

# Association of Abnormal Intrapartum Cardiotocograph with Abnormal Umbilical Cord pH in Term Pregnant Women

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## ABSTRACT

**Objective:** To determine the frequency of abnormal intrapartum cardiotocography (CTG) and its association with abnormal umbilical cord arterial pH in term pregnant women.

**Study design and setting:** Descriptive longitudinal study conducted in the Department of Gynaecology and Obstetrics, Dow University Hospital, Karachi.

**Methodology:** A total of 178 term pregnant women (37–41 weeks), aged 20–45 years with gravidity and parity =1, were enrolled using non-probability consecutive sampling. Continuous intrapartum CTG was performed and classified according to prespecified criteria. Women with abnormal CTG received conservative measures according to ward protocol, followed by repeat CTG after 30 minutes. Umbilical cord arterial blood was collected immediately after birth for pH analysis; cord pH <7.2 was classified as abnormal. Data were analyzed using SPSS version 22.

**Results:** Abnormal intrapartum CTG was observed in 24/178 women (13.5%). Overall, abnormal umbilical cord arterial pH (<7.2) occurred in 18/178 neonates (10.1%). Abnormal cord pH was present in 11/24 neonates (45.8%) after abnormal CTG compared with 7/154 (4.5%) after normal CTG ( $p < 0.001$ ). Late decelerations and reduced variability showed the highest proportions of abnormal pH among the abnormal CTG patterns, although subgroup analyses were limited by small cell counts.

**Conclusion:** Abnormal intrapartum CTG occurred in 13.5% of term pregnancies and was significantly associated with abnormal umbilical cord arterial pH (<7.2). Cord arterial pH provides an objective biochemical measure that can complement CTG interpretation at birth.

**Keywords:** Cardiotocography, umbilical cord pH, fetal acidemia, term pregnancy, intrapartum monitoring.

## How to cite this Article:

Sofia, Nusrat U. Association of Abnormal Intrapartum Cardiotocograph with Abnormal Umbilical Cord pH in Term Pregnant Women. J Bahria Uni Med Dental Coll. 2026;16(4):987-93 DOI: <https://doi.org/10.51985/JBUMDC20261076>

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## INTRODUCTION

Intrapartum fetal surveillance is an essential element of practice in contemporary obstetrics due to the possibility of a sudden alteration of fetal oxygenation during labour. Periodic uterine contractions decrease uteroplacenta blood flow and adaptation to this physiological stress in most fetuses occurs due to cardiovascular compensatory processes. Nevertheless, in the presence of placental reserve deficiency or cord compression, uterine hyperstimulation, maternal hypoxia, hypotension, infection, or other intrapartum complications, fetal hypoxemia can be diagnosed.<sup>1</sup> Cardiotocography (CTG) has become quite common and is used to measure the fetal heart rate and uterine contractions during labour. It gives constant data on the basal fetal heart

rate, variability, asymmetries, and deceleration of the fetus in relation to the activity of the uterus. The key goal of CTG is to identify fetal compromise at an early stage before it is too late. Abnormal patterns like tachycardia, decreased variability, repeated late decelerations, increased decelerations, and non-reassuring persistent traces can indicate the developing fetal hypoxia or acidosis.<sup>2</sup> Interpretation of CTG is still difficult although it is very prevalent. The visual interpretation will be subjective, which can differ among the observers. CTG is very sensitive in identifying potential fetal compromise, but low in specificity, and potentially results in a higher rate of operative delivery without necessarily better neonatal outcome. A false-positive CTG result may result in unwarranted emergency caesarean delivery or instrumental birth and a false-negative or slow response may result in the inability to recognize evolving fetal acidosis. Hence, it is crucial to correlate with neonatal objective indicators.<sup>3</sup>

Umbilical cord blood gas analysis offers first hand data on the condition of the fetal acid-base at birth. Umbilical venous blood indicates the placental and maternal acid base homeostasis, whereas the umbilical arterial blood assures more accurately about the metabolic condition of the

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Received: 04-06-2026  
Accepted: 17-09-2026

1st Revision: 07-07-2026  
2nd Revision: 13-07-2026

neonates since the blood that comes back to the placenta is fetus. The importance of cord arterial pH, base deficit, lactate, and bicarbonate can be used to differentiate between respiratory acidosis and metabolic acidosis. Metabolic acidosis has better links to neonatal morbidity when compared to isolated respiratory acidosis.<sup>4</sup> A common objective measure of intrapartum hypoxia and fetal acidemia is cord pH. Though various cut-offs have been applied in the literature, the pH below 7.2 is usually considered as abnormal in most of the clinical research, even though many other studies would define more severe acidemia as a lower pH (e.g., pH less than 7.1 or less than 7.0). In the current research, the operational definition of abnormal cord pH was cord pH less than 7.2 in line with the synopsis protocol.<sup>5</sup> Abnormal CTG and abnormal cord pH have clinical significance to the relationship. In case abnormal CTG has a strong predictive ability of the abnormal cord pH, then it will allow timely intervention during parturition. In case the connection is not strong, using CTG alone can lead to unwarranted delivery of operations. The research has found fluctuating effects of suspicious or pathological CTG and cord blood parameters. This difference can be associated with the variations in CTG classification, population risk, labour management, timing of intervention, cord sampling procedure, as well as, pH cut-off.<sup>6,7</sup> The recent researches have revived the interest in the diagnostic limitations regarding CTG and the significance of cord blood analysis. Tomich et al. compared intrapartum CTG categories and fetal acidemia with umbilical cord blood analysis, and discovered that CTG had a limited capacity to spot acidemia in low-risk pregnancies.<sup>8</sup> Paikaray et al. found that, suspicious and pathological patterns of CTG were associated with cord blood pH, base excess and lactate, referring to the cord blood gas parameter objective assessment.<sup>9</sup> Intrapartum CTG has become the standard practice in tertiary care hospitals of Pakistan, and it is specifically applied to women, who have been admitted in active labour or to those women who are in need of continuous monitoring. Nonetheless, domestic information regarding the incidence of abnormal intrapartum CTG and its relation to cord pH are still scarce. Differences in monitoring regimens, personnel training, assessment of fetal blood samples, time to delivery, and cord gas assessment can impact results.<sup>10</sup> The current research was thus prepared to address the frequency of abnormal intrapartum CTG in term pregnant women as well as frequency of abnormal umbilical cord pH in women with abnormal intrapartum CTG in a tertiary hospital in Karachi.

## METHODOLOGY

This descriptive longitudinal study was conducted in the Department of Gynaecology and Obstetrics, Dow University Hospital, Karachi, from 4 March 2026 to 3 June 2026 after institutional review board approval. The study objective was to determine the frequency of abnormal intrapartum CTG in term pregnant women and its association with abnormal

umbilical cord arterial pH. Written informed consent was obtained from all participants before enrollment, and confidentiality was maintained. The sample size was determined in the WHO sample size calculator in one population proportion. Previous evidence regarding the frequency of the occurrence of the abnormal intrapartum CTG was 13.3% and assumed as expected after all. The level of confidence set was 95 and a margin of error was maintained at 5%. Using the formula  $n = Z^2 p(1-p)/d^2$ , where  $Z = 1.96$ ,  $p = 0.133$ , and  $d = 0.05$ , the required sample size was calculated as 177.3. A final sample size of 178 patients was obtained by rounding. Consecutive sampling was also employed but this was not probability sampling, all eligible women who were in labour during the period under study were recruited up to the level that the desired sample was achieved. Included pregnant women at term who had a gestational age of 37 weeks determined by the last menstrual period or first-trimester dating ultrasound. Single pregnancies that were confirmed through ultrasound were considered. Women who were parity and gravidity 1 or more and at least 20-45 years old of maternal age were included. Out were women with known fetal congenital abnormality, chromosomal abnormality, intrauterine fetal death prior to labour, preexisting maternal cardiovascular or respiratory disease, diabetes mellitus, hypertension with severe characteristics, or pre-delivery exposure to medications that altered fetal heart rate patterns (tocolytics or magnesium sulfate). Patients who did not give a consent were also omitted.

Beginning with enrollment, demographic and obstetric information was recorded on a structured proforma. Variables were maternal age, gestational age, occupational status, gravida, parity, mode of delivery, intrapartum CTG status, specific CTG abnormality, repeated CTG response after conservative measures, umbilical cord pH. Constant monitoring of the fetus was done during labour. CTG was or was not applied based on the synopsis definition of what it is. The cardiotocograph was classified as abnormal when one or more of the following existed: fetal tachycardia with a baseline of above 170beats per minute, low variability of less than 5 beats per minute, early decelerations with the fall of at least 15beats per minute during a duration of at least 15 seconds during uterine contraction, late decelerations with a fall at least 30beats per minute during a period of at least two minutes, or reduced variability at least 5beats per minute during a period of at least 15 seconds during uterine contraction. Any woman, having abnormal CTG, was left in the lateral position and kept on inhalation of oxygen and water hydration as per the research study. Repeat CTG done after 30mins with persistence or improvement of abnormal trace noted. The clinical team was in charge of making decisions on obstetric management based on institutional practice and fetal-maternal condition. Umbilical cord arterial blood was collected immediately after birth into a pre-

heparinized syringe and transported directly to the laboratory for pH analysis. Cord pH <7.2 was defined as abnormal. The source manuscript does not document the exact elapsed time from collection to laboratory analysis; this interval should be confirmed from the original laboratory protocol before submission to improve reproducibility. The main outcomes were the frequency of abnormal intrapartum CTG, the overall frequency of abnormal cord pH, and the association between CTG status and cord pH. Data were entered and analyzed using SPSS version 22. Quantitative variables such as maternal age and gestational age were summarized as mean  $\pm$  standard deviation, and categorical variables as frequency and percentage. Associations between categorical variables and abnormal CTG or abnormal cord pH were assessed using the chi-square test when expected cell counts were adequate. Fisher's exact test was used for 2 $\times$ 2 comparisons with small expected cell counts, including subgroup analyses among the 24 women with abnormal CTG. Maternal age, gestational age, parity, gravidity, occupational status, and mode of delivery were stratified. A two-sided p-value =0.05 was considered statistically significant.

## RESULTS

A total of 178 term pregnant women admitted in labour were included. Mean maternal age was  $28.6 \pm 5.4$  years. Sixty-eight women (38.2%) were aged 20–25 years, 72 (40.4%) were aged 26–35 years, and 38 (21.3%) were aged >35 years. Mean gestational age was  $38.8 \pm 1.1$  weeks; 91 women (51.1%) were at 37–38 weeks and 87 (48.9%) at 39–41 weeks. Forty-six women (25.8%) were employed and 132 (74.2%) were unemployed. Gravida 1–2 was recorded in 96 women (53.9%) and gravida =3 in 82 (46.1%). Parity 1–2 was present in 118 women (66.3%) and parity =3 in 60 (33.7%). Normal vaginal delivery occurred in 129 women (72.5%), while 49 (27.5%) underwent lower-segment caesarean section. Abnormal intrapartum CTG was identified in 24/178 women (13.5%), while 154/178 (86.5%) had normal CTG. Among the abnormal CTG patterns, reduced variability was recorded in 9 women, late decelerations in

8, tachycardia in 7, prolonged decelerations in 6, and early decelerations in 5; some women had more than one abnormal feature. Conservative measures (left lateral positioning, oxygen administration, and hydration) were followed by CTG improvement in 10/24 women (41.7%), while abnormality persisted in 14/24 (58.3%).

Abnormal umbilical cord arterial pH (<7.2) was found in 18/178 neonates (10.1%) overall. It occurred in 11/24 neonates (45.8%) after abnormal CTG and in 7/154 (4.5%) after normal CTG ( $p<0.001$ ). Mean cord pH was  $7.22 \pm 0.09$  in the abnormal-CTG group and  $7.31 \pm 0.06$  in the normal-CTG group ( $p<0.001$ ). Mode of delivery differed by CTG status. Lower-segment caesarean section was performed in 15/24 women (62.5%) with abnormal CTG and 34/154 (22.1%) with normal CTG. Abnormal CTG was therefore more frequent among women delivered by caesarean section than among those delivered vaginally. Stratified analysis showed slightly higher proportions of

Table 1. Baseline demographic and obstetric characteristics, n=178

| Variable                        | Frequency / Mean                 | Percentage / SD |
|---------------------------------|----------------------------------|-----------------|
| Maternal age, years             | <b>28.6 <math>\pm</math> 5.4</b> |                 |
| Age 20–25 years                 | 68                               | 38.2%           |
| Age 26–35 years                 | 72                               | 40.4%           |
| Age >35 years                   | 38                               | 21.3%           |
| Gestational age, weeks          | <b>38.8 <math>\pm</math> 1.1</b> |                 |
| Gestational age 37–38 weeks     | 91                               | 51.1%           |
| Gestational age 39–41 weeks     | 87                               | 48.9%           |
| Employed                        | 46                               | 25.8%           |
| Unemployed                      | 132                              | 74.2%           |
| Gravida 1–2                     | 96                               | 53.9%           |
| Gravida =3                      | 82                               | 46.1%           |
| Parity 1–2                      | 118                              | 66.3%           |
| Parity =3                       | 60                               | 33.7%           |
| Normal vaginal delivery         | 129                              | 72.5%           |
| Lower segment caesarean section | 49                               | 27.5%           |

Table 2. Frequency and pattern of abnormal intrapartum cardiotocograph

| CTG variable                           | Frequency | Percentage            |
|----------------------------------------|-----------|-----------------------|
| Normal intrapartum CTG                 | 154       | 86.5%                 |
| Abnormal intrapartum CTG               | 24        | 13.5%                 |
| Tachycardia >170 beats/min             | 7         | 3.9%                  |
| Reduced variability <5 beats/min       | 9         | 5.1%                  |
| Early decelerations                    | 5         | 2.8%                  |
| Prolonged decelerations                | 6         | 3.4%                  |
| Late decelerations                     | 8         | 4.5%                  |
| More than one abnormal CTG feature     | 7         | 3.9%                  |
| CTG improved after 30 minutes          | 10        | 41.7% of abnormal CTG |
| CTG remained abnormal after 30 minutes | 14        | 58.3% of abnormal CTG |

Table 3. Association of intrapartum CTG with umbilical cord pH

| CTG status   | Abnormal cord pH <7.2 | Normal cord pH =7.2 | Total | p-value |
|--------------|-----------------------|---------------------|-------|---------|
| Abnormal CTG | 11 (45.8%)            | 13 (54.2%)          | 24    | <0.001  |
| Normal CTG   | 7 (4.5%)              | 147 (95.5%)         | 154   |         |
| Total        | 18 (10.1%)            | 160 (89.9%)         | 178   |         |
| Mean cord pH | 7.22±0.09             | 7.31±0.06           |       | <0.001  |

Table 4. Stratified associations of maternal/obstetric factors with abnormal CTG and, among abnormal CTG cases, cord pH Fisher's exact test was used for the n=24 abnormal-CTG subgroup because of small cell counts; subgroup p-values are exploratory.

| Variable                                                                              | Abnormal CTG<br>n/N (%) | Normal CTG<br>n/N (%) | p-value |
|---------------------------------------------------------------------------------------|-------------------------|-----------------------|---------|
| Age 20–25 years                                                                       | 7/68 (10.3%)            | 61/68 (89.7%)         | 0.352   |
| Age 26–35 years                                                                       | 10/72 (13.9%)           | 62/72 (86.1%)         |         |
| Age >35 years                                                                         | 7/38 (18.4%)            | 31/38 (81.6%)         |         |
| Gestational age 37–38 weeks                                                           | 10/91 (11.0%)           | 81/91 (89.0%)         | 0.301   |
| Gestational age 39–41 weeks                                                           | 14/87 (16.1%)           | 73/87 (83.9%)         |         |
| Employed                                                                              | 5/46 (10.9%)            | 41/46 (89.1%)         | 0.532   |
| Unemployed                                                                            | 19/132 (14.4%)          | 113/132 (85.6%)       |         |
| Gravida 1–2                                                                           | 11/96 (11.5%)           | 85/96 (88.5%)         | 0.416   |
| Gravida =3                                                                            | 13/82 (15.9%)           | 69/82 (84.1%)         |         |
| Parity 1–2                                                                            | 14/118 (11.9%)          | 104/118 (88.1%)       | 0.393   |
| Parity =3                                                                             | 10/60 (16.7%)           | 50/60 (83.3%)         |         |
| Normal vaginal delivery                                                               | 9/129 (7.0%)            | 120/129 (93.0%)       | <0.001  |
| Lower segment caesarean section                                                       | 15/49 (30.6%)           | 34/49 (69.4%)         |         |
| Among women with abnormal CTG (n=24): abnormal cord pH by CTG feature / delivery mode |                         |                       |         |
| Tachycardia present                                                                   | 3/7 (42.9%)             | 4/7 (57.1%)           | 1.000   |
| Reduced variability present                                                           | 6/9 (66.7%)             | 3/9 (33.3%)           | 0.206   |
| Early decelerations present                                                           | 1/5 (20.0%)             | 4/5 (80.0%)           | 0.327   |
| Prolonged decelerations present                                                       | 3/6 (50.0%)             | 3/6 (50.0%)           | 1.000   |
| Late decelerations present                                                            | 6/8 (75.0%)             | 2/8 (25.0%)           | 0.082   |
| More than one abnormal feature                                                        | 5/7 (71.4%)             | 2/7 (28.6%)           | 0.182   |
| CTG improved after 30 minutes                                                         | 2/10 (20.0%)            | 8/10 (80.0%)          | 0.047   |
| CTG remained abnormal after 30 minutes                                                | 9/14 (64.3%)            | 5/14 (35.7%)          | 0.047   |
| Normal vaginal delivery                                                               | 2/9 (22.2%)             | 7/9 (77.8%)           | 0.105   |
| Lower segment caesarean section                                                       | 9/15 (60.0%)            | 6/15 (40.0%)          | 0.105   |

abnormal CTG among women aged >35 years, those at 39–41 weeks, gravida =3, and parity =3, although these differences were not statistically significant. Among women with abnormal CTG, abnormal cord pH was more frequent with persistent abnormal CTG after conservative measures, late decelerations, reduced variability, and caesarean delivery. Because several subgroup cells were small, these analyses were evaluated with Fisher's exact test and should be interpreted as exploratory.

## DISCUSSION

This descriptive longitudinal study evaluated the frequency of abnormal intrapartum CTG and its association with

abnormal umbilical cord arterial pH in term pregnancies at a tertiary-care hospital in Karachi. Abnormal CTG occurred in 13.5% of women, while abnormal cord pH (<7.2) occurred in 18/178 neonates (10.1%) overall and in 45.8% of neonates after abnormal CTG. The significant CTG–pH association supports the clinical value of CTG as a screening tool while also showing that more than half of abnormal CTG traces were not accompanied by biochemical acidemia. The frequency of abnormal CTG in the present study (13.5%) was close to the 13.3% estimate reported by Ray and Ray and used for sample-size calculation.<sup>11</sup> Differences in reported frequencies across studies may reflect variation in case selection, obstetric risk profile, CTG classification, duration

of labour monitoring, and timing of assessment. In the current study, it was found that 45.8% of women with abnormal CTG were experiencing abnormal cord pH. This was similar to prior studies which reported abnormal cord pH in around forty percent of women with nonreassuring CTG patterns.<sup>11</sup> This result implies that the significant clinical association between abnormal CTG and fetal acid-base imbalance is both clinically meaningful and indicates that it lacks specificity in that over half of abnormal CTG findings showed normal cord pH. Paikaray et al. found out that pathological CTG was characterized by low mean cord pH, elevated rate of abnormal blood gas parameters in contrast with suspicious CTG.<sup>1</sup> Their results confirm our finding that not every CTG aberration is equal in its jeopardy. Late decelerations and low variability were more closely linked with abnormal cord pH in our study compared to early decelerations. This is mostly physiological since late decelerations are symptomatic of uteroplacental underdevelopment and slow heart rate recovery following contractions of the fetus and low variability could be indicative of poor fetal autonomic reaction.

According to Tomich et al., the accuracy of intrapartum CTG predictive of fetal acidemia based on analyses of umbilical cord blood in low-risk pregnancy was limited.<sup>2</sup> This is a tentative meaning that our research upholds. Even though an abnormal CTG was strongly linked to an abnormal cord pH a large proportion of abnormal traces were not acidotic. Thus, CTG must be used to inform clinical evaluation, however it must not be read on its own. The results of Leoni et al. also revealed poor performance of CTG in the detection of fetal acidemia in the event the objective outcome was cord arterial pH.<sup>3</sup> In comparison with their results, the current research was more associated with abnormal pH, perhaps due to our lower cut-off at <7.2 as compared to more stringent ones that included cut-offs at <7.1. A higher pH cutoff improves the identification of mild acidosis and can enhance the perceived correlation with CTG abnormality. Paladugu, et al. observed that umbilical cord arterial pH is an objective technique to diagnose intrapartum fetal hypoxia amid cases categorized as fetal distress on CTG.<sup>4</sup> This is the same opinion that is held in the current study. The pH of cord blood is a biochemical validation, and it assists in assessing the correctness of intrapartum decisions. It can also facilitate fetal status at birth neonatal triage, and documentation.

Olofsson assessed cord blood pH, gases, and lactate, and highlighted how they are interpreted and used clinically at birth.<sup>5</sup> Cord pH had been employed as a primary biochemical endpoint in our study. Even though pH in itself is clinically helpful, the additional information regarding the metabolic acidosis is offered by base deficit and lactate. Subsequent local research ought to incorporate pH, base excess, bicarbonate, lactate and Apgar scores to help differentiate better between respiratory and metabolic acidosis. The

average cord pH of women with abnormal CTG was minimized compared to normal CTG. This result is in accord with the pathophysiology behind CTG monitoring. When the supply of oxygen is low, and fetal adaptive responses are triggered, fetal hearts may experience abnormalities in heart rate. Changes in fetal heart rates however could also be caused by non-hypoxic factors that include fetal sleep cycle, maternal fever, drugs, fetal movement, cord compression without sustained hypoxia, or technical artifact.

Among women with abnormal CTG, late decelerations and reduced variability had relatively high proportions of abnormal cord pH. However, after use of two-sided Fisher's exact tests for the small subgroup counts, none of the individual CTG features reached conventional statistical significance; late decelerations had  $p=0.082$  and reduced variability  $p=0.206$ . These findings should therefore be treated as exploratory rather than definitive evidence of feature-specific risk. Reduced variability was accompanied by abnormal cord pH in 6/9 cases (66.7%), but this subgroup comparison was not statistically significant on Fisher's exact testing ( $p=0.206$ ). Reduced variability may occur with hypoxia and acidosis but can also be influenced by fetal sleep state, medications, prematurity, or technical factors; interpretation therefore requires clinical context. Early decelerations showed the lowest proportion of abnormal cord pH (1/5, 20.0%) among the displayed CTG features. This is biologically plausible because early decelerations are commonly associated with fetal head compression and are often benign when isolated. Nevertheless, the subgroup size was small, and the Fisher's exact  $p$ -value was 0.327. Persistence of abnormal CTG after conservative measures was associated with a higher proportion of abnormal cord pH than improvement after 30 minutes (9/14 [64.3%] vs 2/10 [20.0%]; Fisher's exact  $p=0.047$ ). This finding suggests that persistent abnormality may identify a higher-risk subgroup, although the small sample requires cautious interpretation.

Mode of delivery was strongly associated with abnormal CTG. LSCS occurred in greater number in the women who had abnormal CTG compared to those with normal CTG. This is agreeable to the previously established effect of CTG on rate of delivery of operation. The review by Al Wattar et al. examined various intrapartum fetal surveillance techniques and found that various monitoring approaches have an effect on operative delivery and neonatal outcomes.<sup>7</sup> Our research also reveals that abnormality CTG commonly results in the caesarean birth. Nevertheless, the correlation between abnormal CTG and caesarean birth should be taken in moderation. Caesareans might be justified due to abnormal CTG indicative of actual fetal compromise, whereas other cases could be as a result of low specificity of CTG. Hence, standardized of interpretation of CTG, second opinion, and documentation of response to conservative measures could contribute to minimization of unnecessary operative delivery.

According to Tome et al., theirs found no improvements in maternal and neonatal outcomes as a result of routine admission CTG in low-risk term pregnancies.<sup>10</sup> Although, unlike in our study, our intrapartum CTG outcomes were compared to admission CTG outcomes, their results highlight that the indiscriminate use of CTG is not necessarily associated with better outcome. The usefulness of CTG is reliant on a patient selection, proper interpretation, and prompt clinical response.

Oluwasomidoyin et al. evaluated admission CTG and neonatal outcomes and discovered that there was a connection between abnormal admission traces and adverse consecutive neonatal indicators.<sup>12</sup> This argues in favor of the idea that CTG has the power to detect fetuses at risk, yet the classification of CTG and local management guidelines have a role in clinical utility. Abnormal intrapartum CTG showed a relationship with abnormal cord pH in our study, and abnormal intrapartum CTG is used to monitor fetal health. Silva et al. compared the nuchal cord and perinatal outcomes in which tight nuchal cords were linked to certain early neonatal outcome but not extensive adverse outcomes.<sup>13</sup> This is applicable since the compression of the cord may lead to periodic or extended decelerations on CTG. Abnormalities of the cord were not separately studied in the present study, but nuchal cord, true knot, cord prolapse, and cord around body are the potential add values that can be added in the future. According to Brodowski et al., a pattern of CTGs in nuchal cord was typically not linked with higher perinatal morbidity.<sup>14</sup> This supports the notion that there is no uniformity between the presence of abnormal CTGs and fetal acidemia. Not all the abnormalities could be a persistent metabolically impaired cord compression. Cord pH test can be used to differentiate these situations. Eenkhoorn et al. summarized research on fetal heart rate variability and neonatal outcome and pointed out methodological variability in the CTG research.<sup>15</sup> This applies to our study since it is hard to make comparisons across studies in cases where there are varying CTG definitions, pH thresholds, and neonatal outcomes. There needs to be standardized definitions to improve evidence synthesis. Miyata et al. made a comparison between artificial intelligence and human expert interpretation of CTG and discovered that automated methods could aid in the prediction of fetal asphyxia.<sup>16</sup> In our study CTG interpretation was based on visual interpretation, but the interpretation could be based on computerized or AI-assisted interpretation to decrease subjectivity and interobserver variability.

Recent reviews have also focused on advances in fetal heart rate monitoring with the limitations of CTG analyses, interpretation of signals, and novel technologies.<sup>17</sup> In busy and low-resource tertiary environments, enhancement of CTG training and standardization might be more practical

in the short term in comparison with advanced technologies. Nevertheless, in the future computerized CTG and decision-support tools can be significant.<sup>18</sup> The findings have several practical implications. Abnormal CTG occurred in 13.5% of term labours, and abnormal cord pH occurred in 10.1% of neonates overall. Nearly half of abnormal CTG cases had cord pH <7.2, while 4.5% of neonates after normal CTG also had abnormal pH. Persistent abnormal CTG after conservative measures was associated with a higher proportion of abnormal pH. These observations support CTG as a useful screening tool but also reinforce the value of objective neonatal acid–base assessment where available. There are a number of strengths in the study. It comprised a specific operational definition of abnormal CTG, singleton pregnancies were considered, cord arterial blood was sampled at the time of birth, cord pH was a biochemical end, and the relationship to maternal and obstetric factors was assessed. It also had the pragmatic labour-room course of deliberate measures and reiteration CTG amidst aberrant traces. Limitations: This was a single-centre study using non-probability consecutive sampling, which limits generalizability. CTG interpretation was based on visual assessment, and interobserver agreement was not evaluated. Cord blood assessment was limited to pH without base deficit, lactate, bicarbonate, or other neonatal outcomes such as Apgar scores, NICU admission, or neonatal encephalopathy. The exact collection-to-analysis interval for cord arterial blood was not documented in the source manuscript. Finally, only 24 women had abnormal CTG, resulting in small subgroup cell counts and limited statistical power for feature-specific analyses. Irrespective of these shortcomings, this research favours the use of intrapartum CTG as a convenient method of screening potential cases of fetal acidemia during term pregnancies. It also highlights the importance of cord pH where it is available and objectively assess neonatal acid–base conditions, audit CTG interpretation, and inform neonatal care.

## CONCLUSION

Abnormal intrapartum CTG was observed in 13.5% of term pregnancies. Abnormal umbilical cord arterial pH (<7.2) occurred in 10.1% of neonates overall and in 45.8% of neonates after abnormal CTG. Abnormal CTG was significantly associated with abnormal cord pH, while persistent abnormal CTG after conservative measures was associated with a higher proportion of abnormal pH. CTG remains a useful intrapartum screening tool, and cord arterial pH provides an objective measure of fetal acid–base status at birth.

**Conflict of Interest:** None

**Fundings:** No funding source

**Use of Artificial Intelligence (AI) Disclosure:** There is no AI tool used.

**Authors Contribution:**

**Sofia:** Conception and design, acquisition of data analysis and interpretation of data, drafting and critical revision, final approval of the version to be published.

**Uzma Nusrat:** Conception and design, acquisition of data analysis and interpretation of data, drafting and critical revision, final approval of the version to be published

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