International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Rana IS et al. Int J Reprod Contracept Obstet Gynecol. 2026 Dec;15(12):4878-4885
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20264402

Original Research Article

Association of cardiotocography diagnosis of intrapartum fetal distress with immediate postpartum acidaemia in the fetal umbilical artery

Ishita Samarpal Rana*, H. V. Ravi Gowda

Department of Obstetrics and Gynaecology, J.J.M. Medical College, Davangere, Karnataka, India

Received: 26 September 2026
Accepted: 02 November 2026

***Correspondence:**
Dr. Ishita Samarpal Rana,
E-mail: ranaishitasamarpal@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Intrapartum hypoxia is a preventable cause of neonatal morbidity and mortality. Cardiotocography (CTG) is the commonest method of intrapartum surveillance, but its low specificity contributes to operative delivery for suspected fetal distress. Umbilical artery blood gas analysis provides an objective measure of fetal acid-base status. The objectives were to determine the incidence of cord blood acidaemia after caesarean section (CS) for non-reassuring CTG and its association with neonatal outcomes.
Methods: A prospective cross-sectional study of 60 women with singleton, term (≥37 weeks), cephalic pregnancies delivered by CS for non-reassuring CTG at three teaching hospitals in India, between April 2023 and September 2024. Traces were classified according to NICE clinical guideline CG190. Umbilical artery blood sampled at delivery was analysed on a Radiometer ABL800 Basic analyser; pH <7.2 defined acidaemia. Associations were tested by chi-square test, with $p<0.05$ significant.
Results: Mean maternal age was 26.93±4.83 years, mean gestational age 38.65±1.29 weeks; 63.3% were primigravidae. Acidaemia was present in 11 neonates (18.3%). CTG was pathological in 36 (60.0%), suspicious in 22 (36.7%) and indicated urgent intervention in 2 (3.3%). Acidaemia was significantly associated with CTG category ($p<0.01$), deceleration pattern ($p<0.001$) and the need for resuscitation ($p<0.001$), but not with baseline variability ($p=0.410$). Ten neonates required NICU admission; there were no perinatal deaths.
Conclusions: Acidaemia was confirmed in fewer than one in five neonates delivered by CS for non-reassuring CTG. Abnormal decelerations were the fetal heart rate feature most closely linked to acidaemia and may support more discriminating, better-timed intervention.

Keywords: Acidaemia, Birth asphyxia, Caesarean section, Cardiotocography, Fetal distress, Umbilical cord blood gas analysis

INTRODUCTION

The neonatal period carries the highest daily risk of death of any period in childhood, with mortality in the first four weeks of life approximately 30 times that of the post neonatal period.[1] Despite advances in perinatal, obstetric and neonatal care, birth-related injury remains one of the leading contributors to neonatal mortality worldwide, alongside complications of prematurity and infection.[2] Globally, around four million infants die from asphyxia each year, accounting for roughly 23% of neonatal deaths.[3] The burden is unequally distributed, with an estimated incidence of 1 per 1,000 live births in high income settings compared with 5-10 per 1,000 in low and middle income countries.[4]

Perinatal asphyxia results from impaired blood flow or gas exchange between mother and fetus immediately before, during or after delivery.[5] Impaired placental or pulmonary gas exchange produces progressive hypoxaemia and hypercapnia; when compensatory mechanisms are exhausted, anaerobic metabolism and lactic acidosis follow. The consequences include hypoxic-ischaemic

encephalopathy, seizures, intraventricular haemorrhage, cerebral palsy and developmental delay.[6,7] A substantial proportion of intrapartum hypoxia is preventable, and the principal aim of intrapartum fetal monitoring is to detect hypoxia early enough to avert acidaemia and the cellular injury that accompanies it.[8]

CTG, which records fetal heart rate (FHR) and uterine activity simultaneously, is the most widely applied tool for intrapartum fetal surveillance. It is non-invasive, has no contraindications and provides a permanent record of fetal status. Its widespread adoption has, however, been accompanied by a rise in CS performed for suspected fetal distress without a corresponding reduction in the incidence of cerebral palsy. This happens mainly because CTG is not very specific in detecting true fetal distress.[9,10]

The Apgar score remains the most commonly used bedside assessment of the newborn, but it is influenced by prematurity, meconium-stained liquor, maternal analgesia and observer subjectivity, and may be abnormal in the absence of asphyxia.[9,11] Umbilical artery blood gas analysis offers a more objective index of intrapartum oxygenation: severe interruption of fetal oxygen supply causes tissue hypoxia and metabolic acidosis that is reflected in the pH of blood drawn from the umbilical artery immediately after birth.[4,12]

Because CS carries higher maternal morbidity and mortality than vaginal birth, and because the diagnosis of fetal distress is a major and growing indication for CS in resource-limited settings, establishing how closely abnormal CTG findings correspond to objectively confirmed acidaemia is of direct clinical relevance. The present study was therefore undertaken to determine the incidence of acidaemia in umbilical cord blood pH among neonates delivered by CS for non-reassuring CTG, and to examine the association between cord blood acidaemia and early neonatal outcomes in these deliveries.

METHODS

Study design and setting

This was a prospective, cross-sectional, descriptive study conducted in the Department of Obstetrics and Gynaecology of three teaching hospitals in India. Data were collected over 18 months, from April 2023 to September 2024.

Participants

Women admitted to the labour wards for confinement or further management were screened for eligibility. Inclusion criteria were: antenatal women with a term pregnancy (≥37 completed weeks of gestation); singleton pregnancy; and cephalic presentation. Exclusion criteria were: gestational age below 37 weeks; abnormal biophysical profile or abnormal fetal Doppler on ultrasonography; malpresentation; and CS performed for any indication other than fetal distress. Eligible women were recruited by simple random sampling from the daily admission lists of the three participating hospitals.

Sample size

The sample size was calculated using the formula $n=Z^2\alpha pq/d^2$, where $Z\alpha=1.96$ at the 5% level of significance (95% confidence), p=8.5% (prevalence taken from a previous report), q=91.5%, and d=7% allowable error, with a study power of 80%. This yielded a required sample of 60 participants.[7]

Data collection

After written informed consent, a detailed obstetric history was recorded and a general physical and systemic examination (cardiovascular, respiratory, central nervous system, per abdomen and per vaginum) was performed. Maternal age, weight, parity, gestational age, blood group, comorbid conditions and current medications were documented on a pre-designed, pre-tested proforma. Intrapartum details recorded included whether labour was induced and whether oxytocin was used for augmentation. Neonatal details recorded included sex, blood group, time of birth, gestational age at birth, presence and grade of meconium staining of liquor, and cord loops around the neck.

All clinical and biochemical assessments were performed by a single investigator to eliminate inter-observer variability. Prior to commencement of the study, the investigator was calibrated in the department under the supervision of the guide by repeating examinations on a subset of randomly selected participants on different days. Intra-observer reliability, assessed by Cohen's kappa, was 0.88 for anthropometric measurements and 0.92 for systemic examination. A separately trained recorder documented all findings on the proforma at the time of examination.

CTG

CTG was recorded using a BPL Diva fetal monitor with an external transducer at a paper speed of 1 cm/min. Traces were interpreted and classified according to NICE clinical guideline CG190.[10] Baseline FHR, baseline variability and decelerations were each categorised individually as reassuring, non-reassuring or abnormal, and the trace as a whole was categorised as normal, suspicious, pathological, or indicating the need for urgent intervention. A trace was classified as suspicious when a single feature was non-reassuring and all others reassuring, and as pathological when two or more features were non-reassuring or any single feature was abnormal.

Delivery and umbilical cord blood gas analysis

Women with a non-reassuring trace underwent CS under spinal or combined spinal-epidural anaesthesia. The mean

interval from diagnosis to delivery was 35 minutes. Immediately after delivery, arterial cord blood was drawn from a doubly clamped segment of umbilical cord into a pre-heparinised 2 ml syringe and transported through a pneumatic tube system, reaching the laboratory within 5 minutes of collection. Blood gas analysis was performed on a Radiometer ABL800 Basic analyser, with results available within a mean of 15 minutes. In accordance with institutional protocol, an umbilical artery pH below 7.2 was taken to indicate acidaemia.

Neonatal assessment

Immediately at delivery, respiratory effort, heart rate, colour, muscle tone and reflex irritability were assessed and Apgar scores assigned at 1 and 5 minutes. Neonatal outcomes recorded were the need for active resuscitation, bag-and-mask ventilation, chest compressions and intubation; admission to the neonatal intensive care unit (NICU); the mode of respiratory support required in the NICU; NICU stay exceeding 24 hours; and total duration of hospital stay.

Statistical analysis

Data were entered in Microsoft Excel and analysed using SPSS version 25.0. Qualitative variables are expressed as frequencies and percentages and quantitative variables as mean±standard deviation with range. The chi-square test was used to test the statistical significance of associations between categorical variables and acidaemia, and Student's t-test to compare mean duration of hospital stay between acidaemic and non-acidaemic neonates. A p value below 0.05 was considered statistically significant.

Ethical approval

The study protocol was approved by the Institutional Ethics Committee (approval number JJMMC/IEC-Syc-150-2023). Written informed consent was obtained from every participant before enrolment. The study was conducted in accordance with the Declaration of Helsinki.

RESULTS

Sixty women meeting the eligibility criteria were enrolled and all completed the study; no participant was lost to follow-up. Baseline maternal and neonatal characteristics are shown in Table 1. Mean maternal age was 26.93±4.83 years (range 19-37), mean maternal weight 69.45±12.18 kg (range 50-89) and mean gestational age at delivery 38.65±1.29 weeks (range 37-42). The mean Apgar score was 7.58±1.58 (range 4-10) and the mean duration of hospital stay 5.10±1.61 days (range 2-14).

Obstetric and clinical characteristics are summarised in Table 2. Thirty-eight women (63.3%) were primigravidae and 22 (36.7%) were multigravidae. Forty-one women (68.3%) had no comorbid condition; gestational hypertension and anaemia were the commonest comorbidities, each affecting 5 women (8.3%), followed by gestational diabetes mellitus in 4 (6.7%), hypothyroidism in 3 (5.0%), and severe pre-eclampsia and chronic hypertension in 1 each (1.7%). Forty-nine women (81.7%) were on no medication, 7 (11.7%) on antihypertensives, 3 (5.0%) on levothyroxine and 1 (1.7%) on an oral hypoglycaemic agent. B positive was the commonest blood group (22, 36.7%), followed by A positive (15, 25.0%).

Labour was induced in 37 women (61.7%) and oxytocin was used for augmentation in 32 (53.3%). Of the 60 neonates, 33 (55.0%) were female and 27 (45.0%) male. Liquor was clear in 45 cases (75.0%), thinly meconium-stained in 9 (15.0%) and thickly meconium-stained in 6 (10.0%). Cord loops around the neck were present in 9 neonates (15.0%).

CTG findings are presented in Table 3. The trace was pathological in 36 cases (60.0%), suspicious in 22 (36.7%), and indicated the need for urgent intervention in 2 (3.3%); the latter two cases were a cord prolapse and a grade II abruptio placentae. Considering the individual CTG features, baseline FHR was reassuring in 31 (51.7%), non-reassuring in 23 (38.3%) and abnormal in 6 (10.0%); baseline variability was reassuring in 28 (46.7%), non-reassuring in 27 (45.0%) and abnormal in 5 (8.3%); and decelerations were reassuring in 34 (56.7%), non-reassuring in 17 (28.3%) and abnormal in 9 (15.0%).

Umbilical artery pH was below 7.2 in 11 neonates, giving an incidence of acidaemia of 18.3% among neonates delivered by CS for non-reassuring CTG; the remaining 49 (81.7%) were non-acidaemic. Neonatal outcomes are shown in Table 4. Ten neonates (16.7%) required active resuscitation, 8 (13.3%) bag-and-mask ventilation, 4 (6.7%) chest compressions and 2 (3.3%) intubation. Ten neonates were admitted to the NICU, of whom 4 (6.7% of the cohort) required headbox oxygen, 2 (3.3%) bubble CPAP, 2 (3.3%) nasal cannula oxygen and 2 (3.3%) mechanical ventilation. Eight neonates (13.3%) required NICU observation beyond 24 hours. No neonate developed hypoxic-ischaemic encephalopathy and there were no perinatal deaths.

The association between overall CTG category and acidaemia is shown in Table 5. Acidaemia was present in 1 of 22 neonates with a suspicious trace (4.5%), 8 of 36 with a pathological trace (22.2%) and both neonates in whom the trace indicated a need for urgent intervention (100%). This association was statistically significant (χ^2=12.006, p=0.002).

Associations between the individual CTG features and acidaemia are shown in Table 6. Acidaemia occurred in 6 of 31 neonates with a reassuring baseline FHR (19.4%), 3 of 23 with a non-reassuring baseline (13.0%) and 2 of 6 with an abnormal baseline (33.3%). With respect to baseline variability, acidaemia was present in 7 of 28 neonates with reassuring variability (25.0%), 3 of 27 with

non-reassuring variability (11.1%) and 1 of 5 with abnormal variability (20.0%); this association was not statistically significant (χ^2=1.781, p=0.410). Deceleration pattern showed the strongest gradient, acidaemia occurred in 3 of 34 neonates with reassuring decelerations (8.8%), 2 of 17 with non-reassuring decelerations (11.8%) and 6 of 9 with abnormal decelerations (66.7%), a highly significant association (χ^2=16.586, p<0.001). The need for resuscitation was strongly associated with acidaemia (Table 7). Seven of the 11 acidaemic neonates (63.6%) required resuscitation, compared with 3 of the 49 non-acidaemic neonates (6.1%); expressed the other way, 7 of the 10 resuscitated neonates (70.0%) were acidaemic against 4 of the 50 not requiring resuscitation (8.0%) (χ^2=21.395, p<0.001). Among the acidaemic neonates who required respiratory support in the NICU, 1 received headbox oxygen, 2 bubble CPAP, 2 nasal cannula oxygen and 2 mechanical ventilation; both neonates requiring mechanical ventilation and both requiring bubble CPAP were acidaemic.

Table 1: Baseline maternal and neonatal characteristics, (n=60).

Variables	N	Mean±SD	Minimum	Maximum
Maternal age (in years)	60	26.93±4.83	19	37
Maternal weight (kg)	60	69.45±12.18	50	89
Gestational age (weeks)	60	38.65±1.29	37	42
Apgar score	60	7.58±1.58	4.00	10.00
Duration of hospital stay (in days)	60	5.10±1.61	2.00	14.00

Table 2: Obstetric and clinical characteristics of the study population, (n=60).

Characteristics	N	Percentage (%)
Parity		
Primigravida	38	63.3
Gravida 2	20	33.3
Gravida 3	2	3.3
Comorbid conditions		
None	41	68.3
Gestational hypertension	5	8.3
Anaemia	5	8.3
Gestational diabetes mellitus	4	6.7
Hypothyroidism	3	5.0
Severe pre-eclampsia	1	1.7
Chronic hypertension	1	1.7
Medications		
None	49	81.7
Antihypertensives	7	11.7
Levothyroxine	3	5.0
Oral hypoglycaemics	1	1.7
Blood group		
B positive	22	36.7
A positive	15	25.0
O positive	11	18.3
AB positive	9	15.0
O negative	2	3.3
B negative	1	1.7
Intrapartum and neonatal variables		
Induction of labour performed	37	61.7
Oxytocin used for augmentation	32	53.3
Female neonate	33	55.0
Male neonate	27	45.0
Meconium-stained liquor: absent	45	75.0
Meconium-stained liquor: thin	9	15.0
Meconium-stained liquor: thick	6	10.0
Cord loops around the neck	9	15.0

Table 3: Cardiotocographic findings, (n=60).

CTG finding	N	Percentage (%)
Overall CTG category (NICE CG190)		
Suspicious	22	36.7
Pathological	36	60.0
Need for urgent intervention	2	3.3
Baseline fetal heart rate		
Reassuring	31	51.7
Non-reassuring	23	38.3
Abnormal	6	10.0
Baseline variability		
Reassuring	28	46.7
Non-reassuring	27	45.0
Abnormal	5	8.3
Decelerations		
Reassuring	34	56.7
Non-reassuring	17	28.3
Abnormal	9	15.0

*CTG: cardiotocography; NICE: National Institute for Health and Care Excellence.

Table 4: Immediate neonatal outcomes, (n=60).

Variables	N	Percentage (%)
Outcome		
Umbilical artery pH <7.2 (acidaemia)	11	18.3
Active resuscitation required	10	16.7
Bag-and-mask ventilation	8	13.3
Chest compressions	4	6.7
Intubation	2	3.3
NICU admission	10	16.7
NICU stay >24 hours	8	13.3
Mode of respiratory support in NICU		
Headbox oxygen	4	6.7
Bubble CPAP	2	3.3
Nasal cannula	2	3.3
Mechanical ventilation	2	3.3
Hypoxic-ischaemic encephalopathy	0	0.0
Perinatal death	0	0.0

*NICU: neonatal intensive care unit; CPAP: continuous positive airway pressure.

Table 5: Association between overall CTG category and neonatal acidaemia.

CTG category	Acidaemia present, (n=11)	Acidaemia absent, (n=49)	χ^2 value	P value
Suspicious (n=22)	1 (4.5)	21 (95.5)	12.006	0.002*
Pathological (n=36)	8 (22.2)	28 (77.8)		
Need for urgent intervention (n=2)	2 (100.0)	0 (0.0)		

*Values are n (%) of the row total. Statistically significant ($p<0.05$); chi-square test.

Table 6: Association between individual CTG features and neonatal acidaemia.

CTG features	Acidaemia present (n=11)	Acidaemia absent (n=49)	χ^2 value	P value
Baseline fetal heart rate				
Reassuring (n=31)	6 (19.4)	25 (80.6)	12.006	0.002*
Non-reassuring (n=23)	3 (13.0)	20 (87.0)		
Abnormal (n=6)	2 (33.3)	4 (66.7)		

Continued.

CTG features	Acidaemia present (n=11)	Acidaemia absent (n=49)	χ^2 value	P value
Baseline variability				
Reassuring (n=28)	7 (25.0)	21 (75.0)	1.781	0.410
Non-reassuring (n=27)	3 (11.1)	24 (88.9)		
Abnormal (n=5)	1 (20.0)	4 (80.0)		
Decelerations				
Reassuring (n=34)	3 (8.8)	31 (91.2)	16.586	<0.001*
Non-reassuring (n=17)	2 (11.8)	15 (88.2)		
Abnormal (n=9)	6 (66.7)	3 (33.3)		

*Values are n (%) of the row total. Statistically significant (p<0.05); chi-square test.

Table 7: Association between the need for neonatal resuscitation and acidaemia.

Resuscitations	Acidaemia present, (n=11)	Acidaemia absent, (n=49)	χ^2 value	P value
Required, (n=10)	7 (70.0)	3 (30.0)	21.395	<0.001*
Not required, (n=50)	4 (8.0)	46 (92.0)		

*Values are n (%) of the row total. Statistically significant (p<0.05); chi-square test.

DISCUSSION

In our study out of 60 term singleton pregnancies delivered by CS for a non-reassuring CTG, objectively confirmed acidaemia was present in only 18.3% of neonates. Put differently, more than four in five CSs performed for suspected intrapartum fetal distress delivered a neonate whose umbilical artery pH was normal. This gap between the CTG diagnosis of fetal distress and the biochemical confirmation of hypoxia is the central finding of the study and is consistent with many studies showing the limited positive predictive value of intrapartum electronic fetal monitoring.[13,14]

Our study showed demographic profile of a mean maternal age of 26.93 years and a predominance of primigravidae (63.3%) which is comparable with that reported by Ranjana et al and Khatun et al in similar Indian and South Asian tertiary care populations.[15,16] Labour was induced in 61.7% of women and oxytocin used in 53.3%, a proportion that reinforces the importance of continuous rather than intermittent monitoring in induced and augmented labour, since increased uterine activity raises the risk of fetal hypoxia when it is not closely observed.[17]

A statistically significant association was demonstrated between the overall NICE category of the trace and neonatal acidaemia (p=0.002), with the proportion of acidaemic neonates rising from 4.5% in the suspicious group to 22.2% in the pathological group and 100% in the two cases requiring urgent intervention. This gradient accords with the findings of Aboulghar et al who reported that pathological traces carry a significantly higher risk of acidosis than suspicious traces, and with the observations of Gupta et al and Kanagal et al in comparable prospective cohorts.[13,18,19] The absence of hypoxic-ischaemic encephalopathy and of any perinatal death in the present series suggests that, whatever its specificity, timely action on an abnormal trace remains clinically protective.

When the individual components of the trace were examined separately, the deceleration pattern proved to be the single most discriminating feature, two-thirds of neonates with abnormal decelerations were acidaemic, against fewer than one in ten of those with reassuring decelerations (p<0.001). This finding is in keeping with reports linking late and severe variable decelerations, and the total deceleration area, to cord blood acid-base status.[20,21] By contrast, baseline variability showed no significant association with acidaemia (p=0.410), and reassuring variability was in fact recorded in seven of the eleven acidaemic neonates. Reduced variability is conventionally regarded as a marker of evolving hypoxia, and its lack of discriminating value here is most likely explained by the small number of neonates with abnormal variability (n=5) rather than by any true absence of association.

Acidaemia had clear clinical consequences. The need for active resuscitation was strongly associated with a low cord pH (p<0.001), and both neonates who required mechanical ventilation were acidaemic. Cord blood gas analysis therefore retains value not as a substitute for CTG but as a complementary, objective measure that identifies the subgroup of neonates in whom the CTG abnormality reflected genuine hypoxic compromise. Several authors have made the same argument for routine paired cord gas sampling after operative delivery for fetal distress, both for immediate neonatal care and for audit of intrapartum decision-making.[22-24]

A methodological point worth noting is the definition of acidaemia. The threshold of pH <7.2 applied here follows institutional protocol and is used in a number of published series, but it is more permissive than the values of <7.1 or <7.0 adopted elsewhere, and considerably more permissive than the pH <7.0 with a base deficit ≥12 mmol/l required to define an acute intrapartum hypoxic event of sufficient severity to cause encephalopathy.[25,26] The

incidence of 18.3% reported here is accordingly not directly comparable with series using stricter thresholds, and would be expected to be lower if a lower pH cut-off were applied. Fetuses may tolerate pH values below 7.0 during labour without neurological sequelae, and the clinical significance of a pH between 7.1 and 7.2 in an otherwise vigorous neonate remains uncertain.

Strengths

All CTG traces were interpreted by a single trained observer, which removed inter-observer variability. Blood gas analysis was performed in a single standardised laboratory on a single analyser with a short and consistent sample transport time, minimising pre-analytical error. CTG monitoring and round-the-clock facilities for emergency intervention were available to all participants, and there were no perinatal deaths.

Limitations

The sample size was small and the study was conducted at three teaching hospitals within a single region, so the findings should be extrapolated with caution. Because only women delivered by CS for non-reassuring CTG were included, the study contains no group with a normal trace; sensitivity, specificity and predictive values of CTG could therefore not be calculated, and the study does not address the diagnostic accuracy of CTG. Only umbilical artery pH was recorded; base deficit, lactate and paired venous samples were not analysed, so respiratory and metabolic acidosis could not be distinguished. Finally, follow-up was limited to the immediate neonatal period, and the study was not designed to assess CS rates or long-term neurodevelopmental outcomes such as cerebral palsy.

CONCLUSION

Among neonates delivered by CS for a non-reassuring CTG, acidaemia defined by an umbilical artery pH below 7.2 was confirmed in 18.3%. Pathological traces were significantly associated with acidaemia compared with suspicious traces, and abnormal decelerations emerged as the single fetal heart rate feature most closely linked to a low cord pH. Acidaemia was associated with the need for resuscitation and for respiratory support. Umbilical artery blood gas analysis is a useful complement to CTG for identifying neonates with genuine hypoxic compromise after operative delivery for suspected fetal distress.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Sankar MJ, Neogi SB, Sharma J, Chauhan M, Srivastava R, Prabhakar PK, et al. State of newborn health in India. J Perinatol. 2016;36(s3):S3-8.
2. Liu L, Oza S, Hogan D, Perin J, Rudan I, Lawn JE, et al. Global, regional, and national causes of child mortality in 2000-13, with projections to inform post-2015 priorities: an updated systematic analysis. Lancet. 2015;385(9966):430-40.
3. Lawn JE, Cousens S, Zupan J; Lancet Neonatal Survival Steering Team. 4 million neonatal deaths: when? where? why? Lancet. 2005;365(9462):891-900.
4. Gillam-Krakauer M, Shah M, Gowen CW Jr. Birth asphyxia. In: StatPearls. Treasure Island, FL: StatPearls Publishing; 2025. Available at: https://www.ncbi.nlm.nih.gov/books/NBK430782/. Accessed 10 May 2025.
5. Solevag A, Schmolzer G, Cheung PY. Novel interventions to reduce oxidative-stress related brain injury in neonatal asphyxia. Free Radic Biol Med. 2019;142:113-22.
6. Hakobyan M, Dijkman KP, Laroche S, Naulaers G, Rijken M, Steiner K, et al. Outcome of infants with therapeutic hypothermia after perinatal asphyxia and early-onset sepsis. Neonatology. 2019;115(2):127-33.
7. Viaroli F, Cheung PY, O'Reilly M, Polglase GR, Pichler G, Schmolzer GM. Reducing brain injury of preterm infants in the delivery room. Front Pediatr. 2018;6:290.
8. Kapaya H, Williams R, Elton G, Anumba D. Can obstetric risk factors predict fetal acidaemia at birth? A retrospective case-control study. J Pregnancy. 2018;2018:2195965.
9. Douglas-Escobar M, Weiss MD. Hypoxic-ischemic encephalopathy: a review for the clinician. JAMA Pediatr. 2015;169(4):397-403.
10. National Institute for Health and Care Excellence. Intrapartum care for healthy women and babies. Clinical guideline CG190. London: NICE; 2014 (updated 2017). Available at: https://www.nice.org.uk/guidance/cg190. Accessed 10 May 2025.
11. Njie AE, Nyandiko WM, Ahoya PA, Moutchia JS. A comparative analysis of APGAR score and the gold standard in the diagnosis of birth asphyxia at a tertiary health facility in Kenya. PLoS One. 2023;18(5):e0285828.
12. Armstrong L, Stenson BJ. Use of umbilical cord blood gas analysis in the assessment of the newborn. Arch Dis Child Fetal Neonatal Ed. 2007;92(6):F430-4.
13. Gupta K, Haritwal A, Makhija B, Bhandari R. Is fetal CTG a reliable indicator of fetal distress? A prospective study on relationship between CTG suspected fetal distress and immediate postpartum umbilical cord blood pH. J Clin Med Kaz. 2022;19(1):57-64.
14. Alfirevic Z, Devane D, Gyte GM, Cuthbert A. Continuous cardiotocography (CTG) as a form of electronic fetal monitoring (EFM) for fetal assessment during labour. Cochrane Database Syst Rev. 2017;2(2):CD006066.
15. Ranjana, Goel N, Rani S. Correlation of cardiotocography with neonatal outcome in term

pregnancies at a tertiary care centre in Bihar, India: a prospective observational study. Panacea J Med Sci. 2023;13(1):202-8.
16. Khatun A, Khanam NN, Nazir F. Role of elaborate cardiotocography (CTG) in pregnancy management. BSMMU J. 2009;2(1):18-24.
17. Ayres-de-Campos D, Spong CY, Chandraharan E; FIGO Intrapartum Fetal Monitoring Expert Consensus Panel. FIGO consensus guidelines on intrapartum fetal monitoring: cardiotocography. Int J Gynaecol Obstet. 2015;131(1):13-24.
18. Aboulghar WM, Ibrahim MA, Allam IS, Hosny W, Otify M. Validity of cardiotocography in the diagnosis of acute fetal hypoxia in low resource settings. Internet J Gynecol Obstet. 2013;17(1):1-8.
19. Kanagal DV, Praveen BK. Intrapartum fetal monitoring and its correlation with umbilical cord blood pH and early neonatal outcome: a prospective cohort study. J South Asian Feder Obst Gynae. 2022;14(1):63-7.
20. Geva N, Geva Y, Katz L, Binyamin Y, Rotem R, Weintraub AY, et al. Correlation between total deceleration area in CTG records and cord blood pH in pregnancies with IUGR. Arch Gynecol Obstet. 2024;310(3):1425-31.
21. Xodo S, Londero AP. Is it time to redefine fetal decelerations in cardiotocography? J Pers Med. 2022;12(10):1552.
22. Paikaray S, Jena SK, Balakrishnan D, Mohanty PK. Correlation of cardiotocography with combined APGAR scores and diagnostic performance of umbilical cord parameters in predicting low combined APGAR scores: a prospective cohort study. Eur J Obstet Gynecol Reprod Biol X. 2024;23:100329.
23. Paladugu V, Sreedhar S, Chitra R, Mannava ST, Sreekumar S, Mangalakanthi J. Association of CTG diagnosis of intrapartum fetal distress and immediate postpartum acidemia in foetal umbilical artery. J Obstet Gynaecol India. 2023;73(1):28-35.
24. Ito A, Hayata E, Nagasaki S, Kotaki H, Shimabukuro M, Sakuma J, et al. Optimal duration of cardiotocography assessment using the iPREFACE score to predict fetal acidemia. Sci Rep. 2022;12(1):13064.
25. Locatelli A, Lambicchi L, Incerti M, Bonati F, Ferdico M, Malguzzi S, et al. Is perinatal asphyxia predictable? BMC Pregnancy Childbirth. 2020;20(1):186.
26. Jonsson M, Agren J, Norden-Lindeberg S, Ohlin A, Hanson U. Neonatal encephalopathy and the association to asphyxia in labor. Am J Obstet Gynecol. 2014;211(6):667.e1-8.

Cite this article as: Rana IS, Ravi Gowda HV. Association of cardiotocography diagnosis of intrapartum fetal distress with immediate postpartum acidaemia in the fetal umbilical artery. Int J Reprod Contracept Obstet Gynecol 2026;15:xxx-xx.