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Fetal heart rate patterns and ECG ST segment changes preceding metabolic acidaemia at birth

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Objectives To compare the rates of abnormal ST segment patterns of the ECG and cardiotocographic (CTG) abnormalities in fetuses with metabolic acidaemia at birth and controls. To evaluate the inter-observer agreement in interpretation of ST analysis and CTG.

Design Case–control study.

Setting Three University hospitals in southern Sweden.

Population Cases and controls were selected from the Swedish randomised controlled trial on intrapartum monitoring, including 4966 fetuses monitored with a scalp electrode.

Methods Two obstetricians independently assessed the CTG and ST traces of 41 fetuses with metabolic acidaemia at birth and 101 controls, blinded to group, outcome and all clinical data. They classified each CTG trace and ST analysis as abnormal or not abnormal, and whether there was indication to intervene according to the CTG or to the CTG + ST guidelines. If their classification differed, assessment by a third obstetrician determined the final classification.

Main outcome measures Rates of CTG and ST abnormalities and decisions to intervene. Rates of inter-observer agreement.

Results CTG was classified as abnormal in 50% and ST in 63% of cases with acidaemia, and in 20% and 34% of controls, respectively. CTG abnormalities were judged to be indication for intervention in 45% and CTG + ST abnormalities in 56% of cases with acidaemia, and in 15% and 8% of controls, respectively. The proportion of agreement between the two initial observers was significantly higher for ST abnormalities (94%) than for CTG abnormalities (73%), and for indication to intervene according to CTG + ST (89%) than according to CTG alone (76%).

Conclusions The inter-observer agreement rate was higher for a decision to intervene based on CTG + ST than on CTG alone.

INTRODUCTION

Although cardiotocography (CTG) monitors the fetal heart rate, it indirectly monitors the fetal brain, controlling the heart rate.¹ In hypoxia, the fetal heart and brain are both organs with preferential oxygen supply, but when compensatory mechanisms fail, changes can be seen in the ST segment of the ECG, together with CTG abnormalities.² This premise, based on the experience from animal experiments, has been the basis for studies of fetal ECG in labour surveillance.^{2–7} In the fetal ECG, hypoxia typically causes an elevation of the ST segment and T wave, due to a catecholamine surge, β -adrenoceptor activation and myocardial glycogenolysis,^{4–6} whereas in some cases ST segment depression appears, probably reflecting endocardial hypoxia.⁷

Two randomised controlled trials have compared intrapartum monitoring with CTG alone and CTG combined with ST analysis.^{8,9} The first randomised controlled trial showed a reduction of operative deliveries for fetal distress,⁸ and the second also a reduction of metabolic acidaemia at birth with combined CTG + ST monitoring.^{9,10} Metabolic acidaemia is a marker of anaerobic metabolism and hypoxia, and although most neonates with this degree of acidaemia do well, they are at risk of neonatal encephalopathy and seizures.^{11,12} Data on CTG abnormalities and ST events in cases of asphyxia in the Swedish randomised controlled trial have been reported according to intention to treat.⁹ However, the rates of ST abnormalities in cases of acidaemia and in healthy controls have not previously been determined.

The first aim of the present study was to compare the rates of abnormal CTG and ST patterns in acidaemia cases and controls. Because ST information had been collected in a concealed fashion in the CTG arm of the randomised controlled trial, we also had the opportunity to evaluate the sensitivity and specificity for ST changes in predicting acidaemia. The second aim was to assess the reproducibility of ST and CTG assessments.

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METHODS

The Swedish randomised controlled trial was conducted during 18 months at three labour wards in southern Sweden.⁹ Women in active labour at more than 36 completed gestational weeks, with singleton fetuses in cephalic presentation, were included in the study after a decision to apply a fetal scalp electrode for continuous internal CTG recording. This relative high risk group corresponded to about a third of all women delivered at these departments during the study period. The STAN S 21 prototype fetal heart monitor (Neoventa Medical, Gothenburg, Sweden) randomly allocated the parturients at power-on to either monitoring with CTG only (CTG group), or to CTG combined with ST analysis (CTG + ST group). In both trial arms, fetal ECG signals were automatically stored in digital form. No ECG information was available in the CTG group, whereas in the CTG + ST group both the T/QRS ratio and significant ST events were displayed by the ST log.

Management in the CTG group was guided by CTG interpretation according to FIGO guidelines,¹³ with an option of fetal scalp blood sampling. In the CTG + ST group, the management was guided by CTG interpretation supported by computerised ST waveform assessment (ST log) according to the trial protocol (Table 1). The ST log automatically informed online about the occurrence of any ST events listed in the protocol. Intervention, as described above for the CTG group, was indicated at the occurrence of pre-terminal CTG, regardless of ST waveform, or in instances of abnormal or intermediate CTG patterns with ST events as listed in the protocol. No intervention was recommended if the CTG was normal, irrespective of ST changes. Fetal scalp blood sampling was optional also in this group.

Cord artery metabolic acidaemia was defined as a cord artery pH < 7.05 and base deficit in the extracellular fluid compartment (BD_{ecf}) >12.0 mmol/L, using the Siggaard-Andersen Acid Base Chart algorithm.¹⁴ Totally, 46 newborns had metabolic acidaemia at birth, 31 in the CTG group and 15 in the CTG + ST group.⁹

The study was approved by the Research Ethics Committees at the respective universities, and all participating women gave their informed consent before entering the study.

After completion of the randomised controlled trial, two obstetricians reviewed the CTG and ST traces from the 46 cases with metabolic acidaemia, together with the traces of three controls without acidaemia for each case. The reviewers were blinded to whether trace belonged to cases or controls, as well as to all other clinical data, including stage of labour and interventions. As controls, the infant delivered closest in time before and the two infants born after each case were chosen, provided the cord pH was >7.04 and BD_{ecf} < 12.1 mmol/L. Cases and controls with a total monitoring time of less than 40 minutes were excluded, since the first 30 minutes of monitoring were not assessed, and abnormalities during the last 10 minutes were not taken into account in the general classification (see below). We also excluded cases and controls where the monitoring was terminated more than 30 minutes before birth.

Obstetricians A and B had about 15 years and 10 years of clinical experience of CTG interpretation, respectively, and research experience of the method. Both obstetricians had taken part in the randomised controlled trial, and had about five years experience of the STAN method. The individual parameters of the CTG and ST traces were classified for the second 30-minute period, and for the last two 30-minute periods. The first 30-minute period was not assessed, as the STAN monitor needs 20 minutes for assessment of ST changes. Thereafter, the complete trace was classified, and the duration of CTG and ST changes assessed. In addition, the main classification was made according to whether or not the CTG and ST traces were abnormal for more than 10 minutes. An abnormal CTG was defined according to the FIGO criteria,¹³ and did not include intermediary CTG changes ('suspect' or 'non-reassuring' CTG) but only patterns defined as clearly abnormal. An abnormal ST event was defined according to the computer log: episodic rises in the T/QRS ratio exceeding 0.10, baseline rises exceeding 0.05 or repeated biphasic ST segments of type 2 or 3. In cases of reduced signal quality, visually detectable rises in the ST segment exceeding these levels were also defined as abnormal. Each observer also determined whether she/he found an indication to intervene because of CTG abnormalities, or due to combined CTG and ST changes according to the protocol used in the randomised controlled trial (Table 1). In the randomised controlled trial, a continuously abnormal CTG lasting for 60–90 minutes in the CTG + ST arm was also an indication to intervene, but, in the present study, such abnormality was only evaluated as need for intervention according to the CTG. Indication to intervene was defined as a need to take some action—delivery, scalp blood pH or alleviation of a probable cause of hypoxia. If observers A and B differed, the trace was also reviewed by a third

Table 1. CTG and ST changes indicating intervention according to the CTG + ST management protocol of the Swedish randomised clinical trial.

	Intermediary CTG ^a	Abnormal CTG	Pre-terminal CTG
Episodic T/QRS rise	>0.15	>0.10	regardless ST
Baseline T/QRS rise	>0.10	>0.05	regardless ST
Biphasic ST	continuous >5 minutes or >2 episodes	continuous >5 minutes or >1 episode	regardless ST

^a In the evaluation of ST abnormalities independently of the CTG in the present study, these ST changes were considered significant.

Table 2. Occurrence of CTG and ST abnormalities, and indication to intervene according to the three interpreters. Values are presented as *n* (%).

	Interpreter A		Interpreter B		Final classification ^a	
	Acidaemia (<i>n</i> = 41)	Controls (<i>n</i> = 101)	Acidaemia (<i>n</i> = 41)	Controls (<i>n</i> = 101)	Acidaemia (<i>n</i> = 40) ^b	Controls (<i>n</i> = 101)
Abnormal CTG	27 (66)	34 (34)	16 (39)	14 (14)	20 (50)	20 (20)
Significant ST events ^c	25 (61)	35 (35)	26 (63)	37 (37)	26 (63)	34 (34)
Indication to intervene						
According to CTG	25 (61)	26 (26)	16 (39)	10 (10)	18 (45)	15 (15)
According to CTG + ST	21 (51)	12 (12)	23 (56)	14 (14)	23 (56)	8 (8)

^a Including the assessment by interpreter C.^b Interpreter C found one CTG trace impossible to interpret (*n* = 40 for CTG classification).^c Changes that would have been indication to intervene if CTG were at least intermediary.

obstetrician (C), expert on fetal monitoring, deciding the final classification.

The results were statistically evaluated with the Medcalc statistical software (version 5). Fisher's exact test was used for discrete variables and the odds ratios (OR) with 95% confidence intervals (CI) were given.

RESULTS

Of 46 cases with metabolic acidaemia in the randomised controlled trial, two cases were excluded due to too short monitoring, and one was excluded as monitoring was

interrupted more than 30 minutes before birth. Two monitoring traces could not be found, leaving 41 cases for inclusion. Of these, 13 had been monitored with CTG + ST analysis in the randomised controlled trial, and 28 with CTG alone. After corresponding exclusions, 101 controls were included, 59 from the CTG + ST arm and 42 from the CTG arm of the randomised controlled trial.

According to the final classification, abnormal CTG patterns were identified in 50% and ST abnormalities in 63% of the cases with acidaemia, and in 20% and 34% of controls, respectively (Table 2). Of the 41 cases with acidaemia, 25 had ST events signalled by the log and one had visually detectable ST changes. Among the 101 controls, these figures were 31 and 3, respectively. The most common ST abnormalities were baseline T/QRS rises, occurring in 20 of the 41 cases (49%) and in 29 of 101 controls (29%). Episodic T/QRS rises occurred in seven cases (17%) and in eight controls (8%), and biphasic or negative ST changes were seen in seven cases (17%) and in four controls (4%).

CTG abnormalities were judged to be indication to intervene in 45% and CTG + ST abnormalities in 56% of cases with acidaemia, and in 15% and 8% of controls, respectively.

The sensitivity and specificity of the two methods to detect metabolic acidaemia are presented in Table 3. The individual interpretations by the two primary interpreters of CTG and ST abnormalities and indication to intervention differed. Interpreter A was more prone to judge the CTG

Table 3. Sensitivity and specificity of predicting metabolic acidaemia at birth for the CTG and ST abnormalities and indications to intervene according to the three interpretations (95% confidence intervals in brackets).

	Interpreter A	Interpreter B	Final classification ^a
Abnormal CTG			
Sensitivity (%)	66 (51–80)	39 (24–54)	50 (34–66)
Specificity (%)	66 (57–76)	86 (79–93)	80 (72–88)
Significant ST events			
Sensitivity (%)	61 (46–76)	63 (49–78)	63 (49–78)
Specificity (%)	65 (56–75)	63 (54–73)	66 (57–75)
Indication to intervene according to CTG			
Sensitivity (%)	61 (46–76)	39 (24–54)	45 (30–60)
Specificity (%)	74 (66–83)	90 (84–96)	85 (78–92)
Indication to intervene according to CTG + ST			
Sensitivity (%)	51 (36–37)	56 (41–71)	56 (41–71)
Specificity (%)	88 (82–94)	86 (79–94)	92 (87–97)

Nota bene that since a preselected material is analysed and the interventions within the randomised controlled trial were done based on CTG and ST analysis, the figures do not represent the true sensitivity and specificity for metabolic acidaemia at these changes. The results are used for comparison of the two monitoring methods and of the interpreter evaluations. For the information on prevalence see Table 2.

^a Including the assessment by interpreter C.**Table 4.** ST abnormalities and indications to intervene according to CTG + ST guidelines, in cases and controls monitored by CTG only in the randomised controlled trial. Figures according to the final classification.

	Cases (<i>n</i> = 28)	Controls (<i>n</i> = 42)
Significant ST events ^a	18 (64%)	18 (43%)
Indication to intervene according to CTG + ST	15 (54%)	4 (10%)

^a Changes that would have been indication to intervene if CTG were at least intermediary.

Table 5. Rate of agreement for the assessments by interpreters A and B in percent (95% confidence intervals).

Abnormal CTG	73 (66–80)
Indication to intervene according to CTG	77 (70–84)
ST events	94 (91–98)
Indication to intervene according to CTG + ST	89 (84–94)

traces as abnormal, and to suggest intervention, resulting in higher sensitivity, and lower specificity than interpreter B. Interpreter A tended to improve the specificity by using ST analysis, whereas interpreter B tended to improve the sensitivity. For the final assessment, both sensitivity and specificity tended to improve when ST analysis was added to CTG. None of these differences was statistically significant. OR (95% CI) for an indication to intervene in cases with acidaemia, based on ST + CTG compared with CTG only was 1.56 (0.60–4.11). OR (95% CI) for an indication to intervene in controls based on ST + CTG compared with CTG only was 0.57 (0.20–1.52).

For those monitored with CTG only in the randomised controlled trial, ST changes were present in 18 of 28 cases with acidaemia and in 4 of 42 controls, which was similar to the respective rates of 8 of 13 and 4 of 59 cases in the CTG + ST group. Table 4 shows the number of cases with ST events and indication to intervene according to the CTG + ST protocol, for cases and controls monitored by CTG only in the randomised controlled trial. The sensitivity (54%) and specificity (90%) for the CTG + ST protocol to identify acidaemia were similar to the figures in the total material. Because ST information was concealed for the clinicians managing the patients in this group, these figures reflect the true sensitivity and specificity for ST abnormalities to identify acidaemia.

As shown in Table 5, the rates of agreement between the observers were significantly higher for ST abnormalities and indication to intervene according to the CTG + ST guidelines, than for CTG abnormalities alone.

DISCUSSION

The aim of intrapartum fetal surveillance is to prevent intrauterine asphyxia, by identifying fetuses exposed to hypoxia and enabling timely intervention. Electronic fetal heart rate monitoring with CTG has a high sensitivity in identifying hypoxia,¹⁵ and randomised studies have shown that it may reduce morbidity in asphyxia, expressed as the rate of neonatal seizures.¹⁶ Still, despite CTG monitoring, perinatal asphyxia occurs and in such cases, misinterpretation of abnormal CTG traces is often contributing to adverse outcome.¹² Because CTG abnormalities are relatively common also in cases without significant fetal hypoxia, CTG monitoring may also lead to unnecessary interventions.¹⁶ These problems have motivated the development of complementary methods for fetal assessment, of

which ST analysis is one of the most recent. The Swedish randomised controlled trial has shown that using ST analysis of the fetal ECG as a complement to CTG may reduce the occurrence of metabolic acidaemia at birth,⁹ and thus, ST analysis may prevent hypoxia and acidaemia. Because the method has been proven efficacious, it would be expected to be reliable and sensitive.¹⁷

The first aim of the present study was to compare rates of abnormalities in the ST analysis in cases with acidaemia and controls with those of CTG. The rate of ST abnormalities in the controls in this study (34%) was much higher than a previously reported rate of ST abnormalities (8%).¹⁸ This was due to the fact that in the present study, all ST changes were considered regardless of the CTG. An indication to intervene based on CTG + ST was only found to be present in 8%. These results indicate that although solitary ST abnormalities may not be specific for hypoxia, used together with CTG, ST analysis may increase the specificity of fetal surveillance, without losing sensitivity. However, the differences in sensitivity and specificity were not statistically significant, and therefore, no firm conclusions can be drawn from this study. It should be stressed that according to the guidelines for ST analysis, ST events should not be an indication to intervene unless concomitant CTG changes occur, and the results are thus in concordance with these recommendations.

The difference between the two methods was not entirely consistent for the two primary interpreters. When evaluating the need for intervention, the interpreter with the lowest sensitivity for CTG gained sensitivity, and the one with the lowest specificity for CTG gained specificity, by the addition of ST analysis to CTG. This agrees well with the higher inter-observer agreement for ST than for CTG abnormalities. Consequently, fetal surveillance combining the two methods gives a more conform interpretation and management.

We must point out that in the present study it was not possible to assess the true sensitivity or specificity for prediction of metabolic acidosis by CTG, as CTG was used in the clinical management within both groups of the randomised controlled trial. The ST information for the fetuses randomised to CTG only in the randomised controlled trial was concealed for the clinicians managing the patients, and therefore we had the possibility to assess the true sensitivity and specificity for ST abnormalities. Although the numbers were small, the sensitivity and specificity in this subgroup were similar to those in the total material.

Our findings do not show that the addition of ST analysis to CTG increases the sensitivity to detect acidosis significantly when systematically analysed by experienced obstetricians. Probably, the reduction of acidosis achieved with the method in a clinical setting in the randomised controlled trial reflects the fact that ST information is easier to interpret than is the CTG trace. In addition, a method visually pointing out significant ST events forces the clinician to make a more thorough CTG interpretation. Another explanation for the reduction of metabolic acidaemia with ST

analysis is that it gives an additional indication of hypoxia in cases developing acidaemia, which we believe is especially helpful in the second stage of labour where CTG abnormalities are common, and may be difficult to interpret. It must, however, also be pointed out that there are false negative ST traces in cases of acidaemia, as well as there are false negative CTG traces. Of the fetuses in the randomised controlled trial that developed acidaemia despite monitoring, 37% had no significant ST events, and in 50% the CTG was not classified as abnormal.

The inter-observer variability in the detection of ST abnormalities was lower than that for CTG abnormalities. This is not surprising as the ST analysis is primarily performed by the computer, and the monitor flags changes that may be an indication to intervene. Still, the inter-observer agreement was not total. This is mainly explained by differences in the visual assessment of ST trace in cases with low signal quality. It is uncertain how reliable visual analysis is in such cases. Previous studies of CTG have found varying degrees of inter-observer agreement between poor and very good.^{19–23} In our study, the inter-observer agreement for CTG was reasonable; the classification differed most often in the final parts of the recordings, corresponding to the second stage of labour. It should be pointed out that the interpreters had no access to information about the labour, such as stage of labour, use of oxytocin, pain relief methods and maternal temperature. The second stage is the period when the CTG is most difficult to interpret²⁴ and it might be that the second stage is the period during which the addition of ST analysis to CTG is of greatest value. It is also likely that the agreement on the decision to intervene based solely on monitoring traces is lower than if clinical information is available as well.

According to the intervention protocol used in the Swedish randomised controlled trial, and also in the present study, a single ST event may be an indication to intervene provided the CTG is abnormal or intermediary ('non-reassuring') at the moment the ST event appears. Thus, an ST event gives momentary information about the time when there is indication to intervene, whereas CTG is assessed over time, and the exact time point when abnormalities are severe enough to indicate intervention may be less evident. Various CTG abnormalities have different causes and occur in different phases during the development of hypoxia. Some CTG abnormalities (e.g. severe variable decelerations), often caused by cord compression,²⁵ rather reflect that the fetus is at risk of developing hypoxia than hypoxia as such. Tachycardia, another CTG abnormality, may reflect the fetal compensatory mechanisms in hypoxemia before hypoxia develops.²⁶ Late decelerations may reflect hypoxemia in association with uterine contractions,²⁷ whereas decreased beat-to-beat variability, occurring late in the progress, may reflect established hypoxia with cerebral affection.²⁸ Thus, specific pathological CTG changes may have different prognostic significance, and, according to their duration, development in time and relation to the course of labour, the indication

to intervene will differ. In studies of sensitivity and specificity of CTG for adverse outcome, these considerations are difficult to take into account. In clinical practice, a decision on the time and mode of intervention is taken not solely based on the CTG or CTG + ST analysis. The clinical information, risk factors, stage of labour, parity, and so forth must also be considered. The present study also showed that some cases of fetal hypoxia occurred without any ST abnormalities. It is therefore important to consider clinical risk factors and signs, and, as stated in the management protocol of the randomised controlled trial, to act if CTG is pre-terminal and to consider intervention (e.g. fetal scalp blood sampling) in cases of persistently abnormal CTG patterns even if ST is normal.

In summary, ST analysis had a higher inter-observer agreement than CTG analysis, and used as a complement to CTG, it tended to increase the specificity in identifying fetal hypoxia without losing sensitivity, and to give a more conform indication to intervene.

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