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1 **CHANGES IN THE ST-INTERVAL SEGMENT OF THE FETAL**

2 **ELECTROCARDIOGRAM**

3 **IN RELATION TO ACID-BASE STATUS AT BIRTH**

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24 Running title: Fetal ECG changes and acidaemia

25 **ABSTRACT**

26 **Objective:** To assess the occurrence of ST-interval segment changes of the fetal electro-

27 cardiogram (ECG) and cardiotocographic (CTG) abnormalities preceding acidaemia at birth.

28 **Design:** Case-control study.

29 **Setting:** University hospital labour ward.

30 **Sample:** Newborns with severe cord artery metabolic acidaemia (pH <7.00 and lactate $\geq$10

31 mmol/l, n=24), moderate metabolic acidaemia (pH 7.00-7.09 and lactate $\geq$10; n=48),

32 acidaemia (pH 7.00-7.09; n=52), pre-acidaemia (pH 7.10-7.19; n=265), and controls (pH

33 $\geq$7.20; n=117).

34 **Methods:** Monitoring traces were assessed blinded to outcome.

35 **Main outcome measures:** CTG- and ST-changes.

36 **Results:** Any ST-event occurred significantly more often among cases with severe (79%) and

37 moderate (75%) metabolic acidaemia than among controls (50%). The difference was

38 restricted to baseline T/QRS-rises, and to the second stage of labour, during which any event

39 only occurred significantly more often among cases with severe metabolic acidaemia (62%)

40 than among controls (38%). ST-events coincided with abnormal CTG patterns in 67%, 44%,

41 40%, and 28% of cases with severe and moderate metabolic acidaemia, acidaemia and

42 preacidaemia, and in 12% of controls. ST-events with intermediary CTG were similarly

43 frequent in the case groups (0-6%) and controls (4%). The ST-guidelines stated intervention

44 in 96%, 62%, 73% and 49% in these case groups, and 23% of controls.

45 **Conclusions:** Only two of three cases with severe and less than half of cases with moderate

46 metabolic acidaemia were preceded by ST-events coinciding with CTG abnormalities. It is

47   therefore important to intervene for long lasting, rapidly deteriorating, or marked

48   (preterminal) CTG abnormalities, also in the absence of ST-events.

49

50   **Keywords:** fetal monitoring, ECG, ST-analysis, cardiotocography, acidaemia

Introduction

Analysis of the ST waveform in the fetal electrocardiogram (ECG) has been introduced as a complement to cardiotocography (CTG), aiming to reduce unnecessary interventions and the incidence of asphyxia, by increasing the specificity in identifying hypoxia.[1,2] The STAN S21 fetal heart monitor (Neoventa Medical, Gothenburg, Sweden) uses computerized ECG analysis to detect ST-interval changes that may reflect myocardial hypoxia: continuous or episodic rises in T/QRS amplitude ratio, or ST segment depressions ("biphasic ST").[1] The current system has been evaluated in three randomized trials, comparing monitoring with CTG and ST-analysis to CTG monitoring alone.[2-4] The largest trial, from Sweden, showed lower rates of metabolic acidaemia and operative deliveries for fetal distress in the CTG and ST group.[2] The second study from Finland showed a higher rate of acidaemia (pH <7.05) in the CTG and ST group,[3] whereas a smaller French study showed no differences.[4]

If ST-analysis should help us to prevent metabolic acidaemia, warning signs would be expected to appear *before* this degree of acidaemia is reached. Kwee *et al.* reported that, among cases monitored with a good signal quality until delivery, ST changes occurred together with abnormal CTG patterns in all five cases with pH < 7.00 and base deficit >12, but only in six of 13 cases with pH 7.00–7.04 and base deficit >12.[5] At the same time, ST events are frequent also in cases with normal CTG patterns,[6] and in cases without acidaemia.[7] There is still limited information about frequency of different electrocardiographic changes at various degrees of acidaemia. Furthermore, the association between the occurrence of ST-events and acidaemia might differ between the first and second stages of labour, since in a recent study, ST-events were only associated with abnormal CTG patterns during the second stage.[6]

The aim of the present study was to assess how often acidaemia at birth was preceded by different ST events and CTG changes. Specifically, we wanted to assess at which severity of

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75　acidaemia such changes occurred more often than among controls with a normal pH at birth.

76　We also intended to evaluate these associations for the first and second stages of labour.

77

78　**Methods**

79　This was a retrospective study including singleton fetuses in cephalic presentation delivered

80　after 36 completed weeks at Lund University Hospital during April 1st 2002 – September 31st

81　2007, monitored with STAN S21 fetal heart monitor (Neoventa Medical, Gothenburg,

82　Sweden). Indications for STAN monitoring were term high risk labour or CTG abnormalities,

83　but sometimes the monitor was used merely because it was available in the delivery room.

84

85　It was a case-control study, with a control group of fetuses with cord artery pH ≥ 7.20, and

86　four case groups. The first and second study groups included newborns with metabolic

87　acidaemia, defined as cord artery pH below 7.10 (population mean -2 standard deviations

88　(SD)) together with a cord artery lactate ≥ 10 mmol/l (population mean +2SD), sub-grouped

89　into severe (pH <7.00) and moderate (pH 7.00-7.09) metabolic acidaemia. The third group

90　included newborns with non-metabolic acidaemia (pH <7.10, lactate <10 mmol/l), and the

91　fourth newborns with pre-acidaemia (pH 7.10-7.19, representing 1-2 SD below population

92　mean). There were two reasons for using lactate instead of base deficit to define the metabolic

93　component of acidaemia. Firstly, lactate is a directly measured end product of anaerobic

94　metabolism, in contrast to base deficit which is calculated from pH and pCO_2 (with different

95　algorithms resulting in different results). Secondly, a recent study at our University showed

96　that lactate had a higher association with poor 5 minute Apgar scores than base deficit

97　(unpublished data).

98   The labour staff had used the STAN monitor since the Swedish RCT, with principally the

99   same protocol for intervention.[2,8] According to this protocol, intervention was recommended

100   when any ST-event (except solitary biphasic events) appeared together with an abnormal

101   CTG: baseline T/QRS rise >0.05, episodic T/QRS rise >0.10 or 2 repeated or continuous

102   biphasic ST-events. Intervention was also recommended when more pronounced ST-events

103   appeared together with an intermediary CTG: baseline T/QRS-rise >0.10, episodic T/QRS-

104   rise >0.15, or 3 repeated or continuous biphasic ST-events. Furthermore, intervention was

105   recommended if the CTG was considered persistently abnormal for 60 minutes, or

106   preterminal (defined as variability below 2 beats per minute), regardless of the presence of

107   any ST-events. When the protocol stated intervention, the clinician in charge decided which

108   intervention to undertake (delivery, fetal scalp blood sampling (FBS), or removal of a

109   possible cause of fetal distress), depending on the clinical circumstances.

110   Acid base status in cord artery and cord vein blood was routinely assessed by puncture of the

111   vessels immediately after birth. For the analysis, including assessment of the lactate level,

112   ABL 735 (Radiometer, Copenhagen) was used. A complete acid-base status from artery and

113   vein was obtained for 78% of all newborns during the study period. Acid-base values were

114   validated according to the criteria suggested by Westgate at al.[9]

115   From computerized records we identified singleton fetuses with cephalic presentation

116   delivered after 36 completed weeks who had been monitored with STAN and had available

117   acid-base data. Of these, all infants with cord artery pH < 7.10 were included. We also

118   randomly selected 200 infants with cord artery pH 7.10-7.14, 200 with pH 7.15-7.19, and 200

119   controls with pH >7.20. To avoid too many comparisons, it was later decided to group all

120   infants with pre-acidaemia (pH 7.10-7.19) together.

121   In order to evaluate the duration of CTG abnormalities and to assess the ST-analysis during a

122   reasonable period of time we only included those cases monitored for at least 60 minutes. We

123   also excluded cases with a gap between the end of monitoring and birth exceeding 20 minutes

124   as the association between monitoring and acid-base status would be difficult to evaluate.

125   One author (MM) assessed the electronically stored traces using the STAN-Viewer® program

126   (Neoventa Medical, Gothenburg, Sweden). All ST events registered in the ST log were

127   recorded, as well as monitoring time, and time with insufficient signal quality. Cases with low

128   signal quality were not excluded, since periods with low signal quality might be regarded as a

129   limitation of the method (as well as due to the user), but a sub-analysis of the occurrence of

130   ST events and CTG abnormalities was made for cases with metabolic acidaemia and good

131   signal quality during the last hour of monitoring.

132   Each monitoring trace was then assessed by another two independent observers (of whom one

133   was an experienced obstetrician) blinded to acid-base status at birth and other clinical data.

134   The CTG traces were classified as normal, intermediary, abnormal, or preterminal, using the

135   ST guidelines, [8] with the amendment of combined decelerations as a criterion for abnormal

136   CTG, since such a pattern has been associated with excessive oxytocin administration and low

137   scalp blood pH values.[10] The definition of a preterminal pattern was absent variability (< 2

138   beats per minute). The continuous duration of abnormal and preterminal CTG patterns before

139   birth were assessed. The examiners also classified the CTG in association with ST-events, and

140   if and when indications to intervene had occurred according to the guidelines. If the two

141   observers categorized a pattern, or indication to intervene, differently, the trace was finally

142   classified in consensus by the group.

143   In cases where both intermediary and abnormal CTG and ST-abnormalities existed at

144   different times the latter was registered as the indication to intervene. When no other

145   intervention criteria were present, we recorded the presence of a "rapidly deteriorating CTG

146   trace", suggested as a reason to consider intervention in the absence of ST-events.[8]

147   Abnormalities occurring during the last 10 minutes before birth were considered separately,

148   since this was considered too late to allow intervention.

149   Obstetrical data were retrieved from computerized obstetric records. The results were

150   analyzed with the Stat-View® software program. The chi-square test or Fisher's exact test (if

151   any expected number was below 5) were used for discrete variables, and Mann-Whitney test

152   for continuous variables.

153

154   **Results**

155   During the study period there were 17 445 deliveries, about 20% monitored by STAN S21.

156   The computerised search identified 787 cases and controls. Of these, 66 were excluded as

157   they had incorrectly been registered as monitored by STAN,117 as the recordings were less

158   than 60 minutes, 34 since the time interval between the end of monitoring and delivery

159   exceeded 20 minutes, 32 due to missing CTG and ST traces, and one where the breech mode

160   was used incorrectly. In addition, 31 cases and controls with missing venous samples or too

161   small differences in pH or pCO_2 between arterial and venous sample were excluded. After all

162   exclusions, 506 cases were left for analysis: 24 with severe metabolic acidaemia; 48 with

163   metabolic acidaemia; 52 with acidaemia, 265 with pre-acidaemia, and 117 controls.

164   Background obstetrical and neonatal characteristics are presented in Table 1. The case groups

165   had longer duration of labour and higher rates of instrumental delivery than controls.

166   Monitoring time varied between 1 and 18 hours for individual fetuses, and the median

167   monitoring time was higher in the case groups (3.6-4.3 h) than in controls (median 3.2 h)

168   (Table 2). Periods of low signal quality were common; 40-50% of cases and 36% of controls

169   had a period of insufficient signal quality of more than 15 minutes.

170   Half of the controls had at least one ST-event during monitoring (Table 3). Events occurred

171   more often in cases with severe (79%), and moderate (75%) metabolic acidaemia than in

172   controls, and this difference remained significant after excluding events the last 10 minutes

173   before delivery. During the first stage, the occurrence of ST-events was not higher in any

174   study group than among controls, but cases with severe metabolic acidaemia had higher rates

175   of ST events (62%) during the second stage than controls (38%).

176   The most common event, baseline rises of the T/QRS-ratio, was the only type occurring

177   significantly more often in cases with metabolic acidaemia than among controls. A rise of the

178   T/QRS-ratio above 0.10 was more frequent in cases with severe metabolic acidaemia (42%)

179   than among controls (15%). Ten of 72 cases with metabolic acidaemia had episodic T/QRS

180   rises; eight also had baseline rises. In one of the two the remaining cases the episodic rise

181   appeared at the time of spontaneous delivery. Only three cases with metabolic acidaemia (and

182   only two controls) had repeated biphasic ST events.

183   The combined analysis of CTG and fetal ECG is presented in Table 4. The simultaneous

184   occurrence of abnormal CTG and ST-events, being an indication to intervene, was 12% in

185   controls and highest among cases with severe (67%) and moderate (44%) metabolic

186   acidaemia. For traces with good signal quality during the last hour of monitoring, the rates

187   were 12 of 16 (75%) and 14 of 33 (42%) in these groups (not shown in table). Indication to

188   intervene for intermediary CTG patterns and ST-events did not appear significantly more

189   often in any study group than in controls. Any indication to intervene according to the

190   guidelines occurred in 23% of controls, and more often in all case groups (in 96% and 62% of

191   cases with severe and moderate metabolic acidaemia).

192   In the absence of other intervention criteria, a rapidly deteriorating CTG trace was present

193   only in one case of metabolic acidaemia, and in seven cases of pre-acidaemia (but in none of

194   the controls; not shown in table).

195   Three neonates delivered in the first stage by caesarean section had pH <7.10. Two of them

196   had ST-events, and in all three there were preterminal patterns >10 minutes.

197

198   **Discussion**

199   The present material, including a relatively large number of cases with metabolic acidaemia,

200   provided the opportunity to evaluate the relation between different ST-events and CTG-

201   abnormalities and acid-base status at birth. Cases and controls constituted a relatively high

202   risk group, since the STAN method was preferably used for high risk cases. The study groups

203   had longer median duration of labour, and 12-34% longer median monitoring time than

204   controls. This difference might have biased the results towards a higher rate of ST-events

205   among the cases, but the higher rates in cases with metabolic acidaemia remained when

206   calculating the number of events per monitored hour.

207   ST events coinciding with abnormal or intermediary CTG patterns were present in 67% of

208   cases with severe and 48% of cases with moderate metabolic acidaemia (75% and 42% in

209   cases with good signal quality). These figures are in agreement with previous reports in which

210   CTG and ST-changes preceded metabolic acidaemia (pH <7.05 and base deficit >12) in 56-

211   70%,[7,11] and acidaemia (pH <7.05) in 77% of cases.[12,13] With good signal quality, CTG and

212   ST-abnormalities has previously been reported to precede severe and moderate metabolic

213   acidaemia in 5/5 and 6/13 cases, resepectively,[5] and acidaemia in 89%.[12,13]

214   In the present study, an intermediary CTG together with pronounced ST events did not occur

215   more often in any study group than among controls. Possibly this intervention criterion is

216   unnecessary. However, such a conclusion may only be applicable if the CTG classification

217   strictly follows the guidelines. We are aware that many interpreters hesitate to categorise a

218   pattern as abnormal, and two thirds of the traces that we classified as abnormal according to

219   the criteria had been classified as intermediate by the midwives caring for the patients. An

220   intermediary CTG with ST events is therefore a reason for careful CTG evaluation, and the

221   midwife should discuss such cases with an obstetrician.

222   We found no association between ST-events during the first stage of labour and cord artery

223   pH. In a previous study, no association was found between CTG abnormality and ST events

224   during the first stage.[6] Hence, it appears as if most events during the first stage accompany

225   normal CTG patterns, and can be disregarded. However, there is no evidence that ST events

226   coinciding with CTG abnormalities are less significant in the first stage than in the second.

227   Our study only included three cases with pH below 7.10 after (caesarean) delivery in the first

228   stage (two with ST-events and abnormal CTG, and all three with preterminal patterns), which

229   were too few to evaluate the association between ST-events during the first stage and cord

230   artery pH.

231   We found no association between episodic T/QRS-rises or biphasic ST events and cord artery

232   pH. Episodic rises of the T wave are thought to reflect temporary hypoxia. It is conceivable

233   that a hypoxic episode is not associated with acidaemia as long as it is transitory. The results

234   may indicate that the relevance of episodic T/QRS rises is small. Biphasic ST events only

235   occurred in three cases with metabolic acidosis (and in two controls), and the study was

236   underpowered to evaluate the association between these events and acid-base status at birth.

237   Biphasic ST-events have been suggested to reflect depleted fetal glycogen reserves, and have

11

238 been associated with fetal growth restriction in observational studies.[1] In the present study

239 only one case with severe metabolic acidaemia, one with acidaemia, eight with pre-acidaemia

240 and three controls were SGA at birth (Table 1). None of these showed repeated biphasic ST-

241 events.

242 According to guidelines, an abnormal CTG of over 60 minutes and a preterminal pattern are

243 indications to intervene. At least one of these criteria were present in 22 of 24 cases with

244 severe, and in 22 of 48 with moderate metabolic acidaemia. Preterminal patterns were rare

245 among controls (2%) and frequent (83%) in cases with severe metabolic acidaemia. However,

246 only a third of cases with moderate metabolic acidaemia had preterminal patterns, indicating

247 that this, as the word implies, is a late sign of hypoxia.

248 Abnormal CTG patterns lasting more than 60 minutes have been associated with adverse

249 outcome in newborns with acidaemia.[14] However, most acidaemic fetuses did not have

250 abnormalities lasting for one hour, and waiting for 60 minutes is hazardous if the trace is

251 deteriorating. An expert group recently recommended intervention for rapidly deteriorating

252 patterns in the absence of ST events.[8] A definition of rapid deterioration is lacking, and the

253 criterion therefore difficult to evaluate. In our study, it was common that one examiner

254 categorised a decelerative second stage pattern with minimal variability as "rapidly

255 deteriorating" and the other as preterminal, supporting intervention for such patterns. If STAN

256 users recognise that only half to two thirds of cases with metabolic acidaemia are associated

257 with ST events, the importance to intervene in cases with long-lasting, rapidly deteriorating,

258 or preterminal CTG patterns will be better understood.

259 According to the guidelines, intervention was indicated in 96% and 62% among cases with

260 severe and moderate metabolic acidaemia, and in 73% of cases with acidaemia. Still, the rates

261 of operative delivery for fetal distress were only 46%, 23% and 15% in these groups.

262   Although other interventions than operative delivery had sometimes been undertaken, we

263   were worried the criteria that in some cases had been neglected.

264   Intervention was also indicated for 23% of controls. This figure may seem high, even in a

265   high risk population. It should, however, be emphasized that "intervention" must not always

266   be operative delivery. In doubtful cases, FBS for the analysis of pH or lactate may be useful.

267   Actually, the STAN method with the current guidelines has only been evaluated with FBS as

268   an option.[2-4] In the Swedish RCT, FBS was performed at similar rates in the study arms (11%

269   and 9%),[2] whereas in the study from Finland, twice as many samples were taken in the CTG

270   arm (16%) as in the CTG+ST arm (7%).[3] This might be one explanation for the lower rate of

271   acidaemia in the CTG arm of that study. The rate of FBS also differed between the study arms

272   in the French study, but were high in both the CTG (62%) and CTG+ST arm (27%).[4]

273   None of the RCTs indicated an increased rate of operative delivery with the use of STAN. It

274   must be stressed that the occurrence of ST-events should not trigger interventions when CTG

275   patterns are normal – otherwise the high rate of ST-events among non-hypoxic fetuses may

276   lead to many unnecessary and potentially harmful procedures. Even worse is if STAN users

277   become accustomed to the occurrence of ST-events and do not react when indicated, i.e. in

278   the presence of an abnormal CTG. If the additional information provided by STAN is to be

279   useful, as it was in the Swedish RCT,[2] accurate CTG interpretation is essential.

280

281   **Conclusion**

282   This study showed that although the presence of ST-events increases the probability of fetal

283   acidaemia, events are frequent (50%) also among controls with normal cord blood gas values.

284   ST-events together with abnormal CTG patterns, reflecting hypoxia, appear late in the

285   hypoxic process, and are inconsistent, occurring in half of cases with moderate, and in two of

286 three cases with severe metabolic acidaemia. It is therefore important to act when CTG

287 abnormalities are marked or long lasting, also in the absence of ST-events. This is in

288 agreement with recently published recommendations.[8]

289

290 **Disclosure of interests**

291 None of the authors has any commercial or other conflicting interest in STAN or other

292 methods of fetal monitoring.

293

294 **Contribution to authorship**

295 Malin Melin contributed in planning the study, assessed the ST-information from the

296 monitoring traces, and contributed to manuscript writing and data analysis. Anna Bonnevier

297 assessed fetal heart rate traces, and reviewed the manuscript. Monika Cardell assessed fetal

298 heart rate traces, and contributed to manuscript writing. Linda Hogan assessed fetal heart rate

299 traces, and reviewed the manuscript. Andreas Herbst planned and coordinated the study,

300 assessed fetal heart rate traces, analysed the data, and wrote the final manuscript.

301

302 **Details of ethics approval**

303 Since this was a retrospective study of data from patients´ files and monitoring traces from

304 our unit, no ethics committee approval was needed.

305

306 **Funding**

307 This study was carried out without funding.

14

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Table 1. Background obstetrical and neonatal characteristics. Figures are presented as numbers (%) if not stated otherwise. Statistically significant differences (p<0.05) between cases and controls are marked with an asterisk (*).

	Severe metabolic acidaemia[1] N=24	Metabolic acidaemia[2] N=48	Acidaemia[3] N=52	Preacidaemia[4] N=265	Controls[5] N=117
Nulliparous mother	16 (67)	38 (79)	39 (75)	201 (76)	77 (66)
Post term pregnancy (≥42+0 weeks)	5 (21)	6 (12)	7 (13)	23 (9)	13 (11)
Induction of labour	11 (46)*	8 (17)	10 (19)	36 (14)	24 (21)
Duration of 1st stage[6] (h), median (range)	4.2 (0-8.7)	6.4 (0-16.5)*	5.1 (0-17.8)*	5.0 (0-29.0)*	3.5 (0-16.0)
Duration of 2nd stage (h), median (range)	1.1 (0-3.1)	1.5 (0-5.8)*	1.2 (0-7.0)*	1.3 (0-6.2)*	0.9 (0-9.3)
Fetal scalp blood sampling	6 (25)	6 (12)	8 (15)	21 (8)	12 (10)
Instrumental delivery	8 (33)	14 (29)*	15 (29)*	61 (23)*	10 (9)
Caesarean section	4 (17)	3 (6)	0 (0)	10 (4)	7 (6)
Operative delivery for fetal distress	11 (46)*	11 (23)*	8 (15)*	46 (17)*	9 (8)
Male gender	15 (62)	27 (56)	25 (48)	147 (55)	59 (50)
Birth weight (kg), median (range)	3.6 (2.7-4.7)	3.8 (2.8-4.9)	3.6 (2.2-4.9)	3.7 (2.3-5.2)	3.7 (2.4-5.0)
Small for gestational age at birth[7]	1 (4)	0	1 (2)	8 (3)	3 (3)
Cord artery pH, median (range)	6.96 (6.77-6.99)*	7.06 (7.00-7.09)*	7.07 (7.01-7.09)*	7.15 (7.10-7.19)*	7.25 (7.20-7.42)
Cord artery lactate, mmol/l, median (range)	13.2 (10.4-21.0)*	11.2 (10.0-18.0)*	8.7 (5.8-9.9)*	7.2 (3.4-13.3)*	4.6 (1.9-8.9)
5-min Apgar score <7	8 (33)*	3 (6)	6 (12)*	2 (1)	0 (0)
Admission to neonatal care unit	16 (67)*	11 (23)*	8 (16)*	19 (7)	5 (4)

[1] Cord artery pH <7.00 and lactate ≥ 10 mmol/l

[2] Cord artery pH 7.00-7.09 and lactate ≥ 10 mmol/l

[3] Cord artery pH 7.00-7.09, lactate < 10 mmol/l

[4] Cord artery pH 7.10-7.19

[5] Cord artery pH ≥7.20

[6] From regular contractions and cervical dilation ≥4 cm

[7] Below -22% (2 SD) from expected weight at gestational age

Table 2. Background monitoring characteristics. Figures are presented as numbers (%) if not stated otherwise.

Statistically significant differences (p <0.05) compared with controls are marked with an asterisk (*).

	Severe metabolic acidaemia N=24	Metabolic acidaemia N=48	Acidaemia N=52	Preaciemia N=265	Controls N=117
Duration of STAN monitoring (hours)[1]	4.1 (1.2-10.2)	4.3 (1.0-11.8)**	3.6 (1.0-13.8)*	4.2 (1.0-18.6)**	3.2 (1.0-12.0)
Disconnected >15 minutes	1 (4)*	17 (35)	14 (30)	79 (30)	29 (25)
Insufficient signal quality >15 minutes	12 (50)	21 (44)	26 (50)	107 (40)	42 (36)
STAN-monitoring started during the first stage of labour[2]	23 (96)	43 (90)	48 (92)	237 (89)	103 (88)

1. Monitoring time minus disconnected time

2. Monitoring started at least 20 minutes before full cervical dilatation.

Table 3. Occurrence of different types of ST-events in relation to cord artery acid-base status. Figures are presented as numbers (%) if not stated otherwise. Each group is compared with controls (pH $\geq$7.20), and significant differences are marked with * (p <0.05) or ** (p <0.01).

	Severe metabolic acidaemia N=24	Metabolic acidaemia N=48	Acidaemia N=52	Preacidaemia N=265	Controls N=117
Number of ST-events, median (range)	3.5 (0-15)*	2 (0-14)*	1 (0-23)	1 (0-40)	1 (0-73)
Any baseline T/QRS rise (>0.06)	18 (75)*	33 (69)*	27 (52)	144 (54)	56 (49)
Baseline T/QRS rise >0.10	10 (42)*	8 (17)	9 (17)	42 (16)	17 (15)
Any episodic T/QRS rise (>0.10)	3 (12)	7 (15)	10 (19)	33 (12)	11 (9)
Episodic T/QRS rise >0.15	3 (12)	3 (6)	4 (8)	12 (5)	5 (4)
Repeated biphasic ST	1 (4)	2 (4)	4 (8)	13 (5)	2 (2)
Any ST-event[1]	19 (79)*	36 (75)**	31 (60)	151 (57)	58 (50)
Any ST-event[1], last 10 minutes before birth excluded	19 (79)**	32 (67)*	29 (56)	139 (52)	54 (46)
Monitoring started first stage[2]	**N=23**	**N=43**	**N=48**	**N=237**	**N=103**
Any event during first stage[1]	12 (52)	20 (47)	20 (42)	107 (45)	41 (40)
Monitored during second stage[3]	**N=21**	**N=45**	**N=47**	**N=228**	**N=89**
Any event during second stage[1]	13 (62)*	20 (44)	21 (45)	93 (41)	34 (38)
Any event during second stage, last 10 minutes before birth excluded[1]	13 (62)*	15 (33)	18 (38)	77 (34)	28 (31)

[1] Single biphasic events (occurring in 6 cases) were not included, since this is never indication to intervene according to guidelines.

[2] Only those with monitoring started at least 20 minutes before full cervical dilatation included.

[3] Cesarean deliveries before full cervical dilatation and cases with second stage shorter than 20 minutes excluded.