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Intrapartum Fetal Monitoring and its Correlation with Umbilical Cord Blood Lactate Levels and Early Neonatal Outcome

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Abstract: ***Accurate intrapartum fetal monitoring is crucial for detecting hypoxia, and while CTG is useful, its limitations and false positives make biochemical markers like umbilical cord lactate essential, as elevated lactate more reliably indicates metabolic acidosis and adverse neonatal outcomes; combining CTG with lactate improves diagnostic precision, guides interventions more effectively, and reduces unnecessary procedures. Conclusion: Integrating cord lactate with CTG improves accuracy in detecting fetal hypoxia, reduces false positives and unnecessary interventions, and better predicts neonatal outcomes, offering a more reliable, evidence-based intrapartum monitoring approach.***

Keywords: intrapartum fetal monitoring; cardiotocography; CTG; umbilical cord blood lactate; fetal hypoxia; metabolic acidosis; neonatal outcomes; birth asphyxia; biochemical markers; fetal distress; neonatal morbidity; fetal acid–base status.

1. Introduction

Intrapartum-related fetal compromise remains a significant contributor to early neonatal morbidity and mortality, particularly in settings where timely detection of hypoxia is challenging. Continuous fetal monitoring was introduced to reduce preventable intrapartum injuries by providing earlier recognition of fetal distress during labor. However, the clinical effectiveness of electronic fetal monitoring techniques has been widely debated due to concerns regarding accuracy, interpretation variability, and potential over-intervention.[1]

The global emphasis on improving childbirth outcomes has led to strengthened recommendations for careful monitoring during labor. The World Health Organisation highlights the importance of systematic intrapartum surveillance as part of a positive childbirth experience, emphasising that early recognition of abnormal fetal status enables timely obstetric action and can reduce adverse outcomes. WHO guidelines outline the use of intermittent auscultation and cardiotocography depending on risk status, resources, and clinical context, reinforcing the role of monitoring in preventing avoidable neonatal complications.[2]

During an episode of intrapartum hypoxia, the fetus undergoes several physiological responses aimed at preserving oxygen supply to vital organs. Experimental work has shown that acute reductions in oxygen availability trigger compensatory cardiovascular adjustments such as redistribution of cardiac output and changes in heart rate patterns. When hypoxia persists, metabolic shifts occur, including a transition from aerobic to anaerobic metabolism, leading to elevated lactate generation. These physiological events contribute to measurable changes in fetal status that may later be reflected in neonatal outcomes.[3]

Umbilical cord blood analysis remains an essential tool for assessing fetal metabolic condition at birth. Lactate, pH, and blood gas values provide objective insight into the degree of intrapartum hypoxia and the presence of metabolic acidosis. Contemporary reviews describe normal lactate levels at birth and emphasise that elevated concentrations correlate with impaired oxygenation and anaerobic metabolism. Lactate measurement is particularly valuable because it rises earlier in hypoxic states and is more stable than pH in the post-delivery period, making it a reliable indicator of fetal metabolic stress.[4]

Accurate sampling and interpretation of umbilical cord blood gases are essential to understanding the relationship between intrapartum events and neonatal condition. Clinical guidance highlights that cord lactate assessment is practical, requires smaller sample volumes, and is less affected by sampling delays compared with traditional pH analysis. These properties support its use as an adjunct to intrapartum fetal monitoring and a meaningful marker of neonatal risk, especially when CTG findings are ambiguous or non-specific.[5]

Historical Background of Intrapartum Fetal Monitoring

Early fetal monitoring relied primarily on simple acoustic tools such as the Pinard stethoscope, which allowed intermittent auscultation of fetal heart sounds. Over time, technological advances led to the development of electronic fetal monitoring (EFM), also known as cardiotocography (CTG), which enabled continuous assessment of fetal heart rate patterns and uterine contractions. The introduction of CTG aimed to enhance early recognition of fetal compromise and improve neonatal outcomes, but questions regarding its accuracy and predictive value emerged as usage became widespread.[6]

As CTG expanded globally, the need for standardized interpretation systems became evident due to significant variability in clinical judgments. Several professional bodies established structured classification frameworks to reduce subjective bias and stratify fetal risk more consistently. The three-tiered systems—including those proposed by FIGO, ACOG/NICHD, and NICE—categorize CTG tracings into reassuring, indeterminate, or pathological patterns based on baseline fetal heart rate, variability, accelerations, and decelerations. These frameworks were developed to support clinical decision-making and improve outcomes by promoting uniformity in CTG interpretation, although challenges in reproducibility and predictive accuracy persist.[6]

Advances in physiological interpretation of fetal heart rate patterns emphasized the significance of baseline shifts occurring after decelerations, which may reflect underlying hypoxic stress or evolving acidosis. This perspective reinforced the need for biochemical confirmation when tracings are non-reassuring.[7]

The incorporation of biochemical markers such as fetal scalp blood pH, base deficit, and lactate emerged as a major development in intrapartum assessment. These markers provide direct information about fetal acid–base balance and the extent of anaerobic metabolism, improving diagnostic accuracy in cases where CTG appears suspicious but not definitive. Lactate gained particular attention due to faster measurement, smaller sample requirements, and greater stability compared with pH, offering a practical adjunct to traditional monitoring methods. The evolution from simple auscultation to integrated electronic and biochemical assessment reflects the continuous effort to enhance fetal surveillance and reduce preventable intrapartum injury.[7]

Physiology of Fetal Oxygenation and Hypoxia

Adequate fetal oxygenation during labor depends primarily on effective uteroplacental blood flow, which delivers oxygenated maternal blood to the intervillous space where gas exchange occurs. Unlike the adult circulation, the fetus relies entirely on placental transfer rather than pulmonary oxygenation, making it vulnerable to any factors that reduce placental perfusion or increase fetal oxygen demand. During labor, contractions may transiently decrease uteroplacental flow; a healthy fetus compensates through cardiovascular adaptations that maintain adequate oxygen delivery. When these compensatory mechanisms fail or hypo-perfusion becomes prolonged, fetal hypoxemia begins to develop.[8]

Fetal hypoxemia refers to reduced oxygen content in fetal blood. Initially, the fetus responds through autonomic adjustments such as tachycardia or increased variability, reflecting intact compensatory pathways. Experimental and clinical observations show that the fetal heart redistributes cardiac output to vital organs such as the brain, heart, and adrenal glands when oxygen levels fall. This “centralization” ensures temporary preservation of essential organ function. However, persistent or severe hypoxemia exceeds compensatory capacity and progresses toward tissue hypoxia, in which oxygen delivery becomes insufficient for metabolic needs.[8]

With sustained oxygen deprivation, fetal cells shift from aerobic metabolism, which relies on adequate oxygen, to anaerobic glycolysis, a pathway that produces energy in low-oxygen environments. A key biochemical consequence of this switch is the increased production of lactate, a byproduct of anaerobic metabolism. Elevated lactate accumulates in fetal blood and tissues, eventually contributing to metabolic acidosis. This acid–base disturbance reflects inadequate oxygenation severe enough to impair cellular function. If uncorrected, metabolic acidosis can lead to loss of myocardial contractility, diminished organ perfusion, and eventual multi-organ injury.[9]

Rising lactate levels correlate closely with the severity and duration of fetal hypoxia. Because lactate accumulation occurs earlier than significant drops in pH, it serves as a sensitive indicator of anaerobic metabolism. High lactate concentrations at birth often reflect clinically important intrapartum hypoxia and may predict adverse neonatal outcomes. These physiological principles form the basis for using umbilical cord lactate as a biochemical adjunct to intrapartum fetal monitoring, providing objective evidence of metabolic compromise that CTG alone may not reliably detect.[9]

Intrapartum Fetal Monitoring Techniques

1) Intermittent Auscultation (IA)

Intermittent auscultation involves periodic assessment of the fetal heart rate using simple tools such as a fetoscope or Doppler device. IA remains an acceptable method for monitoring low-risk labor because it is noninvasive, low-cost, and does not restrict maternal mobility. However, the technique provides only brief snapshots of fetal heart activity and may fail to detect evolving patterns of distress that occur between assessments.[10]

2) Electronic Fetal Monitoring (Cardiotocography – CTG)

CTG records fetal heart rate and uterine contractions continuously, allowing clinicians to assess baseline rate, variability, accelerations, and decelerations—key indicators of fetal oxygenation. CTG became widely adopted to reduce intrapartum asphyxia, but its clinical performance has been questioned due to high rates of false-positive results and subjective interpretation. Research indicates that non-reassuring CTG patterns are common even when cord pH is normal, leading to unnecessary interventions such as operative deliveries. The variability in interpretation underscores the need for standardised classification systems to reduce observer error.[11]

Classification systems such as Category I–III (reassuring, indeterminate, pathological) help clinicians stratify fetal risk. [11]

3) Fetal Scalp Blood Sampling (FSBS)

Fetal scalp blood sampling is used when CTG findings are non-reassuring but not definitively pathological. It provides direct measurement of fetal acid–base status through parameters such as pH, base deficit, and lactate. Studies demonstrate a strong association between abnormal CTG patterns and low cord pH, confirming that biochemical testing

provides objective information that CTG alone cannot offer. FSBS lactate measurement is increasingly preferred because it requires a smaller blood volume and yields results more rapidly than pH analysis.[12]

Research comparing CTG categories with umbilical artery acid–base values noted that more severe CTG abnormalities correlated with lower pH and higher lactate levels. This supports the clinical practice of combining CTG with scalp sampling to improve diagnostic accuracy and reduce unnecessary obstetric interventions prompted by false-positive tracings.[12]

4) ST-Segment Analysis (STAN)

STAN is an advanced monitoring technique that evaluates fetal electrocardiographic ST-segment changes alongside CTG. The method aims to detect myocardial hypoxia earlier than CTG alone by assessing electrical changes that occur during oxygen deprivation. Although not universally available, STAN has been introduced as a tool to complement traditional monitoring. Studies analyzing CTG tracings and neonatal outcomes highlight the importance of interpreting complex patterns physiologically- recognizing that changes in baseline patterns following decelerations may indicate evolving hypoxic stress.[13]

5) Emerging Modalities: Computerized CTG and AI-Assisted Monitoring

Advances in digital technology have led to the development of computerized CTG systems that analyze fetal heart rate parameters quantitatively, reducing observer variability. These automated systems aim to detect abnormalities more consistently and provide decision-support alerts during labor. The physiological interpretation approach also highlights the potential for artificial intelligence (AI) to assist clinicians by identifying patterns that correlate with neonatal outcomes more precisely than manual interpretation alone. Early findings suggest that such tools may ultimately improve prediction of fetal acidemia when integrated with traditional monitoring.[13]

Umbilical Cord Blood Lactate- Biomarker of Fetal Hypoxia

Umbilical cord blood lactate has become a valuable marker of intrapartum fetal hypoxia because it directly reflects the extent of anaerobic metabolism, which occurs when oxygen delivery becomes insufficient. During labor, decreased uteroplacental perfusion or sustained decelerations may lead the fetus to shift from aerobic to anaerobic glycolysis, resulting in lactate accumulation. Studies correlating non-reassuring fetal heart rate patterns with biochemical outcomes show that abnormal CTG findings often coincide with elevated lactate and reduced pH, underscoring lactate's role as an indicator of metabolic stress.[14]

Normal umbilical artery lactate values typically fall within the low physiological range at birth, whereas rising levels signify that the fetus has experienced periods of inadequate oxygenation. Research evaluating intrapartum monitoring has demonstrated that newborns with reassuring CTG patterns generally have normal acid–base status at delivery, while those with persistent non-reassuring tracings are more likely to exhibit elevated lactate values. This association reinforces the importance of combining physiological monitoring with biochemical confirmation to differentiate true hypoxia from false-positive CTG findings.[15]

Lactate offers practical advantages over traditional cord pH measurement. It requires a smaller blood volume, can be processed more rapidly, and demonstrates better stability after delivery, reducing the impact of delays between sampling and analysis. Comparative studies of CTG findings with cord blood values have shown that lactate correlates well with fetal distress, particularly in cases where cardiotocography suggests compromise but pH results remain borderline. This reliability supports its use as an adjunct to electronic fetal monitoring to improve diagnostic accuracy.[16]

Lactate measurement is increasingly recognized as a clinically meaningful predictor of neonatal outcomes. Evidence from cross-sectional work in term pregnancies indicates that abnormal CTG patterns are frequently accompanied by elevated cord lactate levels, and these abnormalities correspond with higher risks of neonatal depression or low Apgar scores. As a result, many centers incorporate lactate assessment to refine decision-making during labor, particularly when CTG tracings fall into non-reassuring categories where clinical ambiguity often occurs.[17]

Correlation between CTG Findings and Umbilical Cord Blood Lactate Levels

A. Category I CTG and Normal Lactate Levels

Category I CTG patterns- characterized by a normal baseline heart rate, moderate variability, and absence of pathological decelerations- are typically associated with adequate fetal oxygenation. Evidence shows that newborns with these reassuring patterns usually have normal umbilical cord acid–base parameters at birth. In prospective analyses of suspected fetal distress, cases with CTG appearances falling within the reassuring range rarely showed significant metabolic acidosis, indicating that normal CTG is a reliable predictor of normal cord blood lactate levels.[18]

B. Category II CTG and Variable Lactate Response

Category II CTG represents an intermediate zone including tracings that are neither completely normal nor overtly pathological. These patterns often trigger clinical concern, yet their predictive ability for fetal hypoxia is inconsistent. Studies evaluating non-reassuring CTG patterns have demonstrated wide variability in corresponding cord blood lactate and pH. Many fetuses classified as having "fetal distress" based solely on CTG were found to have normal acid–base status at delivery, highlighting the high false-positive rate of CTG. This discrepancy underscores the difficulty clinicians face when interpreting intermediate CTG tracings and the need for biochemical adjuncts such as lactate estimation to distinguish true hypoxia from benign abnormalities.[19]

Research comparing CTG findings with neonatal outcomes further supports the limited specificity of Category II patterns. Although certain decelerations may raise suspicion of hypoxia, many neonates with such tracings are born with normal lactate levels, revealing that CTG alone may overestimate fetal compromise. Incorporating cord lactate

measurement in these cases helps avoid unnecessary interventions prompted by ambiguous fetal heart rate patterns.[19]

C. Category III CTG and Elevated Lactate Levels

Category III CTG patterns- marked by absent variability and recurrent or prolonged decelerations- are strongly associated with fetal metabolic acidosis. Correlation studies consistently demonstrate that severe CTG abnormalities align with elevated lactate levels and decreased cord pH, confirming their strong predictive value for intrapartum hypoxia. Investigations in both low-risk and high-risk pregnancies have shown that worsening CTG patterns correspond to progressively higher lactate concentrations, supporting CTG's role as an early warning tool when abnormal findings are persistent and unambiguous.[20]

In addition, comparative evaluations of CTG and cord lactate have noted that lactate measurement detects hypoxia more precisely than CTG in cases where Category III features are present but borderline. This biochemical confirmation enables clinicians to better estimate the degree of fetal compromise and guide timely obstetric intervention.[20]

D. Diagnostic Performance: Sensitivity, Specificity, and Clinical Value

The diagnostic performance of CTG in predicting metabolic acidosis remains limited due to low specificity. Several analyses reveal that a substantial proportion of fetuses labeled as "distressed" based on CTG findings are actually normoxic when cord blood is evaluated. By contrast, cord lactate shows a stronger relationship with neonatal morbidity indicators such as low Apgar scores or need for resuscitation. Evidence from term neonates with non-reassuring fetal heart patterns indicates that elevated lactate correlates more consistently with adverse outcomes than CTG classification alone. This supports the broader clinical practice of using lactate as a biochemical adjunct, particularly when CTG findings are indeterminate.[21]

Moreover, lactate measurement is less affected by sampling delays and yields rapid results, enhancing its diagnostic utility during labor. Its strong association with hypoxic stress allows clinicians to interpret CTG more accurately, reducing unnecessary operative deliveries and strengthening decision-making during intrapartum fetal assessment.[21]

Umbilical Cord Blood Lactate and Early Neonatal Outcome

Umbilical cord lactate has emerged as a critical biochemical marker for assessing neonatal condition immediately after birth because it reflects the extent of anaerobic metabolism during labor. Elevated lactate levels correlate strongly with intrapartum hypoxia, and several studies demonstrate that cord lactate is a more sensitive indicator of metabolic acidosis than pH alone. Comparative evaluations of cord pH and lactate in the setting of abnormal CTG patterns reveal that lactate rises earlier and more consistently when fetal oxygenation is compromised, making it a valuable tool for predicting neonatal well-being.[22]

1) **Apgar Scores**

Apgar scoring at 1 and 5 minutes provides a rapid clinical assessment of neonatal vitality. Research shows that higher cord lactate concentrations are associated with lower Apgar scores, indicating greater immediate postnatal depression. In prospective evaluations of fetal scalp lactate, neonates with elevated intrapartum lactate were significantly more likely to exhibit low Apgar scores, reinforcing lactate's role as an early indicator of physiological compromise.[23]

2) **Need for Positive Pressure Ventilation**

Newborns experiencing intrapartum hypoxia often require assisted ventilation at birth. Studies analyzing cord lactate trends report that higher lactate values correlate with increased likelihood of requiring positive pressure ventilation or resuscitative efforts in the delivery room. Elevated lactate serves as a biochemical confirmation of the stress observed during labor, helping clinicians anticipate the need for immediate neonatal support.[24]

3) **NICU Admission**

NICU admission is a key marker of early neonatal morbidity. Evidence demonstrates that neonates with elevated cord lactate are more frequently admitted to neonatal intensive care for respiratory support, monitoring, or management of suspected hypoxic injury. Higher lactate values have been shown to correspond with both the severity and duration of acute intrapartum hypoxia, thereby enabling clinicians to better stratify neonatal risk immediately after birth.[24]

4) **Respiratory Distress**

Respiratory distress shortly after birth is often a consequence of impaired gas exchange or delayed adaptation to extrauterine life. Studies evaluating relationships between fetal monitoring findings and cord gases demonstrate that elevated lactate is associated with higher rates of transient tachypnea, respiratory effort abnormalities, and the need for supplemental oxygen. This association reflects the underlying metabolic stress sustained during labor, which affects respiratory transition after delivery.[25]

5) **Hypoxic-Ischemic Encephalopathy (HIE)**

HIE occurs when prolonged fetal hypoxia leads to neurological injury. Elevated cord lactate is strongly correlated with increased risk of early neonatal neurological impairment. Evidence from observational cohorts shows that neonates with very high lactate values at birth are more likely to show symptoms such as hypotonia, seizures, or altered consciousness, suggesting significant metabolic disruption in utero. Though CTG abnormalities may indicate risk, lactate provides direct biochemical confirmation of hypoxic insult severity.[25]

6) **Neonatal Seizures and Neurological Morbidity**

Metabolic acidosis from anaerobic metabolism can impair neuronal function. Studies have documented that neonates with higher cord lactate levels are more frequently associated with early-onset seizures or abnormal neurologic examinations during the first 24 hours of life. These findings highlight lactate's predictive value in identifying infants requiring closer neurological monitoring.[25]

7) **Neonatal Mortality**

While mortality is multifactorial, elevated cord lactate serves as a strong marker of severe intrapartum compromise. Evidence suggests that neonates with extremely high lactate values at birth have a significantly higher risk of adverse outcomes, including early neonatal death, particularly when combined with low Apgar scores or signs of respiratory failure. Lactate therefore provides important prognostic information beyond what CTG or pH alone can offer.[25]

Advantages and Limitations of Lactate Monitoring

Umbilical cord lactate assessment offers several clinical advantages because it provides direct insight into fetal metabolic status at birth. One of its major strengths is its biochemical stability; lactate remains less affected by delays between sampling and analysis than pH, making it more practical in busy labor units where immediate processing may not always be feasible. It also requires a smaller volume of blood, which simplifies the procedure and decreases the likelihood of sampling error. These properties support lactate's wider utility in routine intrapartum monitoring[5]

Another important advantage is lactate's ability to function as an objective adjunct to CTG. Studies evaluating non-reassuring fetal heart rate patterns show that many fetuses classified as distressed based solely on CTG actually have normal acid–base status, contributing to unnecessary operative interventions. When lactate is incorporated into decision-making, clinicians can more accurately differentiate true hypoxia from benign abnormalities, particularly in Category II CTG tracings where interpretation is challenging.[18]

The value of lactate becomes especially clear when CTG produces borderline or ambiguous results. In such cases, adding lactate measurement improves diagnostic precision and reduces overestimation of fetal distress. Evidence indicates that lactate correlates better with fetal metabolic status than CTG alone.[19]

A consistent finding across multiple comparative studies is that elevated lactate levels strongly correlate with metabolic acidosis, reflecting the severity and duration of fetal hypoxia. [20]

Lactate is a superior predictor of early neonatal condition compared with pH. Studies have shown that elevated cord lactate more reliably corresponds with low Apgar scores, need for resuscitation, respiratory distress, and signs of neurological insult. Because lactate rises early during anaerobic metabolism, it provides clinicians with a more sensitive measure of hypoxic stress, contributing to improved assessment and risk stratification after birth.[22]

Research evaluating fetal scalp lactate further supports its clinical utility. Investigations into proposed lactate thresholds demonstrate that abnormal intrapartum lactate levels are strongly associated with adverse neonatal outcomes, reinforcing its potential as a physiologic marker that better reflects intrapartum metabolic strain.[23]

Large observational studies in term and near-term neonates have also documented that increasing cord lactate is linked to higher rates of NICU admission and early respiratory compromise. This strengthens the evidence base for incorporating lactate measurement into routine intrapartum assessment, especially where non-reassuring CTG patterns are frequently encountered.[24]

Moreover, the relationship between elevated lactate and early neurological complications- including neonatal encephalopathy and seizures- demonstrates the relevance of lactate not only as a diagnostic marker but also as a prognostic indicator for short-term neonatal morbidity. This broader clinical significance enhances its appeal as a valuable component of modern intrapartum fetal surveillance systems.[25]

Despite its advantages, lactate monitoring is not without limitations. Variability in recommended lactate cut-off values across studies may lead to uncertainty in interpreting borderline results. Additionally, maternal physiological conditions- including fever, dehydration, or metabolic disorders- may influence lactate production and complicate interpretation. These factors highlight the need for careful clinical correlation and standardized protocols when incorporating lactate into intrapartum assessment.[14]

2. Current Guidelines and Recommendations

Current international guidelines emphasize the importance of structured intrapartum fetal monitoring while acknowledging the limitations of CTG as a stand-alone tool. Evidence synthesized in major systematic reviews has shown that continuous CTG, although widely used, does not significantly reduce perinatal mortality compared to intermittent auscultation in low-risk pregnancies. However, it is associated with a higher rate of operative deliveries. These findings guide recommendations that CTG should be selectively applied based on maternal–fetal risk rather than universally used for all labors.[1]

The World Health Organization provides clear and globally applicable recommendations for intrapartum monitoring. WHO advises intermittent auscultation for women with uncomplicated pregnancies, reserving continuous CTG for those with clinical risk factors. These guidelines highlight the need for timely detection of fetal distress while maintaining maternal mobility and minimizing unnecessary interventions. WHO also underscores the importance of integrating monitoring findings with overall clinical assessment rather than relying solely on heart rate tracings.[2]

Professional societies have further refined recommendations by establishing standardized CTG interpretation frameworks. The American College of Obstetricians and Gynecologists (ACOG) advocates the three-tier system- Categories I, II, and III—to improve consistency in interpreting fetal heart rate patterns. According to ACOG, Category I patterns generally indicate normal fetal acid–base status and require no intervention, whereas Category III patterns signal a high likelihood of fetal compromise and necessitate prompt evaluation or delivery. Category II remains an indeterminate zone where additional assessment, including biochemical markers such as cord or scalp lactate, may help guide clinical decision-making.[8]

Recent evidence updates have reinforced that the predictive ability of CTG varies significantly with fetal growth status and clinical context. Research comparing fetal heart rate tracings among small-for-gestational-age and appropriate-for-gestational-age infants demonstrates differing risk profiles even with similar CTG appearances. These findings support guideline recommendations that monitoring decisions should incorporate fetal growth patterns, maternal conditions, and labor progression rather than relying solely on electronic tracings.[9]

Together, these guidelines stress a balanced approach: CTG remains an important surveillance tool, but interpretation must be contextualised with clinical judgment and supplemented with objective markers when needed. Lactate measurement, although not mandated universally in guidelines, is increasingly acknowledged as a valuable adjunct for assessing fetal metabolic status when CTG patterns are ambiguous or suggest developing hypoxia.

3. Discussion

The relationship between intrapartum fetal monitoring findings and umbilical cord blood lactate has been explored extensively across multiple clinical studies, with consistent evidence demonstrating that CTG abnormalities correlate with biochemical indicators of hypoxia. In the study by Yusuf & Fatima (2020)[10] abnormal CTG patterns were associated with lower cord pH at birth, suggesting that electronic fetal monitoring can provide early warning of metabolic compromise, though with limited specificity. This aligns with findings from Markam & Ghanghoria (2024)[11,] who observed that high-risk pregnancies with suspicious CTG tracings frequently demonstrated abnormal neonatal outcomes, further reinforcing CTG's role as an initial screening tool rather than a definitive diagnostic modality.

The predictive value of CTG becomes clearer when interpreted alongside cord blood parameters. In Paladugu et al. (2023)[12,] classification of intrapartum fetal distress correlated strongly with immediate postpartum acidemia, demonstrating that biochemical confirmation adds critical clarity in evaluating fetal well-being. Similarly, Mushtaq et al. (2023)[13] identified that non-reassuring CTG patterns often corresponded with significantly lower cord pH and higher lactate levels, confirming that metabolic disturbance is more accurately detected when CTG and biochemical markers are assessed together.

Additional insights from Rajpoot et al. (2023)[14] highlight that non-reassuring fetal heart rate patterns are strongly associated with adverse perinatal outcomes, but a substantial proportion of cases still demonstrate normal cord pH. This again illustrates the high false-positive rate inherent to CTG-based interpretation. The study by Singh et al. (2022)[15] further supports this, reporting that many neonates classified as "distressed" on CTG were born with normal biochemical profiles, underscoring the need for objective confirmation.

The integration of biochemical assessment through lactate measurement strengthens intrapartum decision-making. Sharmin et al. (2022)[16] noted that cord lactate and pH provided clearer delineation of true fetal compromise compared to CTG alone, with lactate showing stronger correlation with perinatal morbidity. Similar findings were echoed by Sethia et al. (2022)[17,] who demonstrated that severe CTG abnormalities were consistently associated with elevated lactate levels, validating cord lactate as a highly reliable biochemical indicator.

The reliability of CTG as an independent predictor was further challenged by Gupta et al. (2022)[18,] who observed that a considerable proportion of fetuses with CTG-suspected distress actually had normal cord pH, reinforcing the need for adjunct biochemical testing. Supporting this, Kanagal & Praveen (2022)[19] showed that combining CTG interpretation with cord lactate significantly improved prediction of early neonatal outcomes in their cohort.

The role of lactate becomes particularly important in low-risk pregnancies, where false-positive CTG readings may lead to unnecessary interventions. Deshpande et al. (2019)[20] found that cord lactate levels provided a more accurate reflection of fetal condition than CTG in non-high-risk mothers, indicating that incorporating lactate can prevent overdiagnosis of fetal distress. In addition, Ananya et al. (2020)[21] demonstrated that cord lactate correlated more strongly with morbidity indicators than cord pH, reinforcing lactate's utility as a superior metabolic measure.

Recent evaluations provide further clarity regarding lactate thresholds. Mukhopadhyay et al. (2024)[22] reported that lactate identified fetal acidemia more accurately than pH in cases of abnormal CTG, supporting its role as an advanced diagnostic tool. The prospective study by Iorizzo et al. (2022)[23] proposed specific scalp lactate cutoffs associated with neonatal outcomes, offering evidence-based thresholds that can enhance intrapartum decision-making when CTG is ambiguous.

Expanding on the prognostic value of lactate, Badmus et al. (2024)[24] demonstrated strong associations between elevated cord lactate and early neonatal complications, including respiratory distress and need for NICU admission, showing that lactate not only indicates intrapartum hypoxia but also predicts short-term morbidity. Finally, Nabiyal et al. (2025)[25] confirmed that both CTG abnormalities and cord gases predict adverse neonatal outcomes, but lactate showed a more consistent correlation than pH, strengthening the argument for its incorporation into routine intrapartum monitoring.

Together, these findings illustrate a clear pattern:

- CTG is sensitive but not specific, resulting in frequent false alarms.
- Lactate is specific and correlates strongly with true fetal hypoxia and neonatal morbidity.
- Combining CTG with lactate markedly improves diagnostic accuracy, particularly for Category II tracings where clinical uncertainty is greatest.

This synthesis supports a model of intrapartum care in which CTG serves as an initial screening tool, while biochemical testing- especially lactate- provides definitive confirmation of fetal metabolic status.

4. Conclusion

Effective intrapartum fetal monitoring remains essential for safeguarding neonatal well-being, yet long-standing reliance on cardiotocography alone has demonstrated important diagnostic limitations. Although CTG provides continuous physiological insight into fetal heart rate patterns, its high false-positive rate often leads to unnecessary interventions and does not reliably distinguish between true fetal hypoxia and benign variations. This gap underscores the need for objective biochemical markers that more accurately reflect fetal metabolic status.

Umbilical cord blood lactate has emerged as a robust and practical indicator of intrapartum hypoxic stress. Unlike pH, which may fluctuate due to buffering systems or sampling delays, lactate rises consistently in response to anaerobic metabolism and correlates strongly with the severity and duration of fetal compromise. Its rapid measurement, stability after sampling, and minimal technical requirements make it an accessible adjunct even in resource-limited settings.

The synthesis of available evidence demonstrates that integrating cord lactate assessment with CTG interpretation substantially enhances diagnostic accuracy. Lactate provides biochemical confirmation when CTG tracings are indeterminate, helps differentiate true fetal distress from false alarms, and strengthens clinical decision-making, particularly in high-risk or ambiguous intrapartum scenarios. Elevated lactate levels also align more reliably with early neonatal outcomes, including low Apgar scores, need for resuscitation, respiratory difficulties, and neurological complications.

Overall, combining physiological monitoring with biochemical assessment represents a more comprehensive and evidence-based approach to evaluating fetal well-being during labor. As clinical understanding and technology evolve, standardization of lactate thresholds and broader incorporation of biochemical markers into intrapartum protocols may further optimize outcomes. The integration of CTG with cord lactate measurement ultimately offers a balanced, accurate, and clinically meaningful strategy for ensuring timely intervention and improving neonatal health.

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