



Fetal heart rate changes and cerebral oxygenation measured by near-infrared spectroscopy during the first stage of labour

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Abstract

Objective: To determine the relationship between contraction related changes in fetal heart rate and cerebral oxygenation measured by near-infrared spectroscopy during labour. **Study design:** A specially designed optical probe was inserted through the dilated cervix and placed against the fetal head in 30 women during labour. Alterations in fetal heart rate during the final hour of the first stage of labour were compared with changes in the cerebral haemoglobin oxygenation index (Δ oxyhaemoglobin concentration – Δ deoxyhaemoglobin concentration) measured before, during and after uterine contractions. **Results:** Uterine contractions which were associated with either no alteration, accelerations or early decelerations of the fetal heart rate showed no significant changes in the haemoglobin oxygenation index. Variable, late and prolonged decelerations all showed significant decreases in the haemoglobin oxygenation index ($P < 0.01$) either during (variable) or after (variable, late and prolonged) the uterine contraction. **Conclusion:** The association between variable, late and prolonged FHR decelerations and significant falls in cerebral oxygenation during late labour suggests that these fetal heart rate patterns are associated with an increased risk of fetal cerebral hypoxia.

Keywords: Near-infrared spectroscopy; Fetal brain oxygenation; Fetal heart rate

1. Introduction

Continuous electronic fetal heart rate (FHR) monitoring during labour has achieved widespread acceptance for fetal surveillance since its introduction into clinical practice in the 1960s [1]. However, its use has not been shown to be associated with a clear reduction in perinatal hypoxic-ischaemic brain injury, and difficulties of interpretation have led to an increase in the frequency of operative deliveries [2]. Although alterations in baseline FHR are common during labour [3], little is known about the relationship between these changes and the oxygenation of the fetal brain.

Near-infrared spectroscopy (NIRS) allows several indices of fetal cerebral oxygenation and perfusion to be

observed during labour [4–7]. Preliminary studies using this technique suggested that early FHR decelerations may be associated with transient cerebral desaturation [4] and, more recently, we have shown that late FHR decelerations are associated with a significant transient decrease in cerebral oxygenation [8]. This study was designed to investigate the relationship between a range of contraction-related alterations in FHR that are commonly observed during the late first stage of labour and changes in cerebral oxygenation and haemodynamics.

2. Material and methods

Thirty healthy pregnant women (19 primigravidas, 11 multigravidas), aged between 19 and 39 years (median 27) with uncomplicated pregnancies (range 34–42, median 40 weeks' gestation) were recruited from the labour suite for our study. The results from 10 of the mothers with

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late FHR deceleration contractions have previously been published [8]. All were in uncomplicated labour, with a singleton cephalic presentation and had ruptured amniotic membranes. Nineteen labours were being augmented with intravenous oxytocin infusions (4–30 milliunits/min). Twenty women had effective epidural analgesia (0.25%–0.50% bupivacaine), 7 were using a mixture of 50% nitrous oxide and 50% oxygen (Entonox, British Oxygen Co., Guilford, Surrey, UK) or had received pethidine (50–150 mg by intramuscular injection) and 3 had no analgesia. In all cases, the fetal heart rate and uterine contractions were monitored continuously using cardiotocography (Hewlett Packard 2403G, Hewlett Packard, Böblingen, Germany.), with either internal or external transducers. The study was approved by the local committee on the ethics of human research and informed written consent was obtained beforehand from each woman.

A specially designed fetal optical probe was inserted through the dilated cervix (median 6 cm, range 3–9 cm) and applied against the side of the fetal head. The probe incorporated the ends (optodes) of two flexible fibreoptic bundles mounted at a fixed distance of 3.5 cm apart. Care was taken to avoid the face and ears and the probe was maintained in position by maternal tissue pressure and controlled continuous negative pressure (150 mmHg) provided by a modified wall suction apparatus. Fibre bundles conveyed near infrared light at four different wavelengths between 777 and 913 nm to and from a portable spectrophotometer (NIR0500, Hamamatsu Photonics KK, Hamamatsu City, Japan). Changes in attenuation at each wavelength of light traversing the brain were detected continuously and converted into changes in the cerebral concentrations of oxyhaemoglobin [HbO_2] and deoxyhaemoglobin [Hb] using a previously established algorithm [9–11]. Measurements were collected every second and averaged over each consecutive 10 s period and both the NIRS and cardiotocography data were recorded simultaneously onto a portable computer. The NIRS data were not made available to the clinicians responsible for labour management.

The probe was applied during a routine vaginal examination and all women remained in a comfortable semirecumbent position while observations were undertaken. Measurements were obtained from the time of probe application until after the onset of the second stage of labour. At delivery, blood was taken from the umbilical artery for acid-base analysis. The data collected during the final hour of the first stage of labour (determined in retrospect from the partogram), were analysed using commercial software (SPSS for Windows version 6, SPSS, Chicago, IL).

The tocography data for each contraction were analysed in order to define its time of onset and end. The

onset of the contraction was judged to occur after there had been a significant increase (two standard deviations) from the baseline and similar analysis was used to determine the end of the contraction. Preliminary analysis of the data had shown that the optimal periods for analysing the data were a 30 s period prior to the onset of the contraction to establish the baseline, the period between the onset and the end of the contraction, and a 50 s collection period after the end of the contraction to allow adequate assessment of the effects of any late changes in the fetal heart rate.

The mean changes from baseline in [HbO_2] and [Hb] were calculated before, during and after each uterine contraction. These were then converted into mean changes in the haemoglobin oxygenation index [Hb_{diff}] ($[\text{Hb}_{\text{diff}}] = [\text{HbO}_2] - [\text{Hb}]$) [12]. The mean changes in total cerebral haemoglobin concentration [Hb_{tot}] ($[\text{Hb}_{\text{tot}}] = [\text{HbO}_2] + [\text{Hb}]$) [13] and FHR were also determined for each of the three periods of the contraction.

All the 1 h cardiotocograph traces were reviewed by one author (JADS) who was unaware of the NIRS data. FHR changes during each contraction were placed into one of seven different groups, which were defined as follows [14]:

1. No change: no significant alteration in FHR from baseline or a change of less than 15 bpm.
2. Acceleration: a transient rise in FHR of at least 15 bpm lasting for at least 15 s.
3. Early deceleration: a uniform decrease in FHR of more than 15 bpm which was closely synchronous with the contraction.
4. Variable deceleration: a variable decrease in FHR of more than 15 bpm which showed no consistent relationship to the peak of the contraction.
5. Late deceleration: a uniform decrease in FHR of more than 15 bpm with the deceleration more than 15 s after the peak of the contraction.
6. Combined deceleration: a combination of an early or variable deceleration and a late deceleration in response to a single uterine contraction.
7. Prolonged deceleration: a FHR below 80 bpm for more than 2 min or below 100 bpm for more than 3 min.

For each patient the mean changes from baseline in [Hb_{diff}], [Hb_{tot}] and FHR were calculated for the different FHR changes. A mean value was then obtained for each FHR group, before, during and after the contraction. The 'no change' group of contractions was used as a control group and mean changes from baseline in [Hb_{diff}] and [Hb_{tot}] in this group were analysed using a paired Students *t*-test. Data from the other groups were then compared with the control group by analysis

Table 1
Summary of FHR changes with each uterine contraction

FHR change with contraction	Number of contractions (%)	Number analysed	Number of patients
Control (no change)	142 (26%)	75	23
Acceleration	115 (21%)	58	16
Early	23 (4%)	15	6
Late	46 (8%)	31	11
Combined	30 (5%)	23	4
Variable	186 (34%)	91	21
Prolonged	13 (2%)	7	6
Total	555 (100%)	300 (54% of the total)	30

of variance (ANOVA) using patient number as an additional variable to account for individual patient differences.

3. Results

There was no difficulty experienced in positioning the probe satisfactorily on the fetal scalp and none of the mothers found monitoring with NIRS uncomfortable. A total of 555 contractions were identified from the 30 cardiotocograph traces, of which 300 contractions were suitable for analysis (Table 1). Of the 255 contractions excluded from analysis, 48% had poor quality topographic data which prevented accurate timing of contractions, 24% had poor quality NIRS data, and in 28% uterine contractions were so frequent that a satisfactory baseline could not be established between each contraction.

Seven women had fetal blood samples performed because of FHR changes deemed to be ominous by the attending clinical staff. In all 7 cases, the pH was greater than 7.2. Fourteen of the 30 women had a spontaneous vaginal delivery and the remaining 16 had operative deliveries (7 ventouse, 7 forceps, 2 caesarean sections) per-

formed because of fetal distress (6 cases) or delay in the second stage (10 cases). The median birth weight was 3205 (range 1952–4160) g and the median umbilical artery pH was 7.28 (range 7.08–7.46). Two infants had an Apgar score of 6 or less at 1 min and each required intubation and intermittent positive pressure ventilation. At 5 min, all infants had Apgar scores of at least 9 and none required admission to the neonatal unit.

Mean changes from baseline for each of the different FHR groups of contractions are given in Table 2. Data for the combined FHR deceleration group are not included as they occurred in only 4 patients and were not analysed.

Representative data of changes in $[HbO_2]$ and $[Hb]$ from 3 fetuses demonstrating either no alteration, an acceleration and an early FHR deceleration are illustrated in Fig. 1. Within the 'no change' group of contractions, both $[HbO_2]$ and $[Hb]$ showed a significant decrease during the contraction with a return to the original baseline afterwards. $[Hb_{tot}]$ fell by a mean of 0.42 (S.D. = 0.50) $\mu\text{mol}/100\text{g}$ of brain tissue, ($P < 0.001$) while $[Hb_{diff}]$ showed no change 0.00 (S.D. = 0.40) $\mu\text{mol}/100\text{g}$ during the contraction. Similar changes in $[HbO_2]$ and $[Hb]$ were observed for the acceleration and early deceleration groups. There was no significant differences in mean $[Hb_{tot}]$ and $[Hb_{diff}]$ for all three groups (ANOVA).

The variable, late and prolonged deceleration groups all showed a different pattern, with a rise in $[Hb]$ and a fall in $[HbO_2]$ either during or after the contraction (Fig. 2). In the variable deceleration group, these changes occurred both during and after the period of the contraction and leading to a significant decrease in $[Hb_{diff}]$. In the late and prolonged deceleration groups, the significant fall in $[Hb_{diff}]$ occurred only after the contraction. The mean changes in $[Hb_{tot}]$ were not significantly different from the controls in any of the other contraction groups.

4. Discussion

Cardiotocography remains the cornerstone of intrapartum fetal surveillance, however, the accurate in-

Table 2
Mean changes in total cerebral haemoglobin concentration $[Hb_{tot}]$, haemoglobin oxygenation index $[Hb_{diff}]$ and FHR, during and after uterine contractions associated with different FHR alterations

	$\Delta[Hb_{tot}]$ ($\mu\text{mol}/100\text{g}$)		$\Delta[Hb_{diff}]$ ($\mu\text{mol}/100\text{g}$)		ΔFHR (bpm)	
	During	After	During	After	During	After
Control	-0.42 (0.50)	-0.06 (0.26)	0.00 (0.40)	-0.04 (0.46)	-1.47 (2.70)	-0.80 (2.91)
Acceleration	-0.19 (0.97)	-0.03 (0.55)	0.09 (0.34)	-0.24 (0.43)	6.93** (4.87)	2.20* (7.40)
Early	-0.48 (0.42)	0.04 (0.29)	-0.14 (0.16)	-0.11 (0.27)	-5.78 (3.88)	-0.24 (2.75)
Variable	-0.65 (0.83)	0.06 (0.56)	-0.25* (0.39)	-0.33* (0.38)	-13.27** (10.9)	-4.69* (9.31)
Late	-0.78 (0.49)	-0.16 (0.39)	-0.24 (0.45)	-0.85** (0.46)	-7.30 (6.90)	-10.40** (10.2)
Prolonged	-0.23 (0.95)	-0.06 (0.58)	-0.03 (0.44)	-0.65** (0.68)	-15.03 (17.3)	-28.01** (23.9)

Anova: ** $P < 0.001$; * $P < 0.01$.

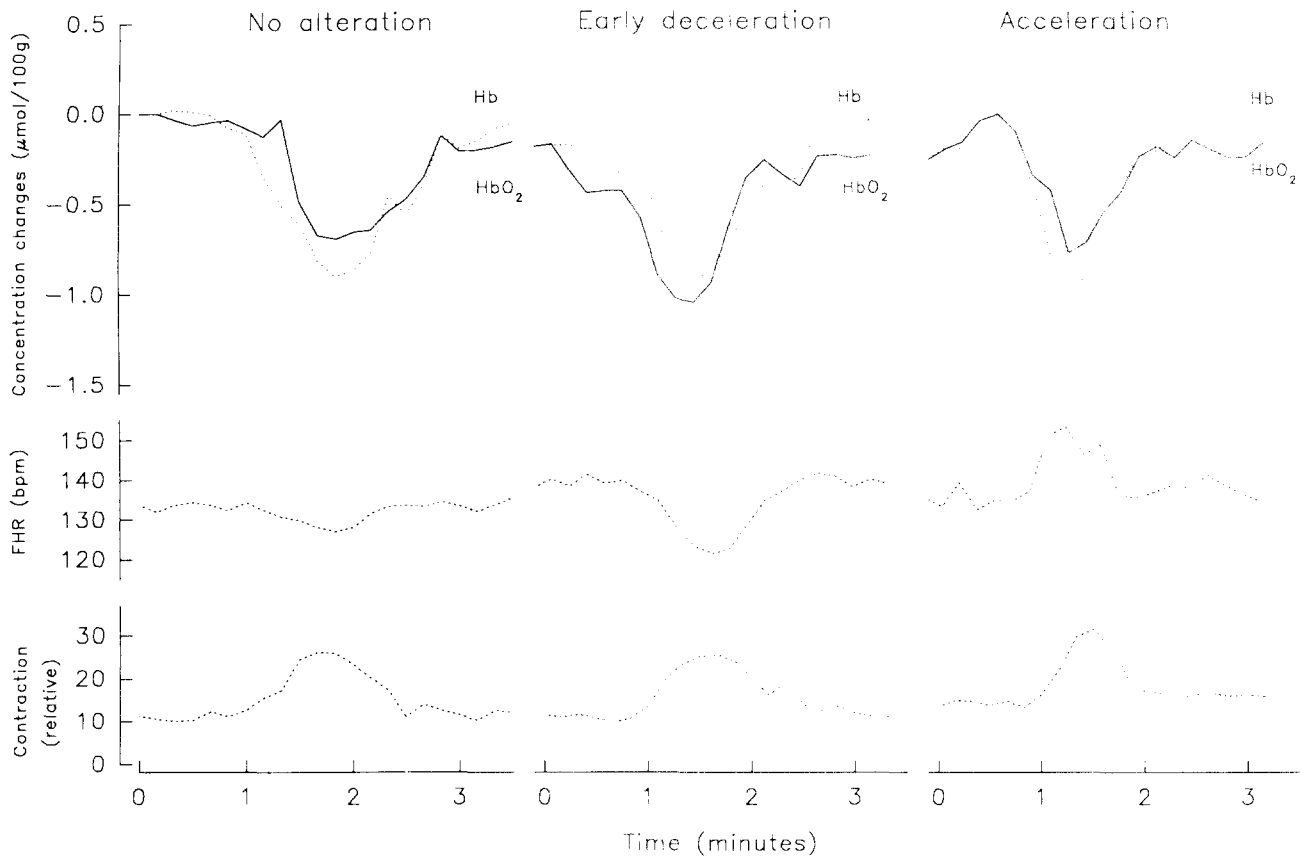


Fig. 1. Representative changes from baseline in cerebral oxyhaemoglobin [HbO₂] and deoxyhaemoglobin [Hb] from 3 fetuses demonstrating either no alteration, an acceleration or an early FHR deceleration.

interpretation of FHR alterations during contractions requires the clinician to have an understanding of the physiological mechanisms involved and, in particular, the relationship with fetal oxygenation. With the use of NIRS, we have demonstrated a close link between different types of fetal heart rate alterations during contractions and simultaneous changes in fetal cerebral oxygenation.

We chose to study the end of the first stage of labour, firstly as this is the most frequent time for fetal heart rate changes to occur; secondly, because FHR alterations during the active phase of the second stage are often difficult to interpret; and thirdly, because the diagnosis of full cervical dilatation provided a specific endpoint to the study. The group of patients that we studied was representative of mothers undergoing uncomplicated labour at term. There was a significant variation within the group in respect to maternal age, parity, gestational age and maternal analgesia. A much larger study would be required in order to investigate whether these factors had an influence on fetal cerebral oxygenation.

Uterine contractions subject the fetal head to con-

siderable compression [15] and in the absence of fetal heart rate changes, we observed parallel falls in cerebral oxyhaemoglobin and deoxyhaemoglobin concentrations, reflecting mechanical expulsion of blood from the cranial cavity without any change in oxygenation [4]. Similar findings were obtained in contractions associated with FHR accelerations, indicating that the acceleration in the fetal heart rate was not linked to significant changes in fetal oxygen delivery. This is consistent with the generally held belief that FHR accelerations in association with uterine contractions are an indication of fetal well-being and have been shown not to be associated with fetal acidemia [16–18].

Early fetal heart rate decelerations were also associated with simultaneous falls in both oxyhaemoglobin and deoxyhaemoglobin concentrations without evidence of deoxygenation. They are thought to be most commonly due to compression of the fetal head, which raises intracranial pressure and increases vagal tone, resulting in a fall in FHR which can be abolished by the administration of atropine [19]. Some authors have previously suggested that early FHR decelerations may lead to a transient reduction of cerebral blood flow and hence of

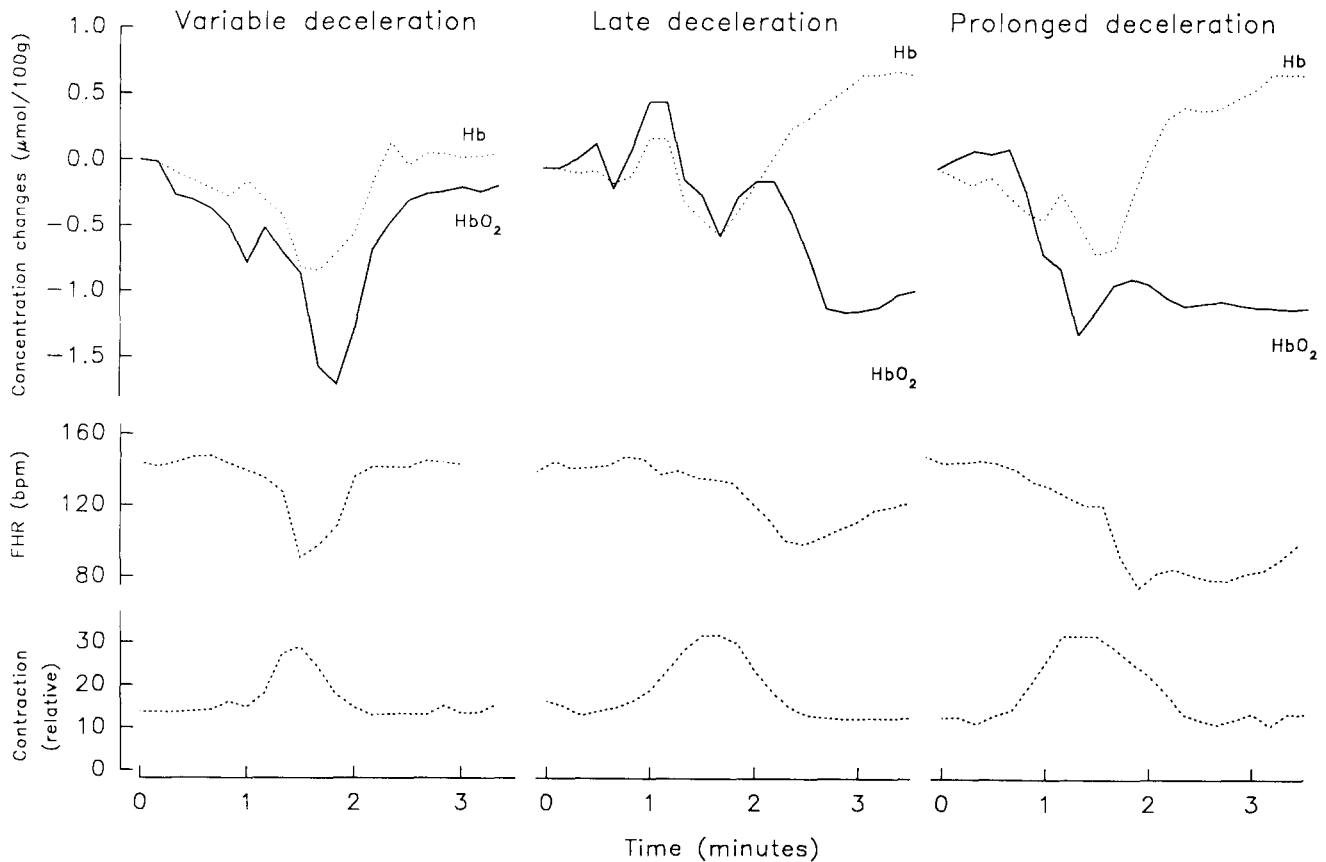


Fig. 2. Representative changes from baseline in cerebral oxyhaemoglobin [HbO₂] and deoxyhaemoglobin [Hb] from 3 fetuses demonstrating either a variable, late or prolonged FHR deceleration.

cerebral oxygenation [20], but our study has not been able to substantiate this. The mean changes in oxyhaemoglobin and deoxyhaemoglobin concentrations we observed were not significantly different from those during the control contractions. This supports the observations of Cibils, who could find no evidence that early uniform decelerations were ominous [21].

Variable FHR decelerations were associated with a significant decrease in cerebral oxygenation, which occurred both during and immediately after the uterine contraction. It is generally thought that variable FHR decelerations are produced by a vagal response and, during the first stage of labour, umbilical cord compression with partial or complete occlusion is believed to be the most common precipitating factor, although mechanical compression of the head has also been suggested as a possible mechanism [22]. Compression of the umbilical vein can interrupt the return of oxygenated blood to the fetus leading to fetal hypoxemia and acidemia, which in turn is likely to trigger the arterial chemoreceptors and produce an abrupt vagal bradycardia [23,24]. Our observations are consistent with this hypothesis. If compression of the umbilical artery had

occurred, this would have been likely to be associated with a transient increase in fetal blood pressure. However, we did not observe any change in cerebral blood volume compared with the control contractions suggesting that cerebral perfusion did not alter significantly, and a large change in fetal blood pressure was therefore unlikely. The risk of developing hypoxia may be suggested by the amplitude, duration and shape of the variable deceleration [14]. We could find no significant differences in changes in cerebral oxygenation or blood volume between variable decelerations when they were classified as either late or early in relation to the uterine contraction.

In contractions associated with late FHR decelerations, we observed a marked decrease in cerebral oxygenation which occurred after but not during the uterine contraction, as we have previously reported [8]. This is consistent with observations obtained in studies of experimental animals [25] which suggested that the delayed fall in FHR reflected the time required for oxygen in the intervillous space to become depleted and for the subsequent passage of a bolus of deoxygenated blood to travel from the placenta to the fetus. Thus, the

arrival of the deoxygenated blood from the umbilical vein caused a simultaneous fall in heart rate and in cerebral oxygenation.

In our study, prolonged FHR decelerations were always associated with a simultaneous and significant fall in cerebral oxygenation. In all cases, recovery of the FHR occurred within 5 min and this was accompanied by a return to baseline of oxyhaemoglobin and deoxyhaemoglobin concentrations, reflecting the transient nature of the changes in cerebral oxygenation. Prolonged FHR decelerations are usually sporadic, and are often related to uterine hyperstimulation, umbilical cord compression and maternal hypoxia or hypotension. Our studies suggest that the fall in heart rate is caused by deoxygenated blood reaching the fetal heart from the umbilical vein and, hence, the heart rate deceleration and the cerebral deoxygenation occurred simultaneously.

In summary, our study showed that uterine contractions during the first stage of labour which were associated with no alterations, accelerations or early decelerations of the fetal heart rate were not associated with any significant reduction in fetal cerebral oxygenation. Conversely, contractions associated with variable, late or prolonged decelerations were associated with significant but transient decreases in cerebral oxygenation as judged by a rise in deoxyhaemoglobin and a fall in oxyhaemoglobin concentrations and hence in haemoglobin oxygenation index. In nearly all cases, cerebral oxygenation returned to baseline as the heart rate normalised. At birth, there were no fetuses with severe acidemia (pH < 7.05) [26] or signs of hypoxic ischaemic encephalopathy, which is not surprising as even FHR patterns considered most ominous are only at best weakly associated with acidemia [18].

FHR patterns that are associated with a decrease in cerebral oxygenation are likely to constitute a greater risk for cerebral damage. However, other factors such as uterine contraction frequency [27], the duration and severity of the hypoxic insult, the timing of the development of acidemia [28] and the susceptibility of the fetal brain to hypoxic-ischaemic injury are clearly important. NIRS allows the physiological effects of fetal heart rate changes on the brain to be observed, and may contribute to an improved understanding of the role of cardiocography in intrapartum fetal surveillance.

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