

Different Types of Variable Decelerations and Their Effects to Neonatal Outcome

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ABSTRACT

Objective: Although the only objective finding of intrapartum fetal distress is obtained through the measurement the fetal scalp pH, this invasive procedure is not available in every institution. The careful examination of fetal heart rate tracings for abnormalities, especially of the most commonly seen one, variable decelerations gains great importance under these circumstances. The aim of the present study is to determine the prognostic significance of variable decelerations in intrapartum fetal heart rate monitoring.

Methods: A total of 96 fetal heart rate tracings were analysed to assess the prognostic significance of variable decelerations. Sixty-six percent (64/96) of cases exhibited atypia characterised with (1) slow return of the fetal heart rate to the baseline; (2) loss of variability during the decelerations; (3) loss of initial and/or secondary accelerations; (4) persistence of secondary acceleration (overshoot); and (5) continuation of the baseline fetal heart rate at a lower level; (6) biphasic deceleration. One and five-minute Apgar scores and umbilical artery pH were used to assess the final fetal condition.

Results: Adverse fetal outcome characterised by fetal acidosis and Apgar score lower than 7 at one and five minutes were uncommon with pure variable decelerations. Typical and atypical variable decelerations were associated with low Apgar scores (<7) at one minute in 9.3% and 54.6% of cases ($p<0.001$) and at five minutes in 6.25% and 25% of cases ($p<0.05$), respectively. In addition umbilical artery pH found to be lower than 7.2 in these cases (18.75% – $p<0.05$). There was no danger for the fetal haemodynamic conditions when typical uterus contraction/variable deceleration ratios were two or more than two. However, risk of fetal hypoxia damage was quite high when this ratio was lower than two in atypical variable decelerations. This result is proven by the 1st and

5th minute low Apgar scores and pH (81.8% and 36.6% respectively). Atypical features are helpful in the identification of distress characterised by low Apgar scores in fetuses with variable decelerations. Admission to the neonatal intensive care unit was more common in patients with atypical variable decelerations in comparison with typical variable decelerations (34.3% versus 3.1%).

Conclusion: While typical variable decelerations are frequently harmless, atypical variations pose a significant risk of fetal hypoxia.

Keywords: variable deceleration, neonatal outcome

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INTRODUCTION

Although the only objective finding of intrapartum fetal distress is obtained through the measurement of the fetal scalp pH, this invasive sampling is not available in every institution. The careful examination of fetal heart rate tracings for abnormalities, especially of the most commonly seen one, variable decelerations gain great importance under these circumstances.

Changes in fetal heart rate may be related to fetal hypoxia. However, the effects of electronic fetal monitoring on the health outcome depend not only on this relation but also on the sensitivity and specificity of electronic fetal monitoring, the prevalence of hypoxia and the availability of effective therapeutic interventions. Evidence on the link between fetal hypoxia and changes in fetal heart rate alone does not provide clinicians with a useful guide to the appropriate use of electronic fetal monitoring. Transient compression of the fetal umbilical cord causes an abrupt reduction in the fetal heart rate, probably mediated by afferent carotid baroreceptors and efferent vagal impulses and is manifested as a “variable deceleration” during monitoring⁽¹⁾. Intrapartum variable decelerations have been associated with fetal acidosis related to umbilical vein compression⁽²⁾. In an animal study Clapp et al⁽³⁾ suggested that intermittent cord occlusion may result in hypoperfusion of developing

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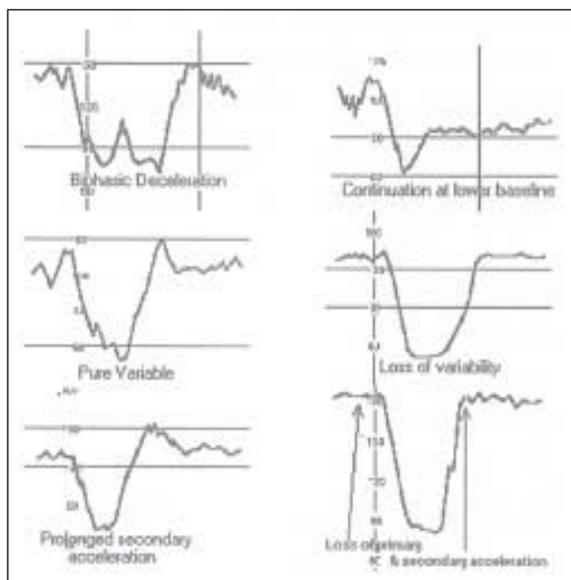


Fig. 1 The examples of typical and atypical variable decelerations.

periventricular white matter, with subsequent necrosis and cyst formation. Associations have been demonstrated between intrapartum variable decelerations and fetal distress⁽⁴⁻⁷⁾.

In the present study we investigated the prognostic significance of variable decelerations during intrapartum fetal heart rate monitoring.

MATERIALS AND METHODS

The study population comprised 96 singletons, nonanomalous pregnancies at or beyond the 34th gestational week and undergoing intrapartum non-stress test and subsequent delivery at Ege University Hospital. The patients with oligohydramnios and demonstrating meconium staining during the active phase of labour were excluded from the study. The patient files were examined (n=4040) retrospectively and the ones with variable decelerations were included in the study retrospectively. The first 30 minutes of fetal heart rate tracings obtained from patients immediately after admission to the labour and delivery unit and the last 30 minutes of fetal heart rate tracings prior to delivery were analysed retrospectively.

Variable decelerations vary markedly in pattern from contraction to contraction and do not reflect the conformation of the uterine contraction curve. The onset of the deceleration bears a variable time relationship to the beginning of the associated uterine contraction⁽¹⁾. Variable decelerations were further classified as mild, moderate and severe:

- a. Mild variable decelerations: These are of less than 30 seconds' duration irrespective of depth, or deceleration not below 80 bpm (beats per minute) regardless of duration, or decelerations 70 to 80 bpm lasting less than 60 seconds.

- b. Moderate variable decelerations: These decelerations are less than 70 bpm lasting 30 to 60 seconds, or deceleration to 70 to 80 bpm lasting more than 60 seconds.
- c. Severe variable decelerations: These decelerations are below 70 bpm lasting more than 60 seconds.

Pure variable decelerations are typical variable decelerations without signs of atypia. They consist of an initial acceleration, rapid deceleration of the fetal heart rate to the nadir, followed by rapid return to the baseline fetal heart rate level with secondary acceleration.

Atypical variable decelerations as described by Krebs et al⁽¹⁾ were recorded. Atypical variable decelerations are prognostically unfavourable variable decelerations with features indicative of fetal hypoxia including:

- a. Slow return of the fetal heart rate to the baseline.
- b. Loss of variability during the deceleration.
- c. Loss of primary and/or secondary acceleration.
- d. Persistence of secondary acceleration (overshoot).
- e. Continuation of the baseline fetal heart rate at a lower level.
- f. Biphasic deceleration.

The examples of typical and atypical variable decelerations are shown in Fig. 1. One- and five-minute Apgar scores and umbilical artery pH were used to assess final fetal condition. The pH values below 7.2 were accepted as fetal acidemia. Statistical analysis was carried out by chi-square test. The data were expressed as numbers and percentages.

RESULTS

The grading of variable decelerations according to amplitude and duration was found to be related to the prognosis as shown in Table I. Apgar score and umbilical artery pH were observed to be significantly lower in cases with severe variable decelerations ($p < 0.001$). In Table II, variable decelerations were classified as typical and atypical with regard to the Apgar scores (lower than 7.0 vs. higher than or equal to 7.0) and neonatal acidosis (pH lower than 7.0). When atypical variable decelerations were excluded from the analysis within the group of cases with typical decelerations, amplitude and duration of decelerations did not have any significant effect on the Apgar scores or the presence of fetal acidosis. ($p > 0.05$). Typical and atypical variable decelerations were associated with low Apgar scores (< 7), at one minute in 9.3% and 54.6% of cases ($p < 0.001$) and at five minutes in 6.25% and 25% of cases ($p < 0.05$), respectively. In addition, umbilical artery pH was found to be lower than 7.2 in these cases (18.75% – $p < 0.05$). The frequency of atypical variable decelerations and their associations with outcome are demonstrated

Table I. The prognosis of variable decelerations, when graded according to amplitude and duration of decelerations.

	n	Apgar 1-min <7	%	Apgar 5-min <7	%	Neonatal acidosis pH <7.2	%
Mild	44	8	18.1 ^a	4	9 ^b	2	4.5
Moderate	42	20	47.6 ^a	10	23.8 ^b	4	9.5
Severe	10	10	100 ^a	4	40 ^b	4	40 ^b

^a p<0.001, ^b p<0.05**Table II. The prognosis of typical and atypical variable decelerations, when graded according to amplitude and duration of decelerations.**

	n	Apgar 1-min <7	%	Apgar 5-min <7	%	Neonatal acidosis pH <7.2	%
Typical Mild	22	2	9	1	4.5	0	0
Typical Moderate	10	1	10	1	10	1	10
Typical Severe	0	0	0	0	0	0	0
Atypical Mild	22	9	40	5	22.1	2	9
Atypical Moderate	32	16	50	9	28.1	6	18.75
Atypical Severe	10	10	100	4	40	4	40

Table III. The frequencies of atypical variable decelerations.

	n	Apgar 5-min <7	%	Neonatal acidosis pH <7.2	%
Loss of primary acceleration	32	6	18.7	2	6.25
Loss of secondary acceleration	42	14	33.3	6	14.2
Loss of variability	22	8	36.3	7	31.8
Continuation at lower baseline	18	6	33.3	6	33.3
Biphasic deceleration	16	6	37.5	6	37.5

Table IV. Relative frequency of variable decelerations per uterine contraction (uterine contraction rate/variable deceleration).

	Uterine contraction/ variable deceleration	n	Apgar 1-min <7	%	Apgar 5-min <7	%	Neonatal acidosis pH <7.2	%
Typical	>2 ^a	16	0	0	0	0	0	0
	<2 ^a	16	3	25	2	12.5	1	6.25
Atypical	>2 ^b	42	16	38	8	19	7	16.6
	<2 ^b	22	18	81.8	8	36.6	8	36.6

^a p<0.001, ^b p<0.05**Table V. Variable decelerations occurring concurrently with fetal loss of variability, tachycardia or bradycardia.**

	n	Apgar 1-min <7	%	Apgar 5-min <7	%	Neonatal acidosis pH <7.2	%
Loss of variability	17	11	51	8	47	5	29.4
Tachycardia (>180 bpm)	5	2	40	0	0	0	0
Bradycardia (<120 bpm)	4	4	100	2	50	2	50

bpm: beats per minute.

in Table III. There were at least two atypia criteria in 60% of fetal heart rate tracings. The most frequent type of atypical variable deceleration was loss of secondary acceleration (65.6%). Loss of variability and continuation at a lower baseline and in the presence of biphasic deceleration were associated with lower Apgar scores and fetal acidosis.

Table IV demonstrates the frequency of variable decelerations relative to the uterine contraction rate. When this ratio is equal to or higher than two, there are no abnormal haemodynamic effects on the fetuses, whereas with ratios lower than two and even in the presence of a single atypical deceleration, the probability of an unfavourable outcome (low 1st and 5th minute Apgar scores and fetal acidosis) is significantly higher. Table V shows the effect of the concurrence of variable decelerations with other fetal heart rate tracing abnormalities (fetal loss of variability, tachycardia or bradycardia) on the perinatal outcome.

The admission to a neonatal intensive care unit is more common in patients with atypical variable decelerations in contrast to patients with typical variable decelerations (34.3% vs. 3.1%).

DISCUSSION

An animal model of Westgate et al⁽⁸⁾ demonstrated that the fetal heart rate variations in response to repeated umbilical cord occlusion is more complex than expected. The initial response to acute asphyxia typically included an increase in fetal heart rate variation, while terminal fetal compromise with profound acidemia and hypotension were accompanied by an increase in fetal heart rate variation in some fetuses and a decrease in others. They suggested that a fetal heart rate overshoot-instability may be a useful marker of fetal decompensation following variable decelerations.

In the present study, adverse fetal outcome was characterised by low Apgar scores and fetal acidosis and was uncommon with pure variable decelerations. Atypical variable decelerations were associated with low Apgar scores (<7) at one minute in 54.6% of cases ($p<0.001$), and at five minutes in 25% of cases ($p<0.05$). In addition, umbilical artery pH was found to be lower than 7.2 in these cases (18.75% – $p<0.05$). In cases with ratios higher than or equal to two, low one-minute Apgar score rate (<7) was found to be 27.5% (16/58) and in cases with ratios lower than two, 57.9% (22/38) ($p<0.01$). Frequencies of variable decelerations relative to the uterine contraction rates higher than two are more probable to result in low one- and five-minute Apgar scores (<7) in contrast to those tracings with ratios higher than two (81.8% vs. 36.6%).

Judge et al⁽⁹⁾ segregated 693 patients at or beyond 30 weeks' gestation with reactive non-stress tests into two groups based on the absence or presence of variable decelerations of 15 seconds or more in duration and a drop of 20 or more beats per minute in severity. Fetuses with antepartum variable decelerations were more likely to demonstrate similar decelerations in labour ($p<0.001$), to undergo operative delivery for a diagnosis of distress ($p<0.05$), to require intensive care nursery admission ($p<0.01$) and to be small for gestational age ($p<0.01$). No significant differences were noted in frequency of nuchal or other cord entanglements, overall caesarian section rate or low pH or Apgar score values.

The association of antepartum variable decelerations and cord complications has been reported by Phelan et al⁽¹⁰⁾ (55.3%), Anyaegbunam et al⁽⁴⁾ (28%), O'Leary et al⁽¹¹⁾ (52.9%) and Meis et al⁽¹²⁾ (27%); however, Druzin et al⁽¹³⁾ (10.7%) and Pazos et al⁽¹⁴⁾ (0%) did not find an increased incidence of funic abnormalities.

Krebs et al⁽¹⁾ reported a high incidence of fetal acidosis and low Apgar scores with atypical variable decelerations. Adverse fetal outcome was uncommon with pure variable decelerations irrespective of the duration and amplitude of the deceleration. Both pure and atypical variable decelerations were associated with other fetal heart rate abnormalities in 60% of the cases. The unfavourable combination with decreased fetal heart rate variability and tachycardia or bradycardia was seen more frequently with atypical than with pure variable decelerations and predicted the highest incidence of low Apgar scores.

Transcervical amnioinfusion is the instillation of a physiologic solution through a transcervical intrauterine catheter to restore amniotic fluid volume. The beneficial effects of amnioinfusion during labour in resolution of repetitive variable decelerations have been demonstrated^(15,16). Maternal and neonatal outcome are not different with continuous or intermittent amnioinfusion protocols but the continuous infusion protocol is associated with an increased cost to the patient because of the use of greater volumes of infusate and use of intravenous pumps to control the rate of intrauterine infusion⁽¹⁷⁾.

In conclusion, atypical features are helpful in the prediction of distress characterised by low Apgar scores in fetuses with variable decelerations. The combination of high uterine contraction/variable deceleration ratios with atypical variable decelerations seems to be best at predicting low Apgar scores and fetal acidemia. Decelerations without atypia are generally harmless, yet when variable decelerations present with atypia and especially at high frequencies, they usually mark fetal hypoxia.

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