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Full length article

International expert consensus statement on physiological interpretation of cardiotocograph (CTG): First revision (2024)

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ABSTRACT

The first international consensus guideline on physiological interpretation of cardiotocograph (CTG) produced by 44 CTG experts from 14 countries was published in 2018. This guideline ensured a paradigm shift from classifying CTG by arbitrarily grouping certain features of the fetal heart rate into different “categories”, and then, randomly combining them to arrive at an overall classification of CTG traces into “Normal, Suspicious and Pathological” (or Category I, II and III) to a classification which is based on the understanding of fetal

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Relative utero-placental insufficiency of labour (RUP-L)
Suggestive of Fetal Inflammation (SOFI)

pathophysiology. The guideline recommended the recognition of different types of fetal hypoxia, and the determination of features of fetal compensatory responses as well as decompensation to ongoing hypoxic stress on the CTG trace. Since its first publication in 2018, there have been several scientific publications relating physiological interpretation of CTG, especially relating to features indicative of autonomic instability due to hypoxic stress (i.e., the ZigZag pattern), and of fetal inflammation. Moreover, emerging evidence has suggested improvement in maternal and perinatal outcomes in maternity units which had implemented physiological interpretation of CTG. Therefore, the guideline on Physiological Interpretation of CTG has been revised to incorporate new scientific evidence, and the interpretation table has been expanded to include features of chorioamnionitis and relative utero-placental insufficiency of labour (RUP-L).

Introduction

The first international expert consensus guideline on Physiological Interpretation of cardiotocograph (CTG) was produced by 44 CTG experts from 14 countries in 2018 [1]. This ensured a paradigm shift in classifying CTG traces by grouping the pre-determined features of the CTG trace into different “categories” often with unscientific time limits, and then, randomly combining them to arrive at an “overall classification” CTG traces into “normal, suspicious, pathological” (or Category I, II or III) categories [2–5]. Instead, the international consensus guideline on Physiological CTG interpretation advocated the classification of CTG traces based on the recognition of different types of fetal hypoxia and assessing the fetal responses to ongoing stress by differentiating features suggestive of fetal compensation from decompensation [1]. The interpretation tools recommended by this international expert consensus statement on physiological interpretation of CTG to aid interpretation of observed fetal heart rate (FHR) changes were aimed at individualisation of care. This should be done by use of the “Fetal Monitoring checklist” to determine whether if THIS fetus was “fit” to undertake the progressively hypoxic journey of labour at the beginning of recording. Once pre-existing fetal compromise has been excluded by this checklist, then, determining the types of fetal hypoxia and the central organ oxygenation (“How is THIS fetus?”) during labour by the use of “Intrapartum Fetal Assessment Tool” was recommended [1].

The above principles of Physiological interpretation of CTG traces have been implemented in more than 20 maternity units in the UK, and several hospitals in Spain, Belgium, France, Italy, Australia, Denmark, Estonia, Switzerland, Lithuania, Romania, Sri Lanka, China, Singapore, Oman and the United Arab Emirates, and several hospitals have demonstrated a reduction in the rate of intrapartum-related hypoxic-ischaemic encephalopathy (HIE), and the rate of emergency caesarean sections for suspected fetal compromise [6,7].

What is the key driver behind the revision of the international expert consensus guideline on physiological interpretation of CTG (IEPIC)?

The first version of this guideline was aimed at recognising different types of fetal hypoxia and determining fetal compensatory responses to ongoing intrapartum mechanical and hypoxic stresses to help improve perinatal outcomes and /or to reduce unnecessary intrapartum operative interventions for women. Since the publication of this guideline in 2018, there have been emerging scientific evidence highlighting the different concepts of physiological CTG interpretation [8], including the ZigZag Pattern [9,10], fetal heart rate cycling [11], features suggestive of chorioamnionitis and inflammation [12–16]. Moreover, some recent animal experimental studies have questioned the role of baroreceptors in the causation of fetal heart rate decelerations [17]. In addition, there have been scientific publications highlighting the importance of “higher than expected baseline fetal heart rate” [18], perinatal outcomes in different types of fetal hypoxia [19,20], and correlation of different types of hypoxia with neurological outcomes [21]. Eventually the CTG features and the pathophysiology of a subtype of hypoxic stress arising at the onset of regular uterine activity have been described under the

definition of RUP-L (Relative Utero-Placental Insufficiency of Labour) [22].

The following changes which are highlighted in this revised International Expert Consensus Statement on Physiological Interpretation of CTG (IEPIC) will replace and supersede the first version of the guideline published in 2018 [1]. However, this revision must be used in conjunction with the original guideline to understand the principles of physiological CTG interpretation (Supplement 1).

a. Mechanisms of fetal heart rate decelerations

The international expert consensus group noted the ongoing controversy due to some researchers who predominantly conduct animal experimental studies questioning the role of baroreceptors in the causation of decelerations [17,23]. This is despite the same research group having stated earlier that baroreceptors do play an initial role in fetal heart rate decelerations, but they are soon overwhelmed by peripheral chemoreceptors [24]. The panel felt that the experimental animal studies which attempt to cause umbilical cord compression by occluding a loop of the umbilical cord with a silicone ring in fetuses subjected to a general anaesthetic and intrauterine invasive procedures to monitor the vital parameters do not truly reflect what really happens during human labour. It has been shown that with the onset of uterine contractions, due to the compression of the placental sinuses, there is a bolus of blood reaching the fetus leading to an increase in fetal oxygen saturation [25]. This initial bolus of increased blood volume at the beginning of uterine contractions is very likely to increase fetal cardiac output, increasing the systemic blood pressure, with the activation of baroreceptors which caused a sudden and an abrupt drop in the fetal heart rate. It is obvious that the isolated compression of the umbilical cord which is performed during experimental animal studies will not have this initial increase in the blood volume and resultant increase in blood pressure, giving the erroneous impression that all decelerations are mediated by peripheral chemoreceptors. This potential confounding effect has been recently highlighted [25]. Moreover, the arguments regarding which receptors mediate the drop in the FHR do not help frontline clinicians who need to understand the underlying mechanisms so that the ongoing stress can be alleviated to improve perinatal outcomes [26]. Based on available data and the reasoning above, the panel concluded that unnecessary academic arguments regarding the receptors with those who conduct animal experimental studies will take the focus away from real-life clinical practice. Therefore, the panel has removed the reference to “baro-receptor” and “chemo-receptor” mediated decelerations, and has simply recommended the classification of decelerations into two types based on the likely underlying cause.

The panel recognises the historical obstetric practice of classifying fetal heart rate decelerations based on the morphology, duration and in relation to the uterine contractions. It is important to appreciate that morphology of observed decelerations (eg., early, variable, late, typical, atypical etc) have been reported to have no correlation with poor perinatal outcomes. Therefore, the panel strongly recommends that the intervening baseline between ongoing decelerations must be scrutinised to determine its stability and the presence of reassuring variability and continuing cycling to determine fetal response to ongoing intrapartum

hypoxic stress. Nonetheless, the panel appreciates that some clinicians, due to the continuing influence of traditional obstetric teaching, may wish to determine the morphology of decelerations, until they develop complete confidence in the principles of physiological CTG interpretation. Therefore, the panel has opted to include two morphological types of decelerations, based on the likely underlying pathophysiological mechanisms.

Any deceleration which has an abrupt drop from the baseline (>30 bpm), reaching the nadir within 30 s from the onset of the decelerations, and demonstrating a quick recovery to the baseline may be termed a “Quicklie” (Fig. 1). These are believed to be due to the compression of umbilical cord, and resultant transient hypoxaemia, and not due to hypoxia and/or acidosis. The intervening baseline and variability should be assessed to determine the oxygenation of the central organs. If such “quicklie” decelerations are associated with an increase in the baseline FHR (i.e., catecholamine surge), then changes in maternal position and/or reducing the rate of oxytocin infusion may help restoring the baseline to normal.

Any deceleration which has a gradual drop from the baseline, and then recovers slowly to the baseline even after the cessation of uterine contractions may be termed “tardy” (Fig. 2). These “tardy” decelerations are due to an ongoing utero-placental insufficiency and may be associated with acidosis if they are associated with a reduced baseline variability. These “tardy” decelerations are often due to a structural damage to the placenta (e.g., infarction, thrombosis or an abnormal placenta), and therefore, cannot be reversed by changes in the maternal position or administration of fluids to the mother.

Important Note

Regarding the morphology of FHR decelerations, the international expert consensus group recommends that with evolving understanding and confidence in physiological CTG interpretation, clinicians should move away from identifying the morphology of decelerations but assess the intervening baseline FHR for stability, reassuring variability and cycling to determine fetal wellbeing”.

- b. Change in terminology for excessive baseline variability due to a rapidly evolving hypoxia: The ZigZag pattern

Increased variability was referred to “saltatory pattern” which is a general term used to describe an increased baseline variability lasting > 25 bpm lasting for at least 30 min [9]. However, saltatory pattern was found to be very rare ($<5\%$) during labour [27,28], most likely because due to intermittent interruption of fetal oxygenation due to ongoing uterine contractions, it is not possible to have such “uniform” increased bandwidth lasting for 30 min. Gracia Perez-Bonfils proposed to differentiate the use of “saltatory pattern” to refer to a uniform increase in the bandwidth lasting for more than 30 min, which is mostly due to an antenatal acute and profound (non-fatal), hypoxic-ischaemic insult, from the “ZigZag” pattern to refer to an abrupt and erratic up and down fluctuation of the baseline FHR variability (>25 bpm). The latter occurs when the intensity of hypoxic stress increases with insufficient time available at the baseline to ensure adequate gas exchange, and such an erratic fluctuation of baseline FHRV >25 bpm lasts for at least 1 min [9]. It has been reported that the ZigZag pattern persisting for more than 2 min is associated with approximately 11-fold increase in the admission to the neonatal unit [9,10].

Subsequently, it has been reported that marked increased variability lasting for more than one minute during labour was associated with a two-fold increase in neonatal acidosis [29].

Although, the exact mechanism for the ZigZag pattern (Fig. 3) is unknown, it is considered to be due to an autonomic instability, and recent animal experimental studies have suggested that it is predominantly mediated by the parasympathetic nervous system [30].

A ZigZag pattern persisting for more than 1 min requires immediate action to improve fetal oxygenation (reducing or stopping oxytocin infusion and /or administering a tocolytic). If ZigZag pattern is observed with a subacute hypoxic pattern during active maternal pushing, then, immediate cessation of active, directed pushing is recommended to rapidly improve fetal cerebral oxygenation through the carotid arteries [13–16]. If the ZigZag pattern is seen with an increase in the baseline FHR without repetitive decelerations [13–16], then, this should raise the suspicion of fetal neuroinflammation in the context of chorioamnionitis, then, continuing super-imposed hypoxic stress should be avoided to reduce the likelihood of neonatal encephalopathy (NNE).

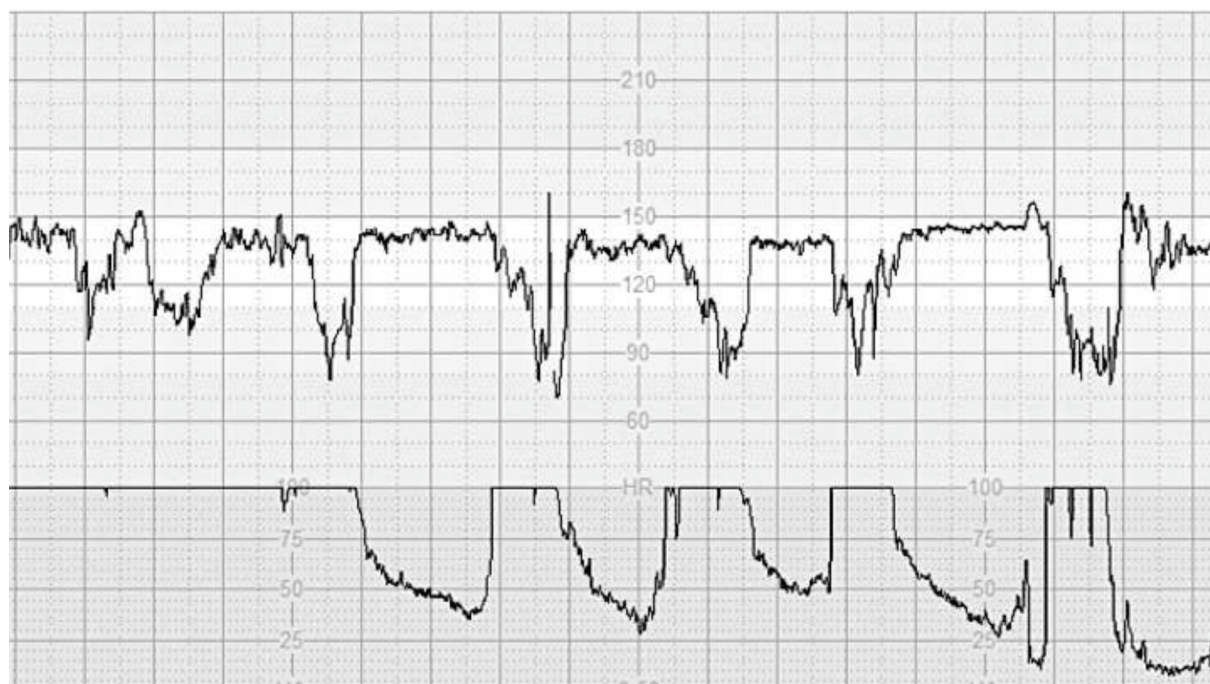


Fig. 1. “Quicklie” Deceleration.

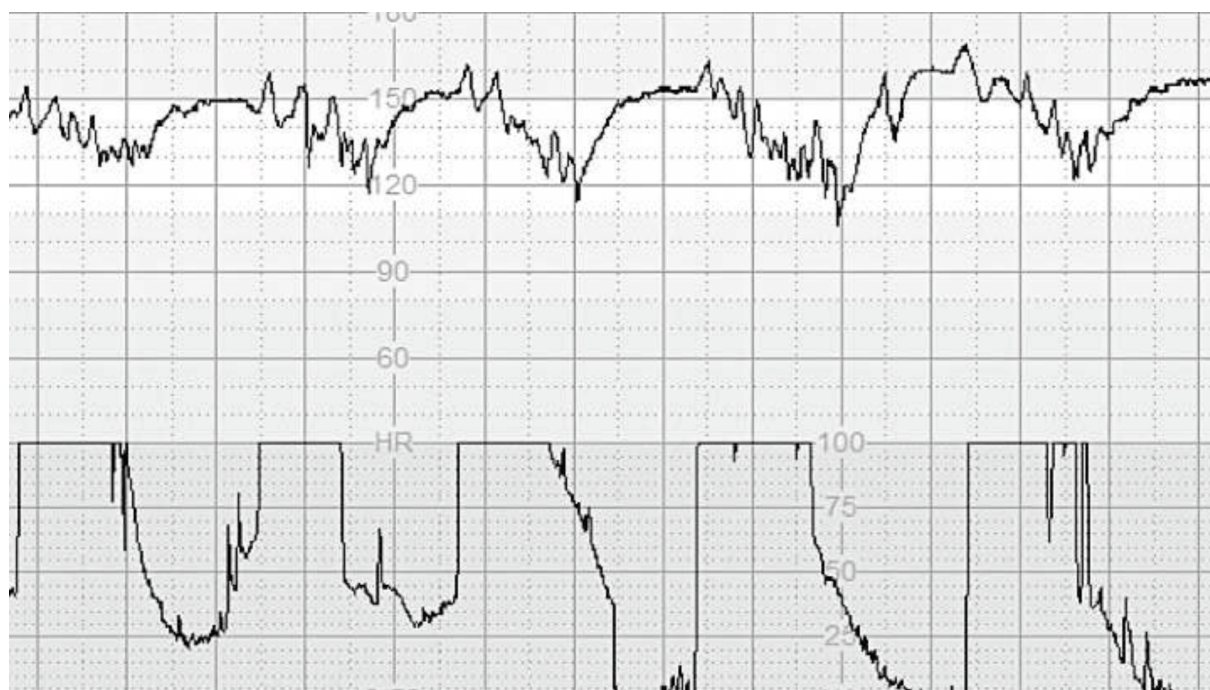


Fig. 2. “Tardy” Deceleration.

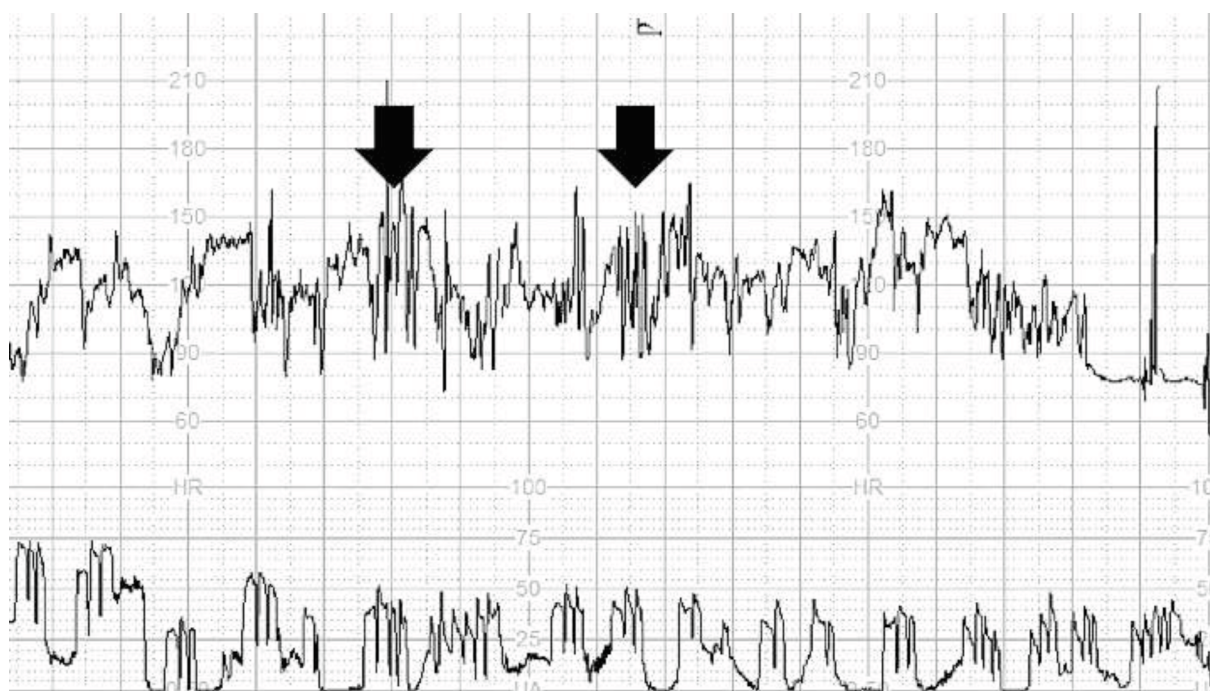


Fig. 3. “ZigZag” Pattern.

c. Features suggestive of fetal inflammation (SOFI) to recognise chorioamnionitis (intraamniotic inflammation and/or infection)

An increase in the baseline FHR by $> 10\%$ without preceding deceleration and/or a baseline FHR $> 10\%$ higher than what is expected for the gestational age should be considered as SOFI [13–16]. Recently, it has been shown that the interleukin-6 (IL-6) levels in the umbilical artery at birth is approximately five-fold higher in fetuses with $> 10\%$ increase in the baseline FHR without repetitive, preceding decelerations [16]. Furthermore, absence of fetal heart rate cycling was also

associated with approximately 4-fold increased prevalence of maternal pyrexia [11]. Recent evidence has shown that in the presence of neuroinflammation (absence of cycling, ZigZag Pattern or sinusoidal patterns) the IL-6 levels in the umbilical cord increase by approximately 4-fold, compared to fetuses with $> 10\%$ increase in the baseline FHR alone [16]. In addition, increased IL-6 levels were associated with a significant increase in the composite adverse outcomes (poor neonatal condition at birth, admission to neonatal unit or special care baby unit), and fetuses with SOFI contributed to approximately 30 % of all cases of CAO (composite adverse outcomes).

Table 1
Intrapartum CTG Classification Tool.

Hypoxia	Features	Management
No Hypoxia	- Baseline appropriate for G.A. and stable - Normal FHR variability and presence of cycling - No repetitive decelerations	- Consider whether the CTG needs to continue. - If continuing the CTG perform routine hourly review to determine the onset of hypoxic or inflammatory/ non-hypoxic stress (see below)
Evidence of Hypoxia		
Chronic Hypoxia	- Higher baseline than expected for G.A. - Reduced variability and/ or absence of cycling - Absence of accelerations - Shallow decelerations - Consider the clinical indicators: reduced fetal movements, thick meconium, bleeding, evidence of chorioamnionitis, post maturity, IUGR	Avoid further hypoxic stress: consider tocolysis if a delay is anticipated to accomplish birth (e.g., operating theatre busy) of if there is evidence of progressive reduction in the baseline FHR. Expedite delivery, if birth is not imminent.
RUPI-L	- A sudden increase in the FHR immediately after the onset of established contractions/ induction of labour - ZigZag pattern and/or widening /deepening of decelerations	Consider the overall clinical context including background risk factors to determine if birth should be expedited.
	Compensated	- Likely to respond to conservative interventions - Regular review every 30–60 min to assess the resolution of the catecholamine response or increased time spent on the baseline FHR. - The wider clinical context such as reduced placental reserve, stage and the rate of progress of labour, presence of meconium or co-existing chorioamnionitis MUST be considered and managed accordingly.
Gradually Evolving Hypoxia	Rise in the baseline (with normal variability and stable baseline) preceded by decelerations and loss of accelerations, with inter-deceleration interval greater than the time spent during decelerations Decompensated - Reduced or increased variability (ZigZag pattern), preceded by repetitive decelerations and an increase in the baseline FHR. - Unstable/ progressive decline in the baseline FHR (step ladder pattern to death)	- Needs urgent intervention to reverse the hypoxic stress (remove prostaglandin pessary, stop oxytocin infusion, and/ or administer a tocolytic) - Delivery should be expedited, if no signs of improvement (restoration of stable baseline FHR and normal variability) are seen
Subacute Hypoxia	More time spent during decelerations (>90 s) than at the baseline (<30 s) May be associated with the “ZigZag” pattern (increased variability) lasting for > 1 min	First Stage - Remove prostaglandins/stop oxytocin infusion - If no improvement is seen, needs urgent tocolysis - If no evidence of improvement within 10–15 min of the above measures, review the overall clinical context, and expedite delivery, if appropriate. Second Stage - Stop oxytocin infusion and stop maternal active pushing during contractions until improvement is noted. - If no improvement in noted, consider tocolysis if delivery is not imminent or expedite delivery by operative vaginal delivery
Acute Hypoxia	Prolonged Deceleration (>3 min)	Preceded by reduced variability and lack of cycling or reduced variability within the first 3 min Immediate delivery by the safest and quickest route Preceded by normal variability and cycling and normal variability during the first 3 min of the deceleration High chance of recovery – see 3 min rule below - Exclude the 3 intrapartum irreversible accidents (i.e. umbilical cord prolapse, placental abruption, uterine rupture – if such an accident is suspected prepare for immediate delivery) - Correct the reversible causes (uterine hyperstimulation/hypertonus, maternal hypotension and sustained umbilical cord compression) - If no improvement by 9 min or any of the accidents diagnosed, immediate delivery by the safest and quickest route
Chorioamnionitis (SOFI)	>10 % increase in the baseline FHR without any repetitive preceding decelerations Neuroinflammation = loss of cycling, ZigZag or sinusoidal patterns	- Consider the overall clinical context including parity and the stage of labour and the rate of progress of labour - In the presence of features of neuroinflammation, expedite birth to avoid the detrimental effects of superimposed hypoxia on the background fetal systemic inflammatory response syndrome (FIRS)
Other Abnormal CTG Patterns	(Double Mountain Peak Sign, Poole Shark Teeth Pattern, Typical Sinusoidal Pattern, uncertain / unstable baseline)	- Escalate to senior team – exclude erroneous recording of maternal heart rate and other non-hypoxic causes such as feto-maternal haemorrhage or chronic fetal anaemia and acidosis as well as fetal cardiac arrhythmias and heart blocks. - Consider the application of a Fetal Scalp Electrode (FSE) to improve signal quality if there is evidence of poor quality recording.

Based on this new scientific evidence since the publication of the last guideline, the international consensus group has included “chorioamnionitis” as an additional parameter in the classification of CTG (Table 1). This term encompasses both intraamniotic infection and/or inflammation due to an ascending infection from the maternal genital tract as well as transplacental passage of infection /inflammatory mediators from the maternal compartment. Based on the published scientific evidence, birth should be expedited if features of neuroinflammation is observed on the CTG trace (Figs. 4 a&b). A scoring system (the “Chorio Duck Score”) has been recently published to help recognise ongoing chorioamnionitis and to enable timely and appropriate action [15]. Although, a Chorio Duck Score > 5 may be used as a clinical guide to timely recognise ongoing chorioamnionitis, evidence from large studies confirm its effectiveness is required prior to recommending this in routine clinical practice.

d. Relative utero-placental insufficiency of labour (RUPI-L)

The international expert consensus group recognised that some fetuses may present with a relative utero-placental insufficiency at the onset of regular uterine activity and may not show any abnormalities in the features of the FHR in the absence of uterine contractions. This relative utero-placental insufficiency may be due to a reduced ratio between placental supply and fetal demand due to a sub clinically impaired placental function [22]. This imbalance might not produce overt manifestations before labour (such as fetal growth restriction or features of chronic hypoxia at antepartum CTG) but it is unmasked only by the onset of regular uterine activity. The onset of regular uterine activity may further diminish the oxygen supply to these fetuses affected by subclinical placental insufficiency because uterine contractions cause intermittent reductions of the perfusion of the uteroplacental bed. Therefore, with the onset of regular or strong uterine contractions (e.g., induction of labour or established labour), these fetuses start manifesting abnormal fetal heart rate patterns which reflect the attempt to compensate the hypoxic stress and maintain adequate perfusion to essential central organs during episodes of transient reduction in oxygenation. The most commonly observed FHR changes on the CTG trace in fetuses with RUPI-L are represented by:

- Wide and deep decelerations as soon as regular uterine activity – either spontaneous or secondary to the use of oxytocin or administration of prostaglandins – begins.
- The decelerations disappear or reduce their width and depth as uterine contractions decrease in intensity and frequency (Figs. 2 and 3).
- Fetal heart rate baseline between fetal decelerations commonly on the upper limit of the normal range. This occurs as a result of the chronic release of adrenal-derived catecholamines in fetuses with a long-standing exposure to subclinical hypoxia. > 10 % increase in the baseline FHR expected for the given gestational age compared to the previous recording and/or > 150 bpm at 41 weeks or > 140 bpm at 42 weeks of gestation should be considered as abnormal for the given fetus.
- Periods of abruptly increased fetal heart rate variability > 25 bpm lasting between one to ten minutes – i.e., “Zig-Zag” pattern – may occur in cases of rapidly evolving hypoxic stress. The exclusive parasympathetic control on the fetal heart leads to the instability of the heart rate pulses and this could result in intermittent oscillations of baseline > 25 bpm

It is essential scrutinise the CTG trace and timely recognise RUPI-L so that fetal decompensation can be avoided by modifying stress or by expediting birth. For specific FHR patterns suggestive of RUPI-L, the reader may wish to read the recent Commentary on RUPI-L (<https://obgyn.onlinelibrary.wiley.com/doi/epdf/https://doi.org/10.1111/aogs.14937>)

e. Interpretation of antenatal CTG traces

The international expert consensus group noted the publications on the role of computerised analysis of cardiograph to determine the short-term variability (STV) during the antenatal period. However, the international expert consensus group on physiological interpretation of CTG emphasizes the importance of considering a range of conditions including inflammation, feto-maternal haemorrhage, chronic fetal anaemia and acidosis which may contribute to fetal compromise during the antenatal period, and these may not be detected by the computerised

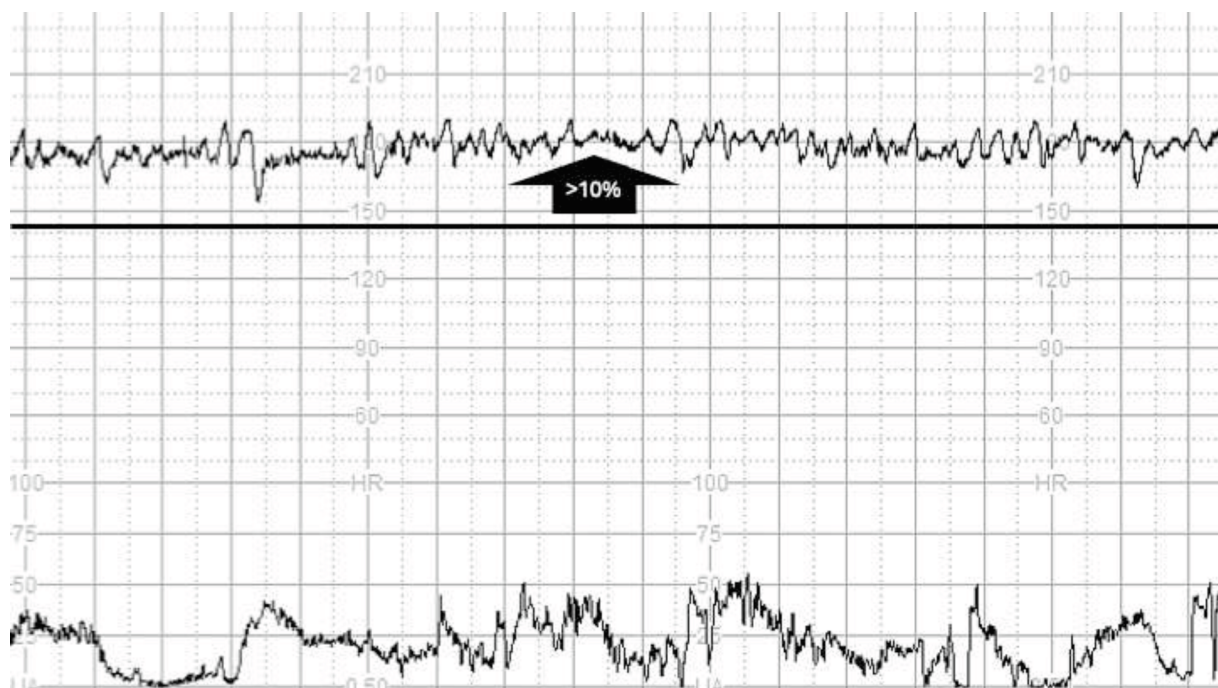


Fig. 4a. SOFI → 10 % increase in the baseline FHR without preceding deceleration and ongoing myometrial irritability.

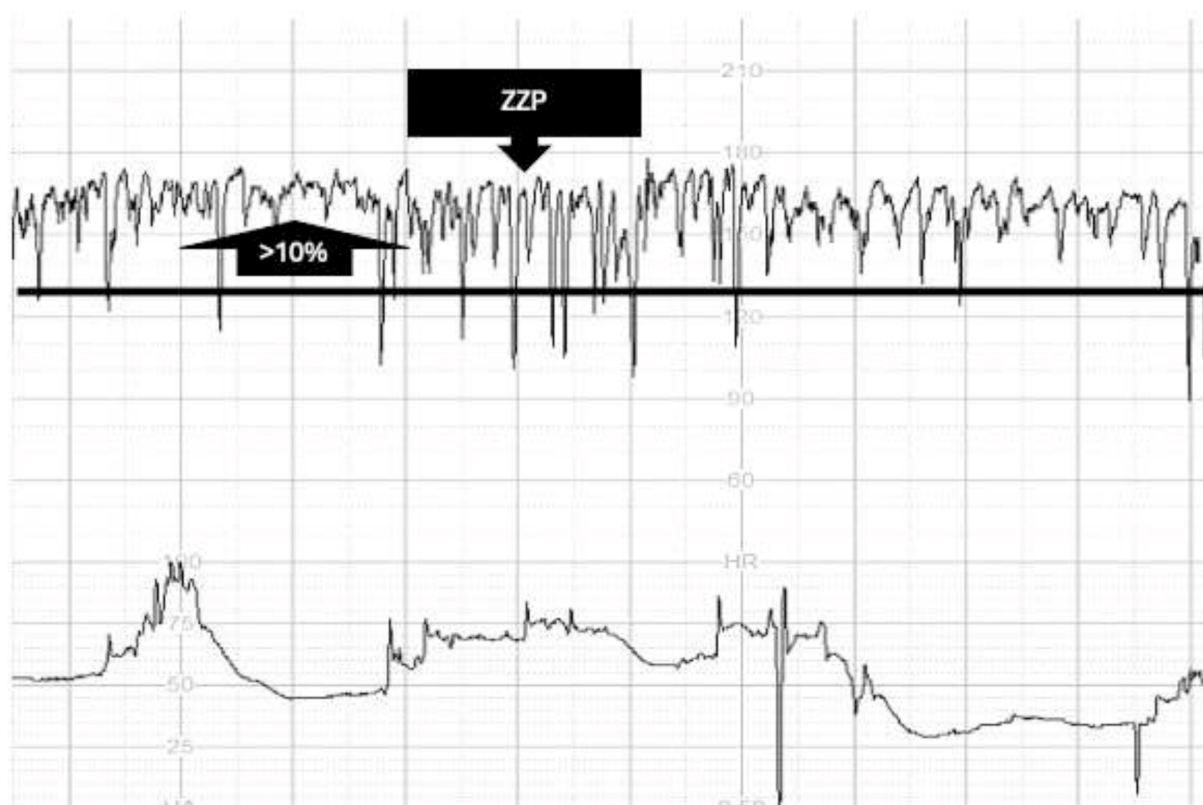


Fig. 4b. SOFI → > 10 % increase in the baseline FHR without preceding deceleration and the presence of the “ZigZag” pattern and ongoing absence of cycling.

CTG. It is important to appreciate that the expected features observed during the intrapartum period such as repetitive decelerations may not be observed before labour due to the absence of ongoing regular or intense uterine contractions. The use of the “CAUTION Checklist” [31] has been proposed as a guide to considering the wider clinical context whilst interpreting CTG traces during the antenatal period, even if the STV is within the normal range for the given gestational age (Table 2). In settings where a computerised antenatal CTG software package is not available, use of the CAUTION checklist is recommended without any reference to STV. See (Fig. 5).

f. Fetal Monitoring checklist

The fetal monitoring checklist which has been recommended in the guideline (2018) to recognise features of chronic hypoxia and pre-existing fetal compromise [32], to ask the question “Is THIS fetus FIT to undertake a progressive hypoxic journey of labour?” has been amended to include chorioamnionitis and RUPI-L (Table 3).

g. Intrapartum fetal Assessment Tool: “How is THIS Fetus?”

If the fetus is deemed “FIT” to withstand the anticipated hypoxic stresses during labour, then, it is important to recognise any new onset of an intrapartum hypoxic or inflammatory stress by the use of the intrapartum fetal assessment tool (Table 4). This tool has been revised to help recognise the features of non-hypoxic causes of fetal compromise. It is important to appreciate that there may be a combination of different types of intrapartum hypoxia with progressively increasing hypoxic stress. For example, a gradually evolving hypoxia may become a subacute hypoxia with the onset of active maternal pushing. The tool has been modified to include the initial heart rate to facilitate the easy recognition of > 10 % increase in the FHR, and to help recognise fetal hypoxic stress superimposed on an ongoing fetal inflammation.

h. Recognition of the “Double Mountain Peak Sign” to recognise erroneous monitoring of the maternal heart rate as fetal heart rate

Large amplitude accelerations coinciding with uterine contractions (the “Double Mountain Peak sign”) or a sudden drop in the observed baseline FHR, sudden disappearing of FHR, a sudden improvement in the baseline FHR variability or disappearance of decelerations may indicate erroneous monitoring of maternal heart rate as FHR [33–35]. In such cases, oxytocin infusion/ active maternal pushing should be immediately stopped until fetal heart rate is appropriately identified (by the use of maternal pulse oximetry, ultrasound scan or application of fetal scalp electrode).

Recently, the use of maternal pulse oximetry, and simultaneous recording of the maternal heart rate has been emphasised to avoid erroneous recording of the maternal heart rate as fetal heart rate [36,37].

i. Clinical practices which are NOT recommended

Fetal scalp blood sampling (FBS)

In addition to repetitive Cochrane Systematic Reviews from 2007, 2013, and 2017 [38] concluding that fetal scalp blood sampling (FBS) did not improve long term perinatal outcomes or reduce intrapartum operative interventions, subsequent studies have shown that repetitive fetal blood sampling increased operative interventions without improving perinatal outcomes [39]. Moreover, a multi-centre study in the UK concluded in 2019 that FBS did not improve perinatal outcomes, but it increased the rate of emergency caesarean section by approximately 60 % [40]. The only randomised controlled trial published so far, which directly compared FBS to assess the lactates and CTG with CTG monitoring alone, (The Flamingo Trial) has also failed to show any

Table 2
The Fetal Monitoring Checklist” Is THIS Fetus FIT to undertake the progressive hypoxic journey of labour?”

Antenatal CTG Tool The CAUTION checklist to detect Antenatal Fetal Compromise.				
Antenatal History:				Sig 2
<u>C</u> ycling absent	YES	NO	Depression of the CNS	
<u>A</u> ccelerations absent	YES	NO	Depression of the somatic NS	
<u>U</u> nstable baseline	YES	NO	Myocardial decompensation	
<u>T</u> ardy recovery (late decelerations)	YES	NO	Utero-placental insufficiency	
<u>I</u> rritability of the uterus/ Inappropriate baseline for gestational age	YES	NO	Potential abruption or chorioamnionitis	
<u>O</u> bvious history: vaginal bleeding, PPROM, reduced fetal movement, abdominal pain	YES	NO	Underlying pathology that may contribute to fetal compromise	
<u>N</u> on-hypoxic features: Zig-zag pattern or sinusoidal	YES	NO	Feto-maternal haemorrhage, chronic fetal anaemia or CNS irritability	
Date and time				
Print name and sign	1)	2)		

benefit of FBS [41]. Therefore, based on current evidence, the risks of FBS outweigh its benefits [42–44]. Therefore, the clinical guideline development group recommends that FBS should no longer be used in clinical practice.

Fetal electrocardiograph (Fetal ECG) / ST-analyser (STAN)

The international expert consensus group noted that the use of fetal CTG (ST-Analyser or STAN) holds a promise due to its reliance on cardiac physiology and the timely recognition of the negative energy balance within the myocardium. However, after reviewing the recent

Table 3
Intrapartum Fetal Monitoring Tool “How is THIS Fetus”?

Fetal Monitoring Checklist: Is THIS Fetus Fit for Labour? Pereira&Chandrarahan 2017			Assessment	
CTG Features / Risk Factors				
1	Baseline fetal heart rate <i>stable and appropriate</i> for the gestational age.	Yes	No	
2	Normal variability and cycling	Yes	No	
3	Presence of <i>TRUE</i> accelerations (not in labour or latent phase of labour)	Yes	No	
4	No shallow/ tardy decelerations	Yes	No	
5	Consider the wider clinical picture: meconium, pyrexia, fetal growth restriction, reduced fetal movements, gestational DM. pre-eclampsia, induction/augmentation, other	Yes	No	
Overall Impression: Normal/ Chronic Hypoxia/ Chorioamnionitis /RUPI/ Other:				
Management Plan:				
Date Time. Name. Signature.				

systematic review and a meta-analysis, which had included all nine RCTs on STAN and has questioned its usefulness in reducing intrapartum operative interventions [45], the use of STAN with the current CTG guideline table (“Normal, Intermediary, Abnormal”) is not recommended. It has been suggested that STAN may be beneficial if a physiological approach is used for CTG/STAN guideline [46,47]. The international expert consensus group will review this recommendation once the physiological CTG/STAN guidelines are fully implemented, and if the emerging scientific evidence after the implementation of the physiological approach confirms the benefits of STAN in reducing intrapartum operative interventions and/or an improvement in perinatal outcomes.

Administration of fluids or oxygen to the mother to correct abnormal FHR changes

Maternal fluids should only be administered to correct abnormalities in the maternal circulation (dehydration, hypotension, sepsis,

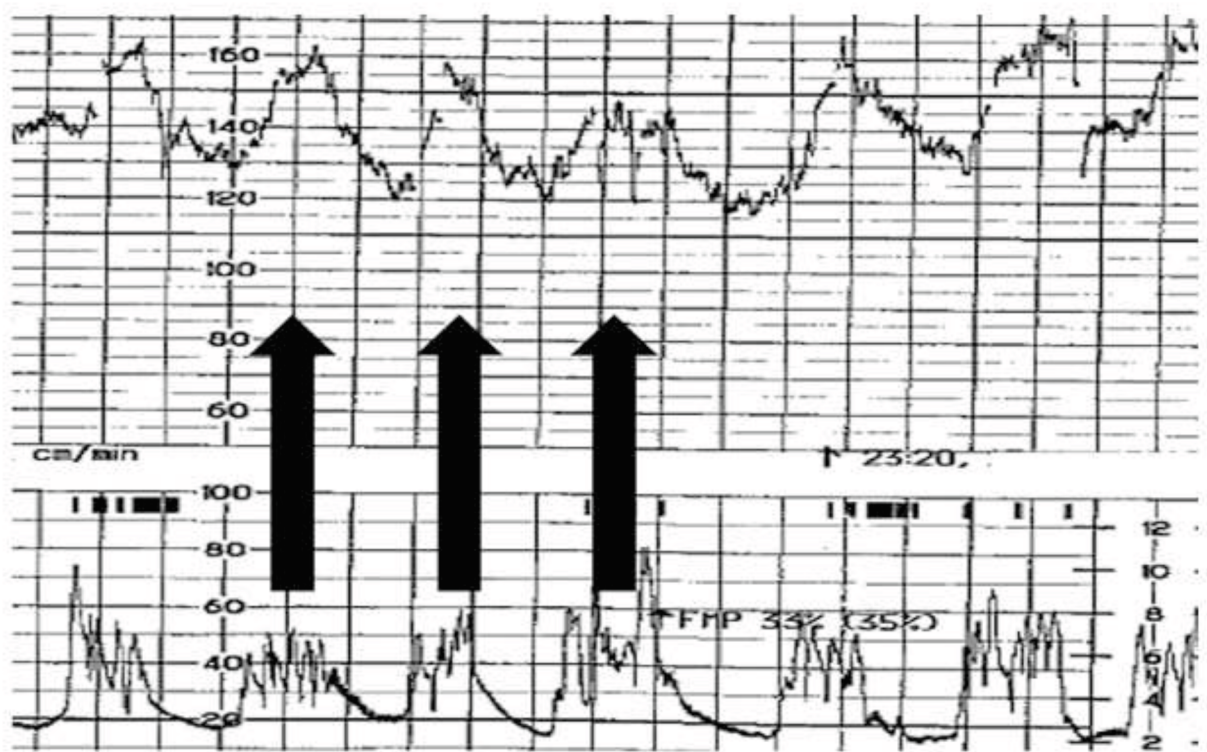


Fig. 5. “Double Mountain Peak” sign.

Table 4
Antenatal CTG Interpretation Tool: the “CAUTION” Checklist.

Intrapartum Fetal Assessment Tool			
Mat Pulse:	Temp:	Initial Baseline FHR	Induced / Augmented labour? Y/N
Risk Factors.			
Current Baseline FHR	Variability	Accelerations	Decelerations Quicklies/ Tardies/Both
Paper speed			
Rise in Baseline (≥10 %)	No	Yes	
Inter-contraction interval < 90 s	No	Yes	
Abnormal Variability (<5 or > 25)	No	Yes	
No Cycling / Loss of Cycling	No	Yes	
Features of Hypoxia TYPE of Hypoxia	No Gradually Evolving/Sub-acute/ Combination/ Acute /None	Yes	
Depression of Fetal Central Organs	No	Yes	
New risk factors noted	No	Yes	
Any signs of chorioamnionitis/ infection?	No	Yes	
Any signs of non-hypoxic compromise, ZigZag or Sinusoidal Patterns?	No	Yes	
Second Opinion needed?	No	Yes	
Recommended Management Plan			
Date:			
Time:			
NAME			
SIGNATURE			

ketoacidosis etc), and should not be administered to correct fetal heart rate abnormalities. NHS Resolution (a body which defends clinical negligence claims against the NHS) Report in 2019 has reported that administration of excessive fluids during labour increases maternal and neonatal morbidity due to fluid overload and electrolyte imbalance and neonatal convulsions due to dilutional hyponatremia [48].

Maternal oxygen supplementation to treat fetal heart rate abnormalities

This has been discontinued in clinical practice for several years as the potential risks outweigh harm [49], and it was not recommended in the first edition of the international expert consensus guidelines on physiological interpretation of CTG in 2018. Recently, the American College of Obstetricians and Gynaecologists (ACOG) have also released a Practice Bulletin, which has stated that based on scientific evidence, routine use of oxygen supplementation in individuals with normal oxygen saturation is not recommended for fetal intrauterine resuscitation [50]. Therefore, maternal oxygen or fluid therapy to correct fetal heart rate abnormalities is no longer recommended in clinical practice [49]. Maternal oxygen supplementation is recommended in all clinical situations where administration of oxygen is essential to ensure maternal wellbeing (e.g., bronchial asthma, maternal sepsis, maternal cardiopulmonary disorders etc).

CRedit authorship contribution statement

Edwin Chandraharan: Conceptualization, Formal analysis, Methodology, Project administration, Supervision, Writing – original draft, Writing – review & editing. **Susana Pereira:** Methodology, Writing – original draft, Writing – review & editing. **Tullio Ghi:** Conceptualization, Resources, Writing – original draft, Writing – review & editing. **Anna Gracia Perez-Bonfils:** Writing – review & editing. **Stefania Fieni:** Writing – original draft, Writing – review & editing. **Yan-Ju Jia:** Writing – original draft, Writing – review & editing. **Katherine Griffiths:** Writing – review & editing. **Suganya Sukumaran:** Writing – review & editing. **Caron Ingram:** Writing – review & editing. **Katharine Reeves:** Writing – review & editing. **Mareike Bolten:** Methodology, Writing – review & editing. **Katrine Loser:** . **Elena Carreras:** Writing – review & editing. **Anna Suy:** Writing – review & editing. **Itziar Garcia-Ruiz:** Writing – review & editing. **Letizia Galli:** Writing – review & editing. **Ahmed Zaima:** Methodology, Resources, Writing – original draft, Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.ejogrb.2024.09.034>.

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