3 studies, gestational ages at delivery were considerably earlier and perinatal mortality rates higher than those in the present study, which may explain the poor predictive ability of foetal heart rate variation in the antenatal population we studied.

The usefulness of foetal heart rate variation in the prediction of adverse perinatal outcome has also been compared with other tools of antenatal surveillance. In a recent study of 337 post-term patients, reduced foetal heart rate variation was found to be superior to umbilical artery Doppler examination and biophysical profile in the prediction of adverse perinatal outcome.⁴ However, other workers have reported data which suggest that reduced foetal heart rate variation is a late sign of foetal impairment which coincides with the occurrence of late decelerations.^{12,15} In a study of 13 fetuses with absent end-diastolic flow in the umbilical artery Doppler waveform, reduced foetal heart rate variation was preceded by a loss of the protective "cerebral sparing" mechanism in response to foetal hypoxia and/or acidosis.¹⁷ The exact timing of the onset of reduced foetal heart rate variation in response to foetal hypoxia is still unknown and further studies are needed to address this issue.

An important finding in this study was the high specificity of foetal heart rate variation in the prediction of adverse perinatal outcome. This is an important aspect of antenatal testing as a clinician should feel reassured by a negative test result. Furthermore, there were four fetuses in our study that had both reduced long-term variation and who developed an abnormal CTG during labour requiring caesarean section for foetal distress. All four were admitted to the NSCU. It is possible that fetuses with intrapartum evidence of

foetal distress are more likely to be admitted to the NSCU when antenatal foetal heart rate variation is reduced.

The results of this study suggest a limited role for antenatal numerical CTG analysis of fetuses at term in the prediction of adverse perinatal outcome. Further studies are needed to evaluate its usefulness in other groups of patients who present earlier in pregnancy or in whom perinatal loss rate is likely to be higher.

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