Intrapartum cardiotocography with simultaneous maternal heart rate registration improves neonatal outcome

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BACKGROUND: Intrapartum cardiotocographic monitoring of fetal heart rate by abdominal external ultrasound transducer without simultaneous maternal heart rate recording has been associated with increased risk of early neonatal death and other asphyxia-related neonatal outcomes. It is unclear, however, whether this increase in risk is independently associated with fetal surveillance method or is attributable to other factors.

OBJECTIVE: This study aimed to compare different fetal surveillance methods and their association with adverse short- and long-term fetal and neonatal outcomes in a large retrospective cohort of spontaneous term deliveries.

STUDY DESIGN: Fetal heart rate and maternal heart rate patterns were recorded by cardiotocography during labor in spontaneous term singleton cephalic vaginal deliveries in the Hospital District of Helsinki and Uusimaa, Finland between October 1, 2005, and September 30, 2023. According to the method of cardiotocography monitoring at birth, the cohort was divided into the following 3 groups: women with ultrasound transducer, women with both ultrasound transducer and maternal heart rate transducer, and women with internal fetal scalp electrode. Umbilical artery pH and base excess values, low 1- and 5-minute Apgar scores, need for intubation and resuscitation, neonatal intensive care unit admission for asphyxia, neonatal encephalopathy, and early neonatal death were used as outcome variables.

RESULTS: Among the 213,798 deliveries that met the inclusion criteria, the monitoring type was external ultrasound transducer in 81,559 (38.1%), both external ultrasound transducer and maternal heart rate recording in 62,268 (29.1%), and fetal scalp electrode in 69,971 (32.7%) cases, respectively. The rates of both neonatal encephalopathy (odds ratio, 1.48; 95% confidence interval, 1.08–2.02) and severe acidemia (umbilical artery pH <7.00 and/or umbilical artery base excess ≤ -12.0 mmol/L) (odds ratio, 2.03; 95% confidence interval, 1.65–2.50) were higher in fetuses of women with ultrasound transducer alone compared with those of women with concurrent external fetal and maternal heart rate recording. Monitoring with ultrasound transducer alone was also associated with increased risk of neonatal intubation for resuscitation (odds ratio, 1.22; 95% confidence interval, 1.03–1.44). A greater risk of severe neonatal acidemia was observed both in the ultrasound transducer (odds ratio, 2.78; 95% confidence interval, 2.23–3.48) and concurrent ultrasound transducer and maternal heart rate recording (odds ratio, 1.37; 95% confidence interval, 1.05–1.78) groups compared with those monitored with fetal scalp electrodes. No difference in risk of neonatal encephalopathy was found between newborns monitored with concurrent ultrasound transducer and maternal heart rate recording and those monitored with fetal scalp electrodes.

CONCLUSION: The use of external ultrasound transducer monitoring of fetal heart rate without simultaneous maternal heart rate recording is associated with higher rates of neonatal encephalopathy and severe neonatal acidemia. We suggest that either external fetal heart rate monitoring with concurrent maternal heart rate recording or internal fetal scalp electrode be used routinely as a fetal surveillance tool in term deliveries.

Key words: cardiotocography, electronic fetal monitoring, fetal heart rate, maternal heart rate, neonatal outcome, perinatal asphyxia

Introduction

Cardiotocography (CTG) is the most widely used method of fetal surveillance during labor, primarily aimed at identifying fetuses at risk of hypoxia.[1] The CTG evaluation of fetal well-being involves electronic monitoring of uterine activity and fetal heart rate (FHR) patterns. Noninvasive methods, such as tocodynamometry and the use of a Doppler ultrasound transducer (US), or invasive approaches with an intrauterine fetal scalp electrode (FSE), are used to capture these signals. Although the internal FSE requires ruptured membranes and has been associated with a small risk of fetal infection and cephalohematoma,[2] along with being more expensive because of the need for a disposable electrode, it typically provides higher signal quality than the external methods.[3,4] Nonetheless, new-generation CTG monitors offer the option of continuous maternal heart rate (MHR) monitoring through maternal electrocardiogram (ECG), pulse oximetry, or a sensor integrated into the tocodynamometer enabling noninvasive simultaneous FHR and MHR monitoring without the need for additional transducers.[5] It is notable, however, that the MHR captured by maternal ECG is not averaged and presents the MHR with true beat-to-beat, whereas the MHR obtained from pulse oximetry or via tocodynamometer is averaged and will not trace the FHR signal concurrently.[5,6]

US placed on the maternal abdomen is the most common method for continuous FHR monitoring.[2,7] In particular,

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AJOG at a Glance

Why was this study conducted?

Recent reports suggest that cardiotocographic fetal heart rate (FHR) monitoring with an external ultrasound transducer (US) can increase asphyxia-related neonatal outcomes compared with other electronic fetal monitoring methods. This could potentially be due to maternal heart rate (MHR) artifacts, which may mask abnormal FHR patterns.

Key findings

Our study demonstrates that there is increased independent risk of asphyxia-related neonatal outcomes for fetuses of mothers monitored during labor by external US alone compared with fetuses of mothers who have concurrent MHR recording or internal fetal scalp monitoring.

What does this add to what is known?

Use of external fetal monitoring without simultaneous MHR monitoring increased the risk of neonatal complications in a large cohort of spontaneous term deliveries. Obstetricians and midwives should be increasingly aware of the effect of US alone for FHR monitoring on potential adverse outcomes.

FIGO (International Federation of Gynecology and Obstetrics) guidelines recommend external FHR monitoring as the preferred method for routine CTG monitoring during the intrapartum period, as long as a tracing of acceptable signal quality can be obtained.[8] In recent reports, however, external FHR monitoring by US alone has been associated with increased risk of unexpected perinatal death potentially because of poor signal quality or MHR artifact.[6] It is known that abnormal FHR patterns may be obscured by maternal pulse, particularly in the second stage of labor.[9,10] Consequently, previous reports have called for research into the simultaneous monitoring of MHR and intrapartum FHR.[11–13]

In the present study, external methods for FHR recording with or without concurrent MHR registration were compared with monitoring with a standard FSE. In a large retrospective birth cohort, the aim of the study was to evaluate the fetal surveillance methods using intrapartum CTG and their association with adverse short- and long-term fetal and neonatal outcomes. In addition, our objective was to determine whether the hypothesized increase in risk of unfavorable perinatal outcome is independently associated with the fetal surveillance method or related to other contributing fetal, maternal, or delivery-related factors.

Materials and Methods

Requirements and characteristics of the participants

The data were collected at the Hospital District of Helsinki and Uusimaa (HUS), Finland between October 1, 2005, and September 30, 2023. The data consisted of the following 4 main components: (1) intrapartum CTG recordings, (2) clinical maternal, fetal, neonatal, and delivery-related data (Table 1), (3) the results of the umbilical artery (UA) blood gas analyses (Table 2), and (4) detailed data on neonatal intensive care unit (NICU) treatment, including neonatal encephalopathy diagnoses.

The cohort included 213,798 term (≥37 weeks of gestation) singleton deliveries with continuously monitored CTG tracings. All women in the cohort were in the active phase of labor with regular uterine contractions and had a spontaneous cephalic delivery. Exclusion criteria were preterm (<37 weeks of gestation) pregnancies, cesarean deliveries and instrumental (forceps or vacuum-assisted) deliveries, breech deliveries, twins, maternal blood-borne contagious diseases or suspected fetal blood disorders (anemia, thrombocytopenia), cases without UA blood gas results, and cases with major congenital malformations.

Data collection

The clinical data were retrospectively collected from electronic obstetrical patient records (Obstetrix [Obstetrix Medical Group, Englewood, CO] and Apotti [Oy Apotti Ab, Helsinki, Finland]). The results of the UA blood gas analyses were obtained from the HUS Weblab Clinical laboratory information system (HUS, Helsinki, Finland). Detailed data on NICU care, intubation for resuscitation, and neonatal encephalopathy diagnoses were collected from the electronic medical records (Centricity Critical Care [GE HealthCare, Chicago, IL]). Apgar scores at 1 and 5 minutes were routinely assessed, and UA blood gas analysis at birth was performed using the Siemens RAPIDLab 248/348 Blood Gas System (Siemens AG, Munich, Germany).

The CTGs were recorded using Avalon FM20/30 (Philips Healthcare, Andover, MA) or HP Series 50A M1350/1A (Hewlett Packard, Boeblingen, Germany) fetal monitors. CTG tracings were recorded with paper speed of 1 cm per minute. No noninvasive fetal ECG, invasive fetal ST-waveform analysis, or other computerized intrapartum surveillance methods were used in the present cohort. Continuous MHR was obtained through tocodynamometry or pulse oximetry, and was presented as a curve on the same display as FHR (Figure 1). The FHR registration method was automatically marked with an electrical stamp on the CTG tracings, and the last FHR registration method used before birth was collected from the patient archive. All CTG recordings were stored in visual and electrical forms in the Milou (Medexa, Limhamn, Sweden) CTG database at the Data Analysis and Management Department of HUS.

Primary and secondary outcomes

The primary outcomes were pH <7.00 or base excess (BE) (negative value on

TABLE 1
Maternal, delivery-related, and neonatal characteristics when intrapartum fetal monitoring was conducted with external ultrasound transducer with or without simultaneous maternal heart rate recording, or with fetal scalp electrode

Maternal, delivery-related, and neonatal variables	Total	External US	External US+MHR	Internal FSE	External US vs external US+MHR Differences (95% CI) *P* value	External US vs internal FSE Differences (95% CI) *P* value	External US+MHR vs internal FSE Differences (95% CI) *P* value
Number	213,798	81,559	62,268	69,971	143,827	151,530	132,239
Maternal variables							
Maternal age ≥35 y	52,166 (24.4)	19,833 (24.3)	15,191 (24.4)	17,142 (24.5)	−0.1 (−0.5 to 0.3) .688	−0.2 (−0.6 to 0.3) .411	−0.1 (−0.6 to 0.4) .660
Obesity, prepregnancy BMI ≥30.0 (kg/m^2)	22,876 (10.7)	7236 (8.9)	6543 (10.5)	9097 (13.0)	−1.6 (−2.0 to 1.2) <.001	−4.1 (−4.4 to −3.8) <.001	−2.5 (−2.8 to −2.2) <.001
Nulliparous	11,7803 (55.1)	44,116 (54.1)	34,434 (55.3)	39,253 (56.1)	−1.2 (−1.7 to −0.7) <.001	−2.0 (−2.5 to −1.5) <.001	−0.8 (−1.3 to −0.3) .004
Gestational age at delivery (wk)	40.1 (±1.2)	40.0 (±1.3)	40.1 (±1.2)	40.1 (±1.2)	−0.1 (−0.3 to 0.2) .513	−0.1 (−0.3 to 0.3) .792	0.0 (−0.2 to 0.2) .880
Smoking	18,601 (8.7)	7094 (8.7)	5292 (8.5)	6215 (8.8)	0.2 (−0.1 to 0.5) .210	−0.1 (−0.5 to 0.1) .181	−0.3 (−0.6 to 0.0) .014
Type 1 or 2 diabetes mellitus	428 (0.2)	93 (0.1)	125 (0.2)	210 (0.3)	−0.1 (−0.4 to 0.3) <.001	−0.2 (−0.4 to −0.1) <.001	−0.1 (−0.2 to 0.0) <.001
Gestational diabetes mellitus	25,870 (12.1)	9223 (11.3)	7621 (12.2)	9026 (12.9)	−0.9 (−0.6 to −1.3) <.001	−1.6 (−1.9 to −1.3) <.001	−0.7 (−1.0 to −0.3) <.001
Preeclampsia	6209 (2.9)	2365 (2.9)	1955 (3.1)	1889 (2.7)	−0.2 (−0.4 to 0.0) .009	0.2 (0.3—0.0) .001	0.4 (0.6—0.2) <.001
Fever (≥38.0°C) during delivery	6842 (3.2)	2610 (3.2)	2063 (3.3)	2169 (3.1)	−0.1 (−0.3 to 0.1) .232	0.1 (−0.1 to 0.3) .262	0.2 (0.0—0.4) .028
Delivery-related variables							
Labor type							
Spontaneous onset	168,687 (78.9)	64,513 (79.1)	51,696 (83.0)	52,478 (75.0)	−3.9 (−4.3 to −3.4) <.001	4.1 (3.8—4.4) <.001	8.0 (7.6—8.4) <.001
Induction	45,111 (21.1)	17,046 (20.9)	10,572 (17.0)	17,493 (25.0)			
Oxytocin augmentation	117,589 (55.0)	44,780 (54.9)	34,185 (54.9)	38,624 (55.2)	0.0 (−0.1 to 0.1) .780	−0.3 (−0.8 to 0.2) .251	−0.3 (−0.8 to 0.2) .272
Meconium-stained amniotic fluid	29,932 (14.0)	9966 (12.2)	7721 (12.4)	12,245 (17.5)	−0.2 (−0.5 to 0.2) .306	−5.3 (−5.6 to −4.9) <.001	−5.1 (−5.5 to −4.7) <.001
Epidural analgesia	116,092 (54.3)	43,937 (53.9)	33,812 (54.3)	38,343 (54.8)	−0.4 (−0.9 to 0.1) .131	−0.9 (−1.4 to −0.4) <.001	−0.5 (−1.0 to 0.0) .068

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TABLE 1
Maternal, delivery-related, and neonatal characteristics when intrapartum fetal monitoring was conducted with external ultrasound transducer with or without simultaneous maternal heart rate recording, or with fetal scalp electrode *(continued)*

Maternal, delivery-related, and neonatal variables	Total	External US	External US+MHR	Internal FSE	External US vs external US+MHR Differences (95% CI) *P* value	External US vs internal FSE Differences (95% CI) *P* value	External US+MHR vs internal FSE Differences (95% CI) *P* value
Mode of delivery							
Spontaneous vertex	207,812 (97.2)	79,357 (97.3)	60,443 (97.1)	68,012 (97.2)	0.2 (0.1−0.4) .008	0.1 (−0.1 to 0.3) .240	−0.1 (−0.3 to 0.0) .145
Other spontaneous cephalic (nonvertex)	5986 (2.8)	2202 (2.7)	1825 (2.9)	1959 (2.8)			
Shoulder dystocia	314 (0.1)	127 (0.2)	86 (0.1)	101 (0.1)	0.1 (0.0−0.1) .552	0.1 (0.0−0.1) .618	0.0 (0.0−0.0) .771
Neonatal variables							
Female sex	106,044 (49.6)	40,453 (49.6)	30,815 (49.5)	34,776 (49.7)	0.1 (−0.1 to 0.3) .388	−0.1 (−0.6 to 0.4) .696	−0.2 (−0.7 to 0.2) .197
FGR	1924 (0.9)	734 (0.9)	630 (1.0)	560 (0.8)	−0.1 (−0.1 to −0.1) .022	0.1 (0.1−0.1) .004	0.2 (0.2−0.2) <.001
SGA (birthweight z-score <2.0 SD-units)	6413 (3.0)	2447 (3.0)	2073 (3.3)	1888 (2.7)	−0.3 (−0.5 to 0.1) <.001	0.3 (0.1−0.5) <.001	0.6 (0.4−0.8) <.001
LGA (birthweight z-score >2.0 SD-units)	6196 (2.9)	2283 (2.8)	1814 (2.9)	2099 (3.0)	−0.1 (−0.3 to 0.1) .021	−0.2 (−0.4 to 0.0) <.001	−0.1 (−0.3 to 0.1) .357
Postterm (≥42.0 wk)	14,752 (6.9)	5294 (6.5)	4421 (7.1)	5037 (7.2)	−0.6 (−0.8 to −0.3) <.001	−0.7 (−0.8 to −0.6) <.001	−0.1 (−0.2 to 0.0) .485

Data are presented as mean±SD or as absolute number (percentage).

BMI, body mass index; *CI*, confidence interval; *FGR*, fetal growth restriction; *FSE*, fetal scalp electrode; *LGA*, large for gestational age; *MHR*, maternal heart rate; *SD*, standard deviation; *SGA*, small for gestational age; *US*, ultrasound transducer.

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TABLE 2
Neonatal outcome when intrapartum fetal monitoring was conducted with external ultrasound transducer with or without simultaneous maternal heart rate recording, or with fetal scalp electrode

Neonatal outcome variables	Total	External US	External US+MHR	Internal FSE	External US vs external US+MHR OR (95% CI)	External US vs internal FSE OR (95% CI)	External US+MHR vs internal FSE OR (95% CI)
Number	213,798	81,559	62,268	69,971	143,827	151,530	132,239
UA acidosis							
Moderate acidemia							
UA pH 7.09 to 7.00	1937 (0.9)	1091 (1.3)	425 (0.7)	421 (0.6)	1.95 (1.75–2.19)	2.24 (2.00–2.51)	1.14 (0.99–1.30)
UA BE −10.0 to −11.9 (mmol/L)	1824 (0.9)	1014 (1.2)	401 (0.6)	409 (0.6)	1.94 (1.73–2.18)	2.14 (1.91–2.40)	1.01 (0.88–1.16)
Severe acidemia							
UA pH <7.00	627 (0.3)	358 (0.4)	139 (0.2)	130 (0.2)	1.97 (1.62–2.39)	2.37 (1.94–2.89)	1.20 (0.95–1.53)
UA BE ≤−12.0 (mmol/L)	551 (0.3)	327 (0.4)	123 (0.2)	101 (0.1)	2.03 (1.65–2.50)	2.78 (2.23–3.48)	1.37 (1.05–1.78)
1-min Apgar score <7	10,049 (4.7)	4020 (4.9)	2988 (4.8)	3041 (4.3)	1.03 (0.98–1.08)	1.14 (1.09–1.20)	1.11 (1.05–1.17)
5-min Apgar score <7	2567 (1.2)	1102 (1.4)	683 (1.1)	782 (1.1)	1.24 (1.12–1.36)	1.21 (1.11–1.33)	0.98 (0.89–1.09)
Intubation for resuscitation	855 (0.4)	369 (0.5)	231 (0.4)	255 (0.4)	1.22 (1.03–1.44)	1.24 (1.06–1.46)	1.02 (0.85–1.22)
NICU admission for asphyxia	4275 (2.1)	1876 (2.3)	1226 (2.0)	1173 (1.7)	1.17 (1.09–1.26)	1.38 (1.28–1.49)	1.18 (1.09–1.28)
Neonatal encephalopathy	223 (0.1)	114	59	50	1.48 (1.08–2.02)	1.96 (1.40–2.73)	1.32 (0.91–1.93)
Early neonatal death	27 (0.01)	16	5	6	2.44 (0.90–6.67)	2.29 (0.90–5.85)	0.94 (0.29–3.07)

Data are presented as absolute number (percentage).

BE, base excess; *CI*, confidence interval; *FSE*, fetal scalp electrode; *MHR*, maternal heart rate; *NICU*, neonatal intensive care unit; *OR*, odds ratio; *UA*, umbilical artery; *US*, ultrasound transducer.

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base deficit) ≤−12.0 mmol/L in UA blood, an Apgar score of <7 at 5 minutes, intubation for resuscitation at delivery, neonatal encephalopathy, early neonatal death (defined as death during the intrapartum CTG monitoring or death of a live-born infant within the first 28 days of life), and a composite neonatal asphyxia outcome, which was defined as the occurrence of ≥1 of the aforementioned outcomes. These neonatal outcomes manifest early, indicating that the fetus may have been compromised during intrapartum fetal surveillance.[1] They are associated with a risk of long-term neurologic adverse outcomes that could potentially be avoided by timely and optimal delivery.[8]

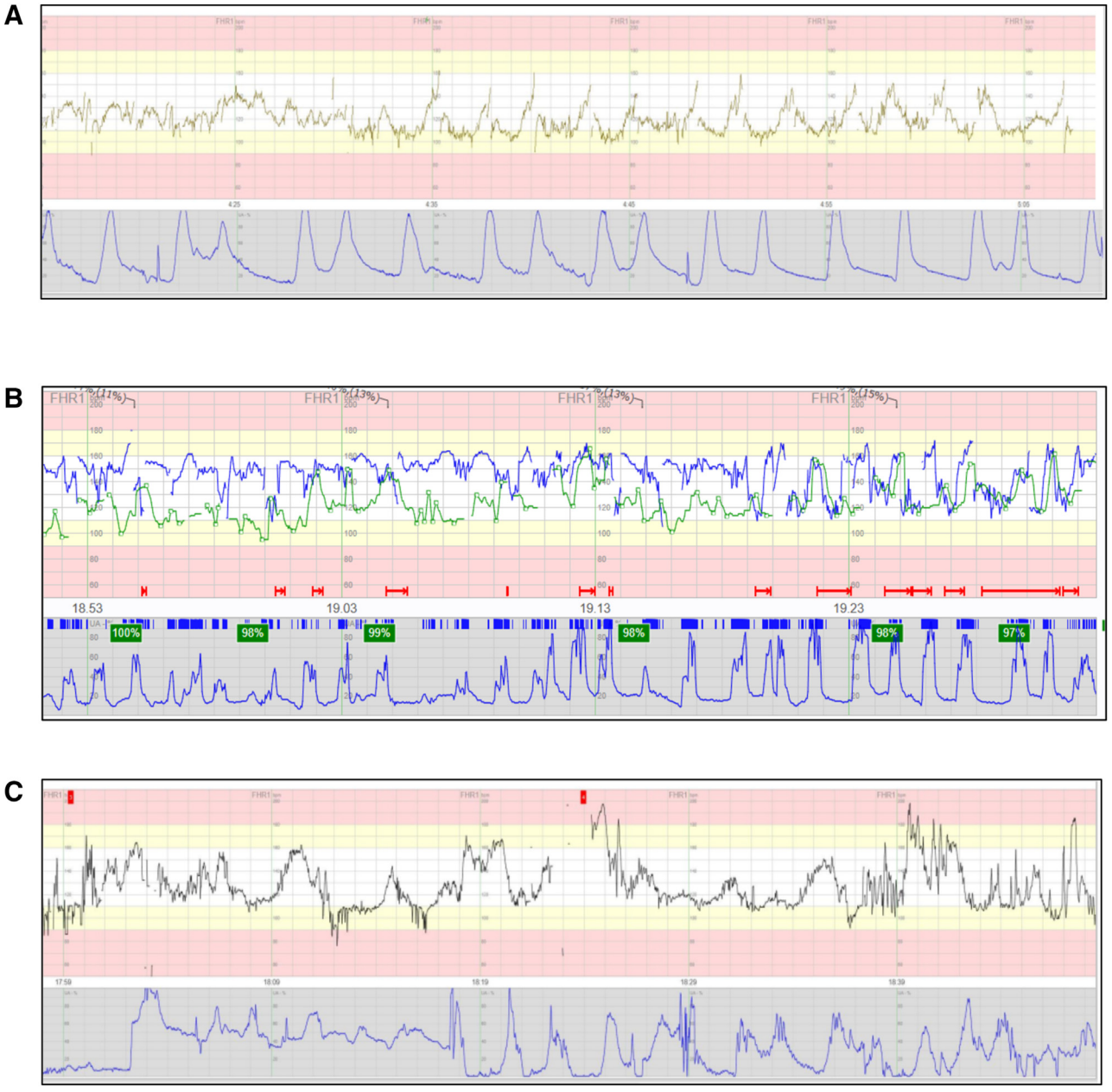
Neonatal secondary outcomes included the statistically defined UA pH limit (mean −2.0 SDs), ranging from 7.09 to 7.00, UA BE from −10.0 to −11.9 mmol/L, and NICU admission for asphyxia.

Data analysis

To analyze continuous variables, we used the analysis of variance, Kruskal–Wallis test, and Mann–Whitney *U* test. The Pearson chi-square and Fisher exact probability test were used for categorical variables. All tests were 2-sided. Values of $P<.05$ were considered statistically significant.

Logistic regression analysis was used to evaluate whether CTG registration method was associated with neonatal acidosis when the models included parity, obesity (prepregnancy body mass index, BMI ≥30.0 kg/m^2), diabetes mellitus (DM) and gestational DM (GDM), postterm (≥42 weeks) gestation, maternal age ≥35 years, preeclampsia, maternal fever ≥38.0°C, smoking, fetal sex, fetal growth restriction (FGR), small for gestational age (birthweight z-score <2.0 SD-units), large for gestational age (birthweight z-score >2.0 SD-units), meconium-stained amniotic fluid, induction of labor, oxytocin augmentation, epidural analgesia, other spontaneous cephalic nonvertex

FIGURE 1
Examples of 3 differently monitored cases with severe neonatal acidemia



The cardiotocograms were registered in the second stage of labor immediately before birth (paper speed, 1 min/cm). **A,** Signal ambiguity during external ultrasound transducer monitoring of FHR and resulting in unexpected early neonatal death. The tracing demonstrates a stable baseline heart rate of 120 bpm, replaced by repetitive accelerations of probable maternal origin during intermittent bearing-down efforts. UA pH of 6.66, UA BE of −21.3 mmol/L, and Apgar score of 0 at 1 and 5 minutes. Vitality of the fetus was confirmed during the evaluation of the fetal head presentation by ultrasound imaging 2 hours before birth. **B,** An FHR tracing (blue trace) with variable decelerations and simultaneous MHR recording (green trace). Generally, identifiable differences in the FHR and MHR exist until FHR decelerations appear with uterine contractions. An increase in MHR and a decrease in FHR are shown, with the latter indicating considerably the transition of the source into a maternal signal. Alarms, indicated by red segment marks, show the possible points when MHR may have been erroneously recorded as FHR. UA pH of 6.99, UA BE of −12.7 mmol/L, and Apgar score of 6 at 1 minute and 8 at 5 minutes. **C,** Unstable baseline FHR monitored via internal fetal scalp electrode. UA pH of 6.97, UA BE of −12.0 mmol/L, and Apgar score of 3 at 1 minute and 6 at 5 minutes. Neonatal encephalopathy.

BE, base excess; *FHR*, fetal heart rate; *MHR*, maternal heart rate; *UA*, umbilical artery.

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(occiput posterior presentation, asynclitism) delivery, year of delivery, and shoulder dystocia (Figures 2 and 3). The logistic regression analysis was performed using the RStudio, version 3.6.0 (RStudio, PBC, Boston, MA), and the odds ratios (ORs) and 95% confidence intervals (CIs) were estimated by fitting logistic regression models.

Ethics declaration

This study was approved by the Ethics Committee and the institutional review board of the Helsinki University Hospital, Finland (HUS/2100/2019, §152/17.7.2019; 29.9.2022, HUS/117/2022). No separate signed consent was required from the participants because of the research method (registry study).

Results

Primary outcome

Of the 328,320 deliveries during the study period, 213,798 (65.1%) met the selection criteria. US alone was the monitoring type in 81,559 (38.1%), US+MHR in 62,268 (29.1%), and FSE in 69,971 (32.7%) cases, respectively.

Figure 2 presents the primary fetal and neonatal asphyxia-related outcomes in women with US alone (N=81,559) compared with women with US+MHR or FSE (N=132,239). Newborns of women with US alone had a 1.7-fold risk of neonatal encephalopathy (OR, 1.70; 95% CI, 1.30−2.21), 2.2-fold risk of UA pH <7.00 (OR, 2.16; 95% CI, 1.84−2.53), 2.4-fold risk of UA BE ≤−12.0 mmol/L (OR, 2.37; 95% CI, 2.00−2.81) reflecting the metabolic component of a low pH, and a 1.2-fold risk of 5-minute Apgar score <7 (OR, 1.22; 95% CI, 1.13−1.32) compared with women with US+MHR or FSE. Moreover, in women with US alone, newborns had a 1.3-fold risk of composite neonatal asphyxia (UA pH <7.00 and/or UA BE ≤−12.0 mmol/L and/or 5-minute Apgar scores <7 and/or neonatal intubation for resuscitation and/or neonatal encephalopathy and/or early neonatal death) (OR, 1.31; 95% CI, 1.25−1.36) compared with the US+MHR or FSE groups. Logistic regression analysis revealed that adjustment for maternal, delivery-related, and fetal risk factors attenuated the association between external US alone and asphyxia-related neonatal outcomes only marginally (Figure 2). In contrast, US alone was also associated with the increased incidence of early neonatal death in the crude analyses (OR, 2.35; 95% CI, 1.09−5.08), but not after adjusting for confounders (Figure 2).

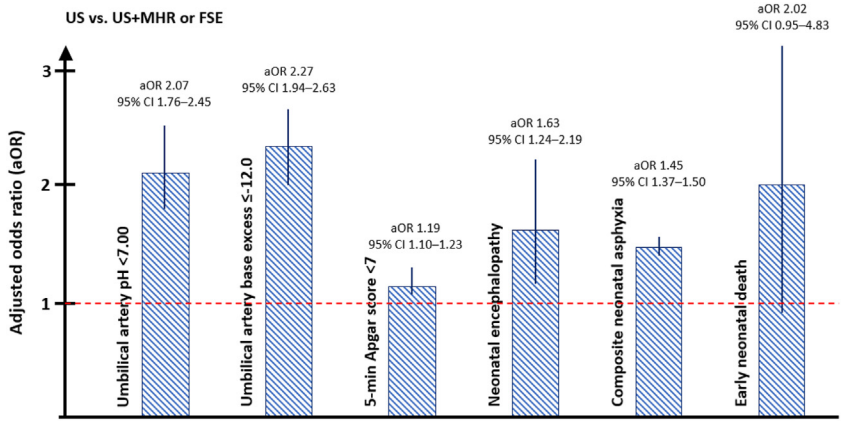
Table 2 shows that compared with US+MHR, risks of neonatal encephalopathy (1.5-fold), UA pH <7.00 (2.0-fold), and UA BE ≤−12.0 mmol/L (2.0-fold) were higher in fetuses of women with US alone. The latter method was also associated with a 1.2-fold risk of both neonatal intubation for resuscitation and with NICU admission for neonatal asphyxia compared with US+MHR (Table 2). Logistic regression analysis revealed that adjustment for maternal, delivery-related, and fetal risk factors had no effect on the association between external US alone and asphyxia-related neonatal outcomes (Figure 3; Supplemental Table).

Furthermore, fetuses with US alone had a 2.0-fold risk of neonatal encephalopathy, 2.4- to 2.8-fold risk of severe neonatal acidemia (UA pH <7.00 and/or UA BE ≤−12.0 mmol/L), and 1.2-fold risk of 5-minute Apgar scores <7 compared with those with FSE monitoring (Table 2). The finding was similar for those with neonatal intubation for resuscitation (1.2-fold) and NICU admission for neonatal asphyxia (1.4-fold) (Table 2).

No difference in the risk of neonatal encephalopathy, UA pH <7.00, 5-minute Apgar score <7, and intubation rates was found in those with US+MHR compared with FSE monitoring (Table 2). However, a 1.4-fold risk of severe neonatal metabolic acidemia (UA BE ≤−12.0 mmol/L) was observed in the US+MHR group compared with those monitored with FSE (Table 2).

FIGURE 2

Primary asphyxia-related outcome of newborns of women with US alone (N=81,559) compared with fetuses of women with US+MHR or FSE (N=132,239)



Adjusted for parity, obesity (prepregnancy body mass index ≥30.0 kg/m^2), diabetes mellitus and gestational diabetes mellitus, postterm (≥42 weeks) gestation, maternal age ≥35 years, preeclampsia, maternal fever ≥38.0°C, smoking, fetal sex, fetal growth restriction, small for gestational age (birthweight z-score <2.0 SD-units), large for gestational age (birthweight z-score >2.0 SD-units), meconium-stained amniotic fluid, induction of labor, oxytocin augmentation, epidural analgesia, other spontaneous cephalic (nonvertex) delivery, year of delivery, and shoulder dystocia. Composite neonatal asphyxia: UA pH <7.00 and/or UA base excess ≤−12.0 mmol/L and/or 5-minute Apgar scores <7 and/or neonatal intubation for resuscitation and/or neonatal encephalopathy and/or early neonatal death.

aOR, adjusted odds ratio; *CI*, confidence interval; *FSE*, fetal scalp electrode; *MHR*, maternal heart rate; *SD*, standard deviation; *UA*, umbilical artery; *US*, ultrasound transducer.

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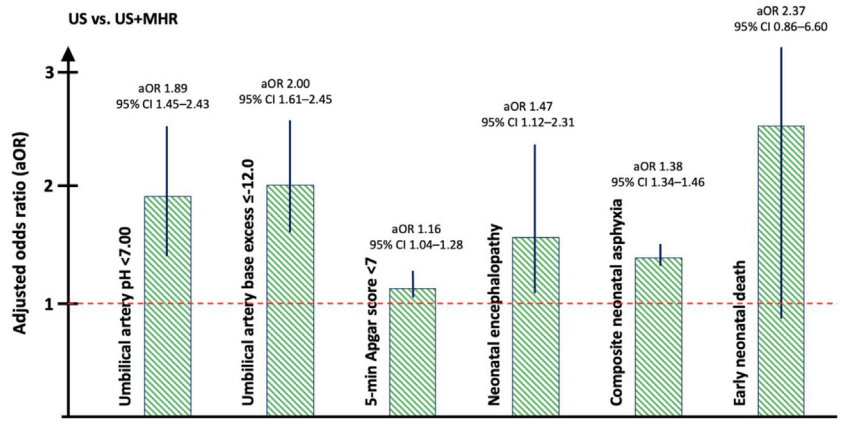
FIGURE 3
Primary asphyxia-related outcome of newborns of women with US alone (N=81,559) compared with newborns of women with US+MHR (N=62,268)



Adjusted for parity, obesity (prepregnancy body mass index ≥30.0 kg/m^2), diabetes mellitus and gestational diabetes mellitus, postterm (≥42 weeks) gestation, maternal age ≥35 years, preeclampsia, maternal fever ≥38.0°C, smoking, fetal sex, fetal growth restriction, small for gestational age (birthweight z-score <2.0 SD-units), large for gestational age (birthweight z-score >2.0 SD-units), meconium-stained amniotic fluid, induction of labor, oxytocin augmentation, epidural analgesia, other spontaneous cephalic (nonvertex) delivery, year of delivery, and shoulder dystocia. Composite neonatal asphyxia: UA pH <7.00 and/or UA base excess ≤−12.0 mmol/L and/or 5-minute Apgar scores <7 and/or neonatal intubation for resuscitation and/or neonatal encephalopathy and/or early neonatal death.

aOR, adjusted odds ratio; *CI*, confidence interval; *FSE*, fetal scalp electrode; *MHR*, maternal heart rate; *SD*, standard deviation; *UA*, umbilical artery; *US*, ultrasound transducer.

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Secondary outcomes

Newborns of mothers with US alone (N=81,559) had a 2.0- to 2.1-fold risk of moderate acidemia defined as UA pH of 7.09 to 7.00 (OR, 2.12; 95% CI, 1.93−2.32) and/or UA BE from −10.0 to −11.9 mmol/L (OR, 2.03; 95% CI, 1.85−2.22), and a 1.3-fold risk of NICU admission for neonatal asphyxia (OR, 1.30; 95% CI, 1.22−1.38) compared with women with US+MHR or FSE (N=132,239). Nonetheless, a 1.2-fold risk of NICU admission for neonatal asphyxia was observed also in women with US+MHR compared with those with FSE monitoring (Table 2).

Other findings

During the 18-year study period, the average annual incidence rate of neonatal encephalopathy was 0.10% (1 per 1000 births), with a decreasing trend from 0.12% to 0.09% (P=.003) over time. Furthermore, the incidence of newborns with severe acidemia (UA pH <7.00 and/or UA BE ≤−12.0 mmol/L) showed an average annual decline of 0.02 percentage units (95% CI, 0.01−0.04; P<.001), decreasing from 0.5% in 2005 to 0.2% in 2023. Concurrently, a temporal increase occurred in the use of both US+MHR (from 5.6% in 2005 to 43.1% in 2023; P<.001) and FSE (from 17.7% in 2005 to 46.8% in 2023; P<.001), respectively. Compared with US only or US+MHR, FSE was the monitoring method most frequently used in deliveries associated with increased risk of adverse fetal outcomes such as type 1 and 2 DM, GDM, induced labor, meconium-stained amniotic fluid, or suspected FGR (Table 1).

Of the 328,320 deliveries, we also evaluated cases where missing UA blood gas samples led to exclusion from the study population. Among these 4991 (0.2%) cases, there were no cases of early neonatal death. However, 1 case with neonatal encephalopathy was found in a newborn monitored by US alone, which would not have affected the results.

Lastly, newborns who were under continuous FHR monitoring during labor with Philips Avalon FM20/30 had a similar risk of neonatal encephalopathy as those monitored with Hewlett Packard HP Series 50A M1350/1A (OR, 0.86; 95% CI, 0.54−1.29).

Comment

Principal findings

The main finding of this study is that the external FHR monitoring method by CTG without concurrent maternal pulse recording is associated with neonatal acidemia and neonatal encephalopathy in spontaneous term cephalic deliveries, suggesting increased risk of fetal asphyxia in these deliveries. Our results indicate that concurrent external recording of FHR and MHR may improve the identification of fetal hypoxia compared with external recording of FHR alone. However, FSE, which was associated with the lowest incidence of asphyxia-related neonatal outcomes, was the most accurate method for assessing fetal status.

Results in the context of what is known

Continuous CTG monitoring during labor is widely used, but its potential for reducing severe adverse neonatal outcomes has been limited.[14,15] In this large 18-year birth cohort study, we retrospectively observed an association of the external FHR monitoring method with severe asphyxia-related neonatal outcomes. The prevalence of neonatal encephalopathy in the studied population was 1 per 1000 deliveries (0.1%), which is consistent with the national prevalence in Finland after term pregnancies (gestational age ≥37 weeks).[16]

To date, 56 years after the development of the first commercial CTG monitor in 1968, no gold-standard fetal monitoring method for prevention of neonatal encephalopathy has been introduced.[17] Some reports suggest an increased risk of intrapartum fetal death with FHR monitoring by US alone compared with other electronic fetal

monitoring methods.[18,19] However, it remains unconfirmed whether external FHR monitoring with simultaneous MHR recording can reduce the likelihood of severe asphyxia-related neonatal outcomes. Published data are mainly derived from studies comparing continuous CTG with intermittent auscultation,[20,21] whereas no studies have compared intrapartum fetal monitoring with an external US vs US+MHR.

In addition, no trials of US+MHR or FSE, including ours, have been sufficiently powered for detecting differences in early neonatal mortality rates. To yield 80% power to detect a 20% difference in the risk of early neonatal death (0.02% in the US-alone group vs 0.008% in the US+MHR or FSE group), a trial would need >305,000 deliveries, probably requiring multicenter collaboration.

Recently, Al Wattar et al[22] performed a meta-analysis including 33 trials (118,863 parturients) to evaluate the effectiveness of fetal surveillance methods in improving maternal and neonatal outcomes. None of the evaluated methods, including intermittent auscultation, traditional and computerized CTG, CTG with fetal scalp lactate and pH analysis, CTG with fetal pulse oximetry, and CTG with fetal ST-analysis, was associated with a reduced risk of neonatal acidemia, NICU admissions, low Apgar scores, or perinatal death.[22] However, the study did not analyze the effects of using CTG with simultaneous MHR recording.

The lower rates of neonatal encephalopathy and severe acidemia when using US+MHR or FSE instead of US alone in the present study could be explained by the following 2 factors, especially in the second stage of labor: (1) lower quality of external US signal when monitoring FHR, and (2) heart rate artifacts. Misinterpretation of MHR as FHR has been previously linked to severe outcomes[8] and is a frequent allegation in malpractice cases.[18] Unintentional acquisition of MHR has been documented in 55% to 90% of CTG recordings using US during the second stage of labor,[11,23,24] affirming that the ambiguities between FHR and MHR occur more frequently than indicated by reports focusing on extreme outcomes such as neonatal mortality. Neilson et al[25] highlighted instances of unexpected adverse neonatal outcomes in monitored labors where the FHR tracing appeared reassuring, yet subtle replacements from other heart rate signal sources, typically maternal, masked signs of fetal compromise. They estimated 5 avoidable cases with adverse perinatal outcome out of 10,000 deliveries because of a poor or ambiguous fetal monitoring signal.[25] Contrasting FHR and MHR patterns during labor, Sherman et al[26] observed that although mothers tend to display heart rate accelerations while pushing, fetuses more commonly exhibit heart rate decelerations, suggesting that repeated accelerations might originate from MHR, especially in scenarios of maternal tachycardia,[26] which is a relatively common finding in the second stage of labor.[27] According to Nurani et al,[28] the incidence of heart rate accelerations coinciding with uterine contractions was almost triple in fetuses monitored using an US compared with FSE.

A case series by Kiely et al[19] underscored a persistent concern regarding ambiguous signal quality in CTG-related fetal and neonatal deaths during a 10-year period, and identified 47 cases of perinatal mortality potentially linked to MHR artifacts during external FHR monitoring. In addition, Paquette et al[23] analyzed 1313 intrapartum CTG tracings displaying both FHR and MHR. Among them, 35 tracings (2.7%) were identified as having ≥1 episodes that could have led to an adverse outcome. In 33 cases, MHR obscured an abnormal FHR tracing, whereas in 2 cases, it masked a normal FHR, potentially leading to misinterpretation and unnecessary intervention based on the tracing.[23] However, FSE is not free from registration uncertainties. Case reports have presented rare cases where a stillborn fetus transmitted an MHR signal through an FSE.[29] Overall, excluding misinterpretation in these cases, the importance of concomitant MHR recording is essential.

In the present study, the association of US alone with asphyxia-related outcomes persisted after we adjusted for other factors known to be related to the risk of neonatal encephalopathy, cord blood acidemia, low Apgar scores, and NICU admission. Notably, FSE proved to be the safest fetal monitoring method, although the FSE-monitored group was overrepresented in many cases at higher risk of perinatal complications, such as maternal hyperglycemic disorders, meconium-stained amniotic fluid, and small-for-date or growth-restricted fetuses, compared with the US and US+MHR monitoring groups. This strengthens the reliability of our findings. However, the better outcome in the FSE arm may also indicate improved CTG interpretation in the high-risk cases (ie, the "Hawthorne effect").[30]

Clinical implications

This definitive study was conducted to explore the safety of different CTG monitoring practices in clinical settings. In North America and Europe, external US monitoring is extensively used for intrapartum fetal surveillance, although concerns exist about its reliability and accuracy.[31] Our findings define that external FHR monitoring without a safeguard of simultaneous MHR recording is associated with increased risk of intrapartum fetal hypoxia, which can be detected as asphyxia-related fetal and neonatal outcomes at birth. Understanding this gap may improve the safety of intrapartum fetal surveillance and enable the clinician to plan adequate monitoring methods.

Research implications

To evaluate the potential long-term effects, future research should include follow-up of offspring who had different types of electronic FHR monitoring during labor in term pregnancies. High-quality multicenter trials, both in high- and low-income settings, are needed to confirm the generalizability of the present study. The possibilities of the use of artificial intelligence and computer analysis for the reliable differentiation between external FHR and MHR signals during labor should be scrutinized in upcoming studies. Furthermore, associations of US alone vs other CTG monitoring methods with important maternal outcomes such

as cesarean delivery, assisted vaginal delivery, and anal sphincter injury should be considered in future trials.

Strengths and limitations

The strengths of this study include the large number of study participants, well-defined criteria for different neonatal asphyxia-related outcomes, and the use of detailed, complete, and high-quality maternal–neonatal and CTG databases. In particular, same doctors and midwives rotate in the HUS maternity hospitals, which potentially strengthens the uniformity of treatment and the consistency of practices. Nevertheless, there are several limitations to our study. First, this study had a retrospective design. Second, we studied parturients in spontaneous term singleton labor, and our results may not apply to other populations, such as those with preterm deliveries, twin deliveries, and forceps or vacuum-assisted deliveries, all of which are known risk factors for heart rate signal ambiguity. Third, fetal blood sampling (FBS) for evaluating fetal acidosis was a relatively common intervention in the cohort, potentially influencing intrapartum care and contributing to differences between the groups. Nonetheless, FBS was not used as a factor in the current study. Finally, it may be difficult to extrapolate our results to the cases in which traditional CTG monitoring of FHR is enhanced by ST-analysis or other computerized method. Multivariate modeling, however, indicated that the type of FHR monitoring was an independent predictor of the severe metabolic UA blood acidemia and neonatal encephalopathy.

Conclusions

This large study showed a considerable benefit of the concurrent use of intrapartum MHR recording in reducing neonatal encephalopathy, severe umbilical cord artery blood acidemia, and a composite of adverse neonatal outcomes in a Finnish cohort of parturients with conventional external electronic FHR monitoring. The fact that hypoxia-related factors are more common in deliveries monitored solely by external US than in those monitored by FSE or concurrent fetal–maternal heart rate recording indicates that the current methods and strategies for intrapartum fetal surveillance are not optimal. Given the importance of timely recognition of abnormal FHR patterns, we suggest that either simultaneous MHR recording or FSE be routinely used during labor in term pregnancies. ■

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Data from this study are available on reasonable request from the corresponding author. The data are not publicly available because of privacy and ethical restrictions.

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SUPPLEMENTAL TABLE

Relationship between the use of external ultrasound transducer monitoring of fetal heart rate without simultaneous maternal heart rate recording and asphyxia-related neonatal outcomes after adjusting for maternal, delivery-related, and neonatal variables

	Severe acidemia UA pH <7.00 and/or UA BE ≤−12.0		Neonatal encephalopathy	
Confounding factors	Odds ratio (95% CI)	*P* value	Odds ratio (95% CI)	*P* value
Maternal age ≥35 y	0.915 (0.788–1.062)	.263	1.096 (0.838–1.433)	.502
Obesity, prepregnancy BMI ≥30.0 (kg/m^2)	0.963 (0.623–1.488)	.863	0.953 (0.717–1.267)	.741
Nulliparous	0.954 (0.811–1.122)	.570	1.059 (0.804–1.395)	.682
Smoking	1.143 (0.681–1.917)	.619	0.969 (0.742–1.266)	.817
Type 1 or 2 diabetes mellitus	3.009 (0.660–13.716)	.154	2.661 (0.585–12.102)	.205
Gestational diabetes mellitus	1.271 (0.825–1.960)	.277	1.154 (0.881–1.512)	.298
Preeclampsia	1.212 (0.269–5.395)	.806	0.689 (0.178–2.669)	.590
Fever (≥38.0°C) during delivery	1.715 (0.793–4.043)	.237	1.373 (0.719–3.626)	.338
Year of delivery	1.258 (0.922–1.584)	.269	1.135 (0.874–1.673)	.238
Induction of labor	1.235 (0.859–1.777)	.254	1.161 (0.885–1.524)	.281
Oxytocin augmentation	1.173 (0.957–1.445)	.102	1.226 (0.934–1.609)	.141
Meconium-stained amniotic fluid	1.186 (0.773–1.821)	.422	0.851 (0.639–1.134)	.273
Epidural analgesia	1.020 (0.824–1.262)	.851	0.943 (0.717–1.240)	.674
Other spontaneous cephalic (nonvertex) delivery	0.709 (0.673–3.294)	.248	0.687 (0.631–3.091)	.203
Shoulder dystocia	0.672 (0.093–24.138)	.370	0.625 (0.116–21.820)	.389
Female sex	1.110 (0.892–1.372)	.355	0.874 (0.663–1.151)	.338
FGR	1.667 (0.480–5.772)	.421	0.718 (0.220–7.341)	.583
SGA (birthweight z-score <2.0 SD-units)	2.168 (0.485–9.689)	.312	0.972 (0.382–8.473)	.652
LGA (birthweight z-score >2.0 SD-units)	2.954 (0.705–12.375)	.138	2.068 (0.786–5.440)	.150
Postterm (≥42.0 wk)	2.558 (0.811–8.062)	.109	1.977 (0.717–5.457)	.186
Bivariate models (N=143,827)	1.943 (1.632–2.384)	<.001	1.472 (1.120–2.312)	<.001
Final model (N=143,827)	1.812 (1.461–2.272)	<.001	1.394 (1.054–2.393)	<.001

Logistic regression models include ultrasound transducer (US) alone (N=81,559) and US+maternal heart rate (N=62,268) groups. Bivariate models: adjusted for oxytocin augmentation, LGA (birthweight z-score >2.0 SD-units), and postterm (≥42.0 weeks) pregnancy. Final model: adjusted for parity, obesity (prepregnancy BMI ≥30.0 kg/m^2), diabetes mellitus and gestational diabetes mellitus, postterm (≥42 weeks) gestation, maternal age ≥35 years, preeclampsia, maternal fever ≥38.0°C, smoking, fetal sex, FGR, SGA (birthweight z-score <2.0 SD-units), LGA (birthweight z-score >2.0 SD-units), meconium-stained amniotic fluid, induction of labor, oxytocin augmentation, epidural analgesia, other spontaneous cephalic (nonvertex) delivery, year of delivery, and shoulder dystocia.

BMI, body mass index; *CI*, confidence interval; *FGR*, fetal growth restriction; *LGA*, large for gestational age; *SGA*, small for gestational age.

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