Journal of
Clinical & Medical Surgery

www.jclinmedsurgery.com

ISSN 2833-5465
Open Access
Volume 3

Short Commentary

Updated NICE Cardiotocograph (CTG) Guideline: Is it Suspicious or Pathological?

Edwin Chandraharan*

Director, Global Academy of Medical Education & Training, London, UK.

***Corresponding Author: Edwin Chandraharan**
Director, Global Academy of Medical Education & Training, London, UK.
Email: edwin.c@sky.com

Article Information

Received: Nov 06, 2023
Accepted: Dec 08, 2023
Published: Dec 15, 2023
Archived: www.jclinmedsurgery.com

Abstract

Cardiotocograph (CTG) trace was introduced into clinical practice in 1960s as a tool to record the changes in the fetal heart rate (cardio) in response to ongoing uterine contractions (toco). The intention was to timely recognise the features of fetal decompensation so that immediate action could be taken to avoid hypoxic ischaemic encephalopathy (HIE) and/ or intrapartum hypoxia-related perinatal deaths, without increasing unnecessary operative interventions to the mother. However, very unfortunately, unlike other tools in clinical medicine, CTG was introduced into clinical practice without any randomised controlled trials or robust scientific studies not only to confirm its effectiveness, but to determine the CTG features which actually reflected fetal central organ oxygenation. This chasm of deficiency of knowledge of fetal physiology created a vacuum of understanding regarding what features were reflecting fetal compromise. Regrettably, this vacuum was soon filled by prominent obstetricians from national societies who were presumed to have the knowledge, but they began the arduous journey of classifying decelerations which are normal fetal cardioprotective reflexes, and they misclassified them as signs of "fetal distress". Classification of these decelerations based on the observed morphology into "reassuring" and "nonreasoning" categories has resulted in disastrous consequences for women and babies. In the UK, since the publication of the first CTG guideline in 2001, due to continuing lack of knowledge and confusion, these guidelines were repetitively revised in 2007, 2014, 2017 and 2022. Despite the international consensus guidelines of physiological interpretation of CTG produced by 44 CTG experts from 14 countries which recommended classification of CTG traces based on the type of fetal hypoxia and fetal response to stress, the latest revised guideline produced by the National Institute of Health and Care Excellence (NICE), had continued the illogical approach of grouping arbitrary features into different categories and then randomly combining them to classify the CTG traces into "Normal, Suspicious and Pathological". Therefore, all practising clinicians who are focussed on protecting their patients from harm have a responsibility to ask the question whether the revised NICE CTG Guideline itself is suspicious or pathological from a patient safety perspective.

Keywords: Cardiotocograph; Suspicious; Pathological; Fetal blood sampling; ZigZag pattern; "How is THIS Fetus?"; Physiological CTG interpretation.

Abbreviations: CTG: Cardiotocograph; HIE: Hypoxic-ischaemic encephalopathy; NICE: National Institute of Health and Care Excellence.

Citation: Chandraharan E. Updated NICE Cardiotocograph (CTG) guideline: Is it suspicious or pathological?. J Clin Med Surgery. 2023; 3(2): 1129.

Introduction

Every human fetus who undertakes the journey of human labour which lasts for approximately 6-10 hours on average, is exposed to an inevitable and gradually progressing hypoxic stress. This is because as the woman begins contracting the uterus to expel her fetus through the bony and soft tissue parts of the maternal birth passage, spiral arterioles which traverse the myometrium to supply the placental bed are compressed, resulting in reduced utero-placental perfusion during uterine contractions. Moreover, depending on the position of the loops of umbilical cord in relation to bony fetal parts and bony maternal pelvis, and the amount of amniotic fluid and Wharton's Jelly available to protect the umbilical blood vessels within the umbilical cord, there may be also varying degrees of umbilical cord compression during a uterine contraction. Both these mechanisms may significantly reduce fetal oxygenation, resulting in intermittent hypoxic stress which may compromise oxygenation of fetal central organs. As the uterine contractions increase in frequency, duration and strength as the labour advances, this intermittent hypoxic stress may get progressively worse, and may increase the likelihood of hypoxic-ischaemic encephalopathy (HIE) or an early neonatal death (ENND).

Fortunately, the vast majority of human fetuses are able to successfully mount effective compensatory responses to this intermittent and gradually evolving and progressive hypoxic stress. Physiological changes that enable intrauterine fetal adaptation to compensate for the relatively hypoxic intrauterine environment, that mimic "An Everest-in-Utero" [1] confer remarkable resilience on human fetuses to withstand transient and brief hypoxic episodes without sustaining any damage. This is analogous to a marathon runner who increases his/her/their heart rate and respiratory rate to ensure adequate oxygenation of their central organs in order to avoid the onset of anaerobic metabolism and the production of lactic acid. Their compensatory response to ongoing hypoxic stress whilst running a marathon may be blunted by pre-existing medical disorders (e.g., coronary heart disease, diabetes, flu), poor individual reserve to withstand hypoxic stress (e.g., age>75), increased metabolic demand (e.g., obesity, hot environmental temperature) and the speed of running (e.g., higher the speed, earlier the exhaustion).

Similarly, antenatal utero-placental insufficiency, adverse maternal environment, loss of fetal compensatory mechanisms and iatrogenic increase in uterine activity (Table 1) may blunt these compensatory responses predisposing to hypoxic-ischaemic injury with potential long term sequalae such as cerebral palsy and learning difficulties and/or perinatal deaths. These wider clinical contexts should always be considered whilst interpreting CTG traces.

The International Consensus Guideline on Physiological CTG interpretation produced by 44 CTG experts from 14 countries in 2018 [2] which was developed to aid interpretation of CTG traces to timely recognise intrapartum hypoxic stress is based on the identification of the combination and/or sequence of that indicate different types of fetal hypoxia, and determination of fetal response to ongoing stress (Table 2). In contrast, the recently updated NICE CTG Guideline continues to include arbitrary parameters grouped into "reassuring, nonreasoning and abnormal" "categories" and then randomly combining them to have an overall classification (Normal, Suspicious, Pathological) without any consideration of different types of fetal hypoxia or the fetal response to stress [3]. Such a non-physiological approach is likely to increase the risks of intrapartum-related hypoxic ischaemic encephalopathy (HIE) and perinatal deaths and/or increase the likelihood of unnecessary operative interventions for women. Therefore, in the interest of patient safety, it is vital to scrutinise the updated NICE CTG Guideline, and from a scientific perspective, ask the question: Is the NICE CTG Guideline itself is suspicious and/or pathological?

Understanding the perilous journey of UK CTG guidelines

The first CTG Guideline was produced by the Royal College of Obstetricians and Gynaecologists (RCOG) in 2001 [3]. It is extraordinary that despite CTG being introduced into clinical practice in 1968 and was being routinely in the UK labour wards, the decision to produce a national guideline on CTG interpretation took 33 years. The driving force was the shocking findings of the Confidential Enquiries into Stillbirths and Deaths in Infancy (CESDI) in1997 [4], which concluded that out of approximately 800 babies who had an intrapartum-related stillbirth in the UK, 50% had a "Grade 3 Substandard Care" (i.e., with a different care, these babies would have most likely survived), and 75% had "Grade 2 and Grade 3 Substandard Care" (i.e., they may have survived). Lack of knowledge regarding CTG interpretation was highlighted as a major factor which contributed to these potentially avoidable intrapartum-related deaths. Regrettably, despite the publication of this report in 1997, it took further 4 years to produce a national CTG Guideline in the UK [3], potentially resulting in the loss of approximately 500 babies during each year due to substandard care due to CTG misinterpretation.

It was obvious to all frontline clinicians who understood fetal physiology that the first CTG guideline classification tool (Figure 1) was deeply flawed both from scientific and common-sense perspectives (Table 3).

Table 3 Categorisation of fetal heart rate traces

Category	Definition
Normal	A CTG where all four features fall into the reassuring category.
Suspicious	A CTG whose features fall into one of the non-reassuring categories and the remainder of the features are reassuring.
Pathological	A CTG whose features fall into two or more non-reassuring categories or one or more abnormal categories.

Table 4 Categorisation of fetal heart rate (FHR) features

Feature	Baseline (bpm)	Variability (bpm)	Decelerations	Accelerations
Reassuring	110–160	≥ 5	None	Present
Non-reassuring	100–109 161–180	< 5 for >40 to <90 minutes	Early deceleration Variable deceleration Single prolonged deceleration up to 3 minutes	*The absence of accelerations with an otherwise normal CTG are of uncertain significance*
Abnormal	<100 >180 Sinusoidal pattern ≥ 10 minutes	<5 for ≥ 90 minutes	Atypical variable decelerations Late decelerations Single prolonged Single prolonged deceleration >3 minutes	

Figure 1: The RCOG CTG Classification Tool (May 2001).

The categorisation recommended by the guideline was both illogical and scientifically impotent because it stated that "all 4 features should be reassuring" for the CTG trace to be classified as normal (Figure 1). However, it stated that the absence of accelerations was of "uncertain significance". If this was the case, then, accelerations should not have been considered whilst classifying the CTG traces as "normal". In the absence of accelerations, if there were early or variable decelerations, the CTG trace would have to be classified as "pathological", increasing the risks of unnecessary operative interventions. Similarly, if there was a single prolonged deceleration of up to 3 minutes and an early deceleration, the CTG trace would have been classified as "pathological" warranting an unnecessary intervention. This oversimplistic and scientifically illogical approach of "mixing and matching" random features to "categorise" CTG traces as "normal, suspicious, pathological" was not only implemented in routine clinical practice, but it was also strictly and religiously audited to ensure compliance of at least 75%.

The RCOG CTG guideline was subsequently acquired by NICE as "inherited" NICE Guideline, however, no action was taken to identify and rectify the flaws (Table 3). It was not surprising that not only the rate of caesarean sections increased in the UK, but the Chief Medical Officer's Report, titled "Intrapartum-related deaths: 500 missed opportunities" published in 2006 highlighted CTG misinterpretation as an important contributory factor for these, very unfortunate avoidable intrapartum-related deaths [5]. It should have been very evident to those who produced the CTG Guideline and the professional bodies who had the responsibility implement evidence-based clinical practice in the UK and to "set standards to improve women's health" that the system of classifying the CTG traces into "normal, suspicious, pathological" had to be abandoned immediately to safeguard women and babies. There was an urgent need to implement a CTG guideline tool that is based on the deeper understanding of fetal physiology to determine the features of different types of fetal hypoxia and fetal response to ongoing hypoxic stress.

Unfortunately, not only this opportunity to rectify the flaws of CTG guideline produced in 2001 was missed, due to some inexplicable and bizarre reason, the NICE CTG guideline development group (GDG) in 2007 chose to make it even more confusing by adding arbitrary time limits to be applied to all human fetuses [Figure 2]. In addition to the flaws identified in 2001 CTG Guideline, further incorporation of unscientific time limits (e.g., decelerations for 50% of contractions for 30 or 90 minutes) not only increased the likelihood of inter and intra-observer variability which would have resulted in variation in management and resultant poor outcomes, but, it would have also contributed to poor perinatal outcomes and an increase in the rate of unnecessary operative interventions [Table 4]. It was not surprising that within 2 years of publication of this confusing guideline with no consideration to fetal responses to stress or features of intrapartum hypoxia, the NHS Litigation Authority (NHSLA)'s Study on Stillbirth Claims highlighted that 34% of all successful clinical negligence claims against the NHS on stillbirths were due to CTG misinterpretation [6]. However, no actions were taken to rectify these very obvious errors (Table 4), despite the knowledge that 34% of babies died solely due to CTG misinterpretation. There was no attempt by professional bodies who had the responsibility to promote evidence-based practice to stop the unphysiological, confusing, CTG guideline with arbitrary, personal opinion-based time limits which was contributing to CTG misinterpretation.

Table 6 Classification of FHR trace features

Feature	Baseline (bpm)	Variability (bpm)	Decelerations	Accelerations
Reassuring	110–160	≥ 5	None	Present
Non-reassuring	100–109 161–180	< 5 for 40–90 minutes	Typical variable decelerations with over 50% of contractions, occurring for over 90 minutes Single prolonged deceleration for up to 3 minutes	The absence of accelerations with otherwise normal trace is of uncertain significance
Abnormal	< 100 > 180 Sinusoidal pattern ≥ 10 minutes	< 5 for 90 minutes	Either atypical variable decelerations with over 50% of contractions or late decelerations, both for over 30 minutes Single prolonged deceleration for more than 3 minutes	

Figure 2: First revision of the NICE CTG Guideline in 2007.

NICE CTG Guideline in was revised again in 2014 [7] following the publications of 10 years of Maternity Claims by the NHSLA in 2012 [8], which highlighted the financial and clinical burden and the human costs of CTG misinterpretation on babies, women and their families. Many consider that NICE 2014 CTG Guideline was one of the worst and the most dangerous of all the CTG Guidelines produced not only in the UK, but in the world. This guideline not only recommended "oral fluids" to treat suspicious and pathological CTG traces without even considering the time taken for absorption of water during labour to treat ongoing CTG abnormalities, but also included several recommendations solely based on the personal opinions of those in the GDG without, or contrary to scientific evidence and basic physiological principles, increasing the risks of maternal and perinatal morbidity and mortality. Furthermore, this CTG guideline openly advised obstetricians to hide the information derived from scientific evidence on FBS from women. Alarmingly, despite concluding based on the review of scientific evidence, that FBS increased the rate of emergency caesarean sections and operative vaginal births, it recommended that women should be advised exactly the opposite: that FBS may reduce the need for further interventions [7]. Although this blatant lack of regard to "Duty of Candour" (a legal requirement in the UK) by the NICE CTG Guideline Group was questioned [9], no action was taken to rectify this recommendation. Women were continued to be provided with incorrect, and misleading information contrary to available scientific evidence, and were not informed that the test that was being performed on their babies did not improve their perinatal outcomes, but FBS has been shown to significantly increase their emergency caesarean sections and operative vaginal births. If honesty and openness had prevailed, and the correct information was given as in other specialties of clinical medicine in the UK, it was very likely that most women would have declined FBS.

Fortunately, for the first time in the history of intrapartum care, the labour ward lead consultants from 19 hospitals in London formed the "Pan London Labour Ward Leads Group" and they signed a joint letter to the President of the Royal College of Obstetricians & Gynaecologists (RCOG), expressing their concerns. Very unfortunately, no action was taken by the professional bodies prior to the intervention of the Pan London Labour Ward Leads group despite the obvious fact that if this flawed CTG guideline was implemented, both maternal and perinatal outcomes would significantly worsen resulting in disastrous consequences to women, their babies and families as well as to frontline midwives and obstetricians. It was not surprising that the Each Baby Counts Report in 2015 highlighted that out of 1136 babies who died or sustained severe brain damage, in 76%

a different care might have made a difference to the outcome (10). Moreover, issues with CTG interpretation and FBS contributed to 61% of all cases of poor outcomes [10].

Unfortunately, it took almost 3 years for NICE to finally revise the guideline in 2017 due to "stakeholder concerns" [11], which resulted in the same errors being perpetuated for 3 years, resulting in disastrous consequences for babies, women and their families. This was evidenced by subsequent Each Baby Counts Report concluding that even in 2016, issues with CTG interpretation and FBS continued to contribute to 60% of poor outcomes [12]. Although, there was evidence from Cochrane Systematic Review from 2013 that FBS did not improve long term perinatal outcomes and did not reduce intrapartum operative interventions [13], and commentaries in the "College Journal" highlighting the dangers [14,15], no action was taken to rectify the error either by the professional bodies or by NICE. This resulted in issues with CTG misinterpretation and FBS continuing to contribute to approximately 60% of severe hypoxic-ischaemic encephalopathy and perinatal deaths even in 2019 [16], and 2020 [17]. The fact that FBS was continued to be recommended by the NICE GDG in 2017 despite of scientific evidence showing benefit, and in fact, scientific evidence from 2015 suggesting that repetitive FBS resulted in doubling of caesarean sections [18] illustrates that personal opinions were prioritised over evidence-based medicine by those who produced CTG guidelines in the maternity service in the UK. It was alarming that despite a UK multi-centre trial in 2019 concluding that FBS did not improve perinatal outcomes, and it had increased intrapartum emergency sections by approximately 60% [19], no action was taken by either professional bodies or NICE to immediately stop the FBS to safeguard women and babies from avoidable harm.

Recently revised & updated NICE CTG guideline (Dec 2022)

The revised NICE CTG Guideline finally stopped recommending FBS due to "lack of evidence" [20], although issues with CTG interpretation and FBS had already contributed to significant patient harm (Figure 3). Several illogical, scientifically impotent and clinically dangerous clinical practices such as administering fluids to the mother which, according to the NHS Resolution Report increased the risks of maternal deterioration and neonatal convulsions [21] were finally stopped by this "revised & updated" NICE CTG Guideline in 2022. Fortunately, this "revised and updated" guideline finally rectified the unscientific and dangerous increase in the threshold of abnormal baseline FHR used in the UK from 2001 (i.e., 180 bpm instead of 160 bpm as per all other international guidelines from 1987) which, most likely had contributed to severe brain damage and deaths of babies in the UK for 21 years.

However, it was regrettable and unacceptable from a patient's safety perspective that professional bodies had turned a blind eye to administering of fluids to the wrong person (i.e., mother) for presumed "suspicious" or "pathological CTGs" until December 2022. Patient centred clinical practice demanded that this illogical practice to have ended much sooner than in 2022. Our patients would have expected that frontline clinicians abandon this unscientific, illogical and potentially harmful practice immediately after the publication of the NHS Resolution Report in 2019 [21] which highlighted the risks of maternal deterioration and neonatal convulsions due to excessive administration of fluids to the mother during labour, dilutional hyponatremia and fluid overload. At the very least, the patients would have expected this potentially dangerous practice to stop immediately after the publication of a *Commentary* highlighting these risks in 2020, in an open access journal which did not require any subscription fees to access [22].

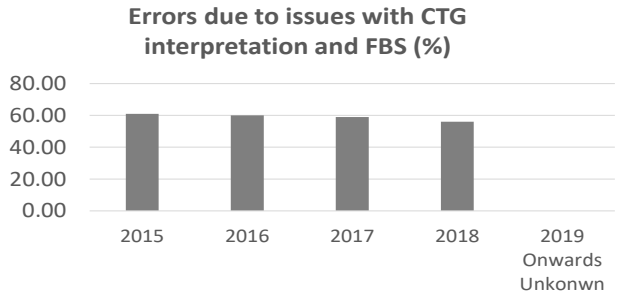

Figure 3: Errors due to issues with CTG interpretation & FBS highlighted by each baby counts reports from 2015-2019.

Regrettably, the fundamental flaws in the NICE CTG guideline which had most likely resulted in harm to babies and women were not rectified even in 2022 [20]. This guideline continued to classify CTG traces into "Normal, Suspicious, Pathological" by randomly grouping certain "features" into different "categories without considering the combination of / sequence of fetal heart rate changes which indicate ongoing fetal hypoxia or features of fetal compensatory response (Table 5). Perpetuating the same errors with regard to the classification of CTG traces into "normal, suspicious and pathological" without considering the type of fetal hypoxia and the features of fetal response to stress, since 2001, despite publications in Open Access Journals (i.e., does not require a subscription fee for the NICE CTG GDG to access) in 2016 [29] is indeed very unfortunate. These flaws in the NICE CTG Guideline were again highlighted in an Invited Commentary in the Journal of Patient Safety and Risk Management in 2019 [31]. From a patient safety perspective, it is very alarming that guideline group had chosen to continue to focus on the morphological appearance of decelerations and grouping arbitrary CTG features with unscientific, illogical time-limits into "normal, suspicious, pathological".

Bizarrely, the updated and revised NICE CTG Guideline appears to have simply lifted "How is this fetus?" which was highlighted in Physiological CTG Masterclasses from 2012, and then translated into several languages, and was also highlighted in several publications [31,32]. Their attempt at introducing "How is the baby?" in conjunction with the use of "normal, suspicious, pathological" classification [33] is scientifically amusing at best, and potentially dangerous at worst. It is scientifically amusing because anyone who understands basic fetal physiology to recognise the types of fetal hypoxia and the CTG features of fetal compensatory responses, and asks the question "How is the baby?" would not arbitrary group CTG features into "normal, suspicious. Pathological". This is because "How is the Baby?" due to understanding of fetal physiological responses and "normal, suspicious, pathological" classification due to the lack of understanding of fetal physiological responses, are mutually exclusive. One cannot classify a CTG trace as "suspicious" (i.e., not sure of the significance of observed CTG features) and then ask the question "How is the baby?" because there are no "suspicious" fetuses. On the other hand, simply transposing some principles from Physiological CTG Masterclasses, without fundamentally changing the CTG interpretation tool-based knowledge of fetal physiology may lead to confusion resulting in poor maternal and perinatal outcomes. Although, there have been several publications on Physiological Interpretation of CTG prior the publication of the revised NICE CTG Guideline [34-54], including improvement in outcomes following the implemen-

tation of Physiological Interpretation of the CTG [55-61], it is indeed regrettable that this revised guideline has disregarded these principles of physiological CTG interpretation. Merely lifting "How is the Baby?" alone from Physiological CTG Masterclasses [33] will not result in improvement in maternal and perinatal outcomes, if the "normal, suspicious, pathological" classification system is continued to be used.

The price of repetition of errors involving CTG interpretation

Albert Einstein once said "insanity is repeating the same processes again and again, and expecting different results". In the UK the same, unscientific "Normal, Suspicious, Pathological" classification system which failed to individualise care was introduced in 2001, and then, repeated in 2007, 2014, and 2017, with disastrous consequences to babies and their families. These have been highlighted in the Chief Medical Officer's Report (2006), NHSLA Report on Stillbirth Claims (2009), NHSLA 10 years of Maternity Claims (2012), and four consecutive Each Baby Counts Reports (2015-2019). As Albert Einstein stated many years ago repeating the same (flawed) CTG classification system ("normal, suspicious, pathological) again and again has not resulted in improvement in perinatal outcomes. In an advanced economy which prides itself of having a "world class maternity service", it is no longer acceptable to have lack of knowledge leading errors due to CTG interpretation to contribute to more than 50% of babies sustaining severe brain injuries or dying due to an intrapartum-related death.

Unsurprisingly, UK public have now demanded criminal prosecution against those who contributed to poor perinatal outcomes (https://www.independent.co.uk/news/health/east-kent-maternity-baby-deaths-b2206143.html), and more recently they have demanded an independent public enquiry (https://www.theguardian.com/society/2023/oct/31/parents-of-babies-who-died-or-were-harmed-in-nhs-care demand-inquiry). This is understandable because, as frontline midwives and obstetricians, if our own babies are brain damaged or died during labour due to an erroneous guideline which classified fetal heart rate traces into "normal, suspicious, pathological" without incorporating fetal physiology, and then recommended to administer fluids to the mother for suspicious CTGs and erroneously recommended to cut the skin of our own babies' scalp check for oxygenation of the fetal brain, then, most likely we would demand criminal prosecution and a public enquiry too. Parents have the right to be shocked when they eventually find out that in the UK, not only it was almost universal to have rolls of "CTG Stickers" with exactly the same parameters which were used in every human fetus, irrespective of the individual fetal reserves or clinical context, the use of these "stickers" were rigorously audited to ensure >75% compliance. This resulted in the strict enforcement and implementation of the wrong tool which was causing patient harm, and financial incentives (discounts for the insurance premium) were provided for demonstrating that there was progress towards 100% of using these wrong CTG tools.

At the time of writing this commentary, the degree of harm caused to women as a result of CTG interpretation have not been monitored or reported. Currently, clinicians have absolutely no idea of the number of women who had massive postpartum haemorrhage, wound complications or died due serious complications such as uterine rupture or placenta accrete spectrum disorders in subsequent pregnancies due to an unnecessary caesarean section being performed for a "pathological CTG" or following an "abnormal" FBS result (which reflected normal acidosis in the peripheral tissues due to centralisation of blood flow to compensate for the ongoing hypoxic stress). Similarly, one has no idea about the rate of perineal tears and trauma sustained by women due to an unnecessary operative vaginal birth due to a "pathological CTG" during the second stage of labour. There is no doubt that the general public would want to investigate these maternal complications in the near future.

The way forward: need for the "Self-Candour Test": The Hippocratic Oath stipulates "first do no harm" and "acting in the best interest of our patients" as key cornerstones of good medical practice. Therefore, in the interest of patient safety, honesty and ensuring our clinical practice is based on current scientific evidence and scientific principles, frontline clinicians should consider the "Self-Candour Test" (Table 6) prior to implementing the updated and revised NICE CTG Guideline in their own clinical practice. If the answer to any of these questions in Table 6 (especially Question 5) is "No", then, it is important to practice the "Duty of Candour" and understand the principles of Physiological Interpretation of CTG [34-54,62-63], and to provide patients with an evidence-based, scientifically acceptable clinical care.

Table 1: Potential causes which may contribute to, and/or accelerate fetal compromise.

Source	Underlying pathology	Likely Mechanisms of fetal compromise
Maternal	Hypertension/Pre-eclampsia	Placental infarction & thrombosis, placental abruption
	Diabetes	Hyperplacentosis / Terminal Villus hypoplasia
	Infection or Sepsis	Bacterial toxins and inflammatory cytokines
	Pyrexia	Increased maternal and fetal metabolic rate and increased oxygen demand
	Thrombotic states, hypercoagulable states (e.g., twins)	Placental thrombosis and infarction and reduced perfusion
	Immunological conditions	Congenital heart block (SLE), autoimmune haemolytic anaemia (rhesus)
	Respiratory disorders including pneumonia	Hypoxaemia and hypoxia and acidosis
	Hypovolumia or hypotension	Reduced placental perfusion
Fetal	Growth Restriction	Reduced feto-placental reserve, and inability to re-distribute and centralise blood flow
	Macrosomia	Increased oxygen requirements
	Oligo or anhydramnios	Increased likelihood of umbilical cord compression
	Chronic fetal anaemia and/or acidosis	Reduced tissue oxygenation and acidosis to the central organs
	Chorioamnionitis	Increased metabolic rate, fetal neuroinflammation and fetal systemic inflammatory response syndrome (FIRS)
Contractions	Increased uterine activity (any increase in the frequency, duration, strength and the basal tone)	Reduced inter-contraction interval for oxygenation of placental villi, and increased likelihood of cord compression and fetal head compression

Table 2: CTG Classification based on International Expert Consensus Guidelines on Physiological Interpretation.

Hypoxia	Features	Management
No Hypoxia	• Baseline appropriate for G.A. • Normal variability and cycling • No repetitive decelerations	• Consider whether the CTG needs to continue. • If continuing the CTG perform routine hourly review. (see CTG Assessment Tool 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, postmaturity, IUGR	• Avoid further stress • Expedite delivery, if delivery is not imminent
Gradually Evolving Hypoxia	**Compensated** Rise in the baseline (with normal variability and stable baseline) preceded by decelerations and loss of accelerations	• Likely to respond to conservative interventions (see below) • Regular review every 30-60 minutes to assess for signs of further hypoxic change, and that the intervention resulted in improvement. • Other causes such as reduced placental reserve MUST be considered and addressed accordingly.
	Decompensated • Reduced or increased variability • Unstable/ progressive decline in the baseline (step ladder pattern to death)	• Needs urgent intervention to reverse the hypoxic insult (remove prostaglandin pessary, stop oxytocin infusion, tocolysis) • Delivery should be expedited, if no signs of improvement are seen
Subacute Hypoxia	• More time spent during decelerations than at the baseline • May be associated with saltatory pattern (increased variability)	**First Stage** • Remove prostaglandins/stop oxytocin infusion • If no improvement, needs urgent tocolysis • If still no evidence of improvement within 10-15 minutes, review situation and expedite Delivery **Second Stage** • 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 minutes)	**Preceded by reduced variability and lack of cycling or reduced variability within the first 3 minutes** Immediate delivery by the safest and quickest route **Preceded by normal variability and cycling and normal variability during the first 3 minutes of the deceleration (see 3-minute rule above)** • Exclude the 3 accidents (i.e. cord prolapse, placental abruption, uterine rupture - if an accident is suspected prepare for immediate delivery) • Correct reversible causes • If no improvement by 9 minutes or any of the accidents diagnosed, immediate delivery by the safest and quickest route
Unable to Ascertain fetal wellbeing (Poor signal quality, uncertain baseline, possible recording of the maternal heart rate)		• Escalate to senior team • Consider Adjunctive Techniques, if appropriate • Consider the application of FSE to improve signal quality

Table 3: Flaws in the RCOG CTG Guideline produced in 2001.

Stipulated Features / Parameters	Why was it flawed?	Likely impact on women and babies
Baseline 110-160	There is a progressive reduction in the baseline FHR as the gestation advances. Therefore, after 40 weeks one cannot use 160 bpm as the upper limit	Babies with chronic hypoxia and chorioamnionitis at term are most likely missed by this approach as they may not be able to increase the baseline FHR above 160 bpm
Abnormal baseline > 180 bpm	It was illogical to define the normal upper limit of the baseline FHR as 160 bpm, and then, artificially increase the upper threshold to 180 bpm.	This potentially dangerous action of artificially increasing the baseline, contrary to scientific evidence was likely to cause injury and damage to fetuses with reduced reserve, chorioamnionitis and chronic hypoxia as they may not be able to increase the baseline

Variability >5 bpm	All biological parameters should have a normal range. The internationally accepted range for normal variability was 5-25 bpm. Use of a single number missed fetuses with excessive variability (>25 bpm) due to rapidly developing hypoxia.	This was one of the most dangerous parameters of this guideline. Based on personal opinion, the authors took away the upper limit (25 bpm), leading to potentially disastrous consequences (stillbirths and brain damage) to fetuses exposed to a rapidly evolving hypoxia.
Reduced baseline FHR >90 minutes as abnormal	Fetal deep sleep lasts for approximately 50 minutes. This artificial increase in the duration without any scientific evidence not only increased the risk of delay in action of fetuses who were exposed to metabolic acidosis and/or CNS depression, consideration of baseline FHR variability in isolation increased the risk of unnecessary operative interventions.	Increased likelihood of hypoxic-ischaemic encephalopathy (HIE) and perinatal deaths due to delay in taking action. Increased likelihood of unnecessary intrapartum operative interventions for women due to the consideration of baseline FHR variability in isolation without incorporating the changes in the baseline FHR and preceding decelerations.
Early decelerations as a non-reassuring feature	This illogical recommendation to classify normal fetal cardioprotective reflexes as "abnormal or pathological" without considering features of fetal compensatory mechanisms.	Increased likelihood of unnecessary intrapartum operative interventions and their complications due to over-reacting, considering the normal fetal compensatory responses as abnormal.
Isolated variable and late decelerations as an abnormal feature	Isolated variable and late decelerations with an intervening stable baseline and reassuring variability are not associated with fetal compromise or acidosis.	Increased the risk of unnecessary operative interventions to women, and their resultant short term and long term complications such as uterine rupture and placenta accrete spectrum disorders (PAS).
Recommending fetal scalp blood sampling (FBS) for pathological CTG traces	This illogical and scientifically flawed practice of sampling the skin of the fetal scalp with the mistaken belief that due to its proximity to the fetal brain it would reflect the oxygen status of the brain. As a result of catecholamine-mediated peripheral vasoconstriction, similar to marathon runners, the skin of the fetal scalp, which is a peripheral non-essential organ, will undergo anaerobic metabolism and produce lactate as a normal compensatory fetal response (centralisation of blood flow). Therefore, sampling the skin will give an erroneous impression of acidosis when the fetal central organs are well perfused. On the contrary, neutralisation of the acid by the surrounding alkaline amniotic fluid or peripheral vasodilatation in chorioamnionitis may result in a false negative result.	False negative result increased the likelihood of hypoxic-ischaemic encephalopathy (HIE) and perinatal deaths due to the continuation of labour due to the false reassurance provided by the normal pH of the fetal scalp. Moreover, the recommendation to blindly repeat the result of FBS every hour had failed to consider several important variables: reduced fetal reserve, poor placental reserve, intensity of ongoing uterine contractions, the type of fetal hypoxia and co-existing pathology such as chorioamnionitis, and this erroneous approach increased the likelihood of poor perinatal outcomes.
Administering fluids to the mother for suspicious or pathological CTGs.	In clinical medicine, one would never administer fluids to the wrong patient. Fluids are only recommended to correct disorders in the maternal compartment (e.g., dehydration, hypotension, sepsis, ketoacidosis etc). Administering fluids to the mother to correct observed CTG abnormalities may cause dilutional hyponatremia and its complications.	NHS resolution Report has highlighted maternal deterioration due to dilutional hyponatremia and neonatal convulsions due to administration of excessive fluids to the mother to treat suspicious and pathological CTG traces. Moreover, this approach resulted in a false sense of security which delayed definitive treatment of ongoing fetal compromise leading to poor perinatal outcomes.

Table 4: Flaws in the NICE CTG Guideline (2007) – in addition to the continuation of the flaws highlighted in Table 3.

Additional Feature / Parameter	Why is it flawed?	What was the likely impact?
Baseline FHR 110-160	A rise in the baseline from 120 bpm to 150 bpm due to a gradually evolving hypoxia or chorioamnionitis will be missed. The CTG "Stickers" with these arbitrary range were used in all human fetuses. This gross failure to individualise care breached the fundamental principle of clinical medicine.	Increased the likelihood of hypoxic-ischaemic encephalopathy and long term sequalae (cerebral palsy and learning difficulties) and perinatal deaths.
Variable deceleration lasting for 50% of contractions for 90 minutes was a "non-reassuring" feature	This was potentially a very dangerous recommendation from the perspective of both the mother and the fetus. Variable decelerations due to umbilical cord compression are not associated with fetal acidosis, and they do not require any intervention. However, according this guideline if there is a combination of loss of acceleration and variable decelerations lasting for > 50% of contractions for 90 minutes, the CTG trace had to be classified as pathological (i.e., a random of combination of 2 "non-reassuring features), which would lead to an unnecessary operative intervention. Conversely, if the fetus had shown repetitive variable decelerations with an unstable baseline and/ or reduced baseline variability (i.e., evidence of decompensation of fetal central organs), an urgent birth was indicated. However, according to this guideline which considered features in isolation, waiting for variable decelerations to last for 90 minutes would have increased the risk of fetal injury.	Unnecessary intrapartum operative interventions (caesarean sections, vacuum and forceps births, episiotomy) and their resultant short term (postpartum haemorrhage, infection, wound breakdown, inadvertent injury to adjacent structures), and long term complications (uterine rupture, placenta accrete spectrum). Delay in accomplishing delivery by adhering to unscientific, personal opinion-based arbitrary time limits increased the likelihood of hypoxic-ischaemic encephalopathy and long term sequalae (cerebral palsy and learning difficulties) and perinatal deaths.
Atypical variable decelerations lasting for 50% of contractions for 30 minutes, and late decelerations lasting for 50% of contractions for 30 minutes resulted in a "pathological CTG"	This was illogical because there was no scientific evidence to suggest that morphology of variable decelerations (typical or atypical) correlated to perinatal outcome, which increased the risk of unnecessary operative interventions. Conversely, a growth restricted fetus with an ongoing utero-placental insufficiency characterised by repetitive late decelerations may not be able to withstand 30 minutes of uterine contractions.	As above

Repeat FBS once in every 60 minutes if the CTG remains "pathological" and in 30 minutes if the CTG trace deteriorates	This recommendation reflected a gross lack of basic knowledge of fetal physiology as well as published evidence suggesting no benefit in improving perinatal outcomes. Firstly, the rate of fall in the pH is not the same in every human fetus because it is determined by individual fetal reserve, depletion of the buffering capacity and the intensity of ongoing hypoxic stress. Furthermore, the same cut-off for an "abnormal" FBS cannot be used in early first stage and late first stage/second stage of labour because scientific evidence suggests a progressive reduction in fetal scalp pH as the labour progresses. The recommendation to repeat FBS for a maximum of 3 times reflects gross lack of knowledge of the rate of progress of labour in primigravidae. If FBS was commenced at 4 cm dilatation, by the time the 3rd FBS was performed, the woman would not have progressed to more than 7 cm, resulting in an unnecessary intrapartum interventions.	As above Women were not informed about the lack of evidence of benefit, and rare but potentially life-threatening complications such as CSF leakage, scalp haemorrhage, haemorrhagic shock and scalp abscesses. Women were not counselled regarding potential false negative results (neutralisation by the alkaline amniotic fluid, vasodilatation and increased peripheral blood flow in chorioamnionitis) or the false positive results (meconium with bile acids, and taking a sample from area od caput).

Table 5: Flaws in the "Updated & Revised" CTG Guideline 2022.

Recommendation	Why is it flawed?	Potential impact on women, babies AND Front-line clinicians
Increased variability >25 bpm is for 10 minutes is "suspicious" and >25 bpm for >30 minutes "pathological". (Pages 19 & 20)	Increased variability >25 bpm (the "ZigZag" Pattern) is associated with a rapidly evolving hypoxia (e.g., injudicious use of oxytocics or active maternal pushing). Scientific evidence have shown that ZigZag Pattern which lasts for more than 2 minutes has been shown to be associated with poor perinatal outcomes and approximately 11 fold increase in the admission to the neonatal unit [23,24].	This dangerous attempt to artificially increasing the duration of increased variability to 10 minutes and 30 minutes for suspicious or pathological CTG traces, respectively, contrary to published scientific evidence, based on personal opinion, is likely to increase the risk of severe hypoxic ischaemic encephalopathy and perinatal deaths due to this delay in ensuring appropriate interventions.
Morphological classification of decelerations and classifying those with "concerning characteristics" as "pathological" (Page 21).	Decelerations are fetal physiological compensatory responses to reduce the myocardial workload and to maintain an aerobic metabolism in response to intermittent and transient hypoxic stress. They are the fetal counterpart of adult respiratory rate. Therefore, similar to the observed increase in the rate and depth of respiration with progressively increasing speed of the treadmill in the Gym, FHR decelerations would become wider and deeper as the hypoxic stress progresses, as the fetus attempts to protect the myocardium. It has been reported that the morphological appearance of decelerations have absolutely no correlation with the perinatal outcomes, and it is the fetal response to ongoing hypoxic stress which is associated with metabolic acidosis [25,26].	Classifying normal fetal physiological responses as "pathological" based on the morphological appearance of the observed decelerations would increase inter and intra-observer variation resulting in variation in maternal and perinatal outcomes in the UK. More importantly, the erroneous, illogical classification of normal fetal physiological cardioprotective responses as "pathological" due to lack of knowledge would lead to unnecessary operative interventions to women with serious consequences (postpartum haemorrhage, sepsis, wound complications and long term complications such as placenta accreta spectrum and uterine rupture).
Repetitive variable or late decelerations lasting for 50% of contractions >30 minutes as "Pathological" (Page 22).	This illogical and potentially dangerous approach which does not consider the fetal response to ongoing hypoxic stress will lead to both unnecessary operative interventions to the mother as well as delay in interventions to fetuses with reduced fetal reserve, co-existing chorioamnionitis or post-term fetuses with a relative utero-placental insufficiency (RUPI). One would not expect a reasonably knowledgeable and reasonably skilled cardiologist to produce a guideline that states that all humans should withstand ST Segment changes, and breathe in a particular manner for 30 minutes during a cardiac stress test (treadmill) to become "pathological".	Increased likelihood of hypoxic-ischaemic encephalopathy and perinatal death due to waiting for a pre-defined time period in fetuses with poor reserves. Increased risk of maternal complications as a result of unnecessary operative interventions which show repetitive variable decelerations but show good evidence of oxygenation to the central organs.
Fetal Scalp Stimulation for fetuses with Sepsis, slow progress of labour or meconium (Pages 26 & 27)	This is one of the most illogical and dangerous recommendations. Fetuses with sepsis, chorioamnionitis or meconium have an alternative pathway of brain damage (inflammatory pathway). Clinicians should scrutinise the CTG trace for features of neuroinflammation [27,28], and if these are present, birth should be expedited without subjecting the fetus to a super-imposed hypoxic stress. Fetal scalp stimulation in an inappropriate test in this situation. Moreover, slow progress of labour requires a careful assessment of the reasons for the slow progress and the management decisions should be made accordingly, irrespective of whether a fetus responds to fetal scalp stimulation or not.	Increased likelihood of hypoxic-ischaemic encephalopathy and perinatal death due to waiting for a pre-defined time period in fetuses with poor reserves. Unnecessary intrapartum operative interventions due to the false negative test.

Overall classification of CTG into "Normal, Suspicious Pathological" (Page 24)	This illogical and dangerous attempt to group random "amber and red" features and based on the number of these "amber and red" classify CTG traces into "normal, suspicious, pathological" appears to illustrate continuing lack of knowledge of CTG interpretation. Fetuses with a combination of CTG features due to an ongoing chronic hypoxia or chorioamnionitis will be missed by these approach. Conversely, fetuses which show a series of sequential changes due to a gradually evolving or a subacute hypoxic stress will also be missed by this approach. Moreover, fetuses which mount a successful compensatory response to an ongoing gradually evolving hypoxic stress will be misclassified as "pathological", leading to unnecessary operative interventions to the mother.	It is very likely that the poor outcomes due to CTG misinterpretation highlighted by repetitive "Each Baby Counts" Reports are likely to continue. Moreover, the exponential increase in the caesarean section rate in the UK that has been observed ever since the CTG Guideline stating "Normal, Suspicious, Pathological" in 2001 is likely to continue. This is because it has been shown that the vast number of CTG abnormalities have no corelation with fetal metabolic acidosis, and the false positive rate of a "pathological" CTG trace is >90%.

Table 6: The "Self-Candour Test".

	Honest "Self-reflection Questions"	Yes	No
1	Is it scientifically acceptable to erroneously classify normal fetal reflex responses to reduce their myocardial workload (i.e., decelerations) based on their morphology into "suspicious or pathological"?		
2	Are the arbitrary time limits (e.g., 50% of contractions for >30 minutes) based on robust scientific evidence?		
3	Does the guideline indicate the features of different types of fetal hypoxia, fetal compensatory responses and features of neuroinflammation such as presence or absence of FHR cycling, to help optimise perinatal outcomes?		
4	Based on basic principles of clinical medicine, is it acceptable to assign different features with arbitrary time limits into different categories, and them combine randomly to classify the CTG traces into normal, suspicious and pathological?		
5	Would I allow any other doctor to apply a tool which is not based on scientific principles and its use may increase unnecessary operative interventions with resultant complications and /or increase the likelihood of misinterpretation resulting poor perinatal outcomes on my close friend or my family?		

Conclusion

The "updated & revised" NICE CTG Guideline has finally stopped FBS and the administration of fluids to the mother for the treatment of suspicious and pathological CTG traces, after 21 years of introducing these potentially dangerous, illogical clinical practices in the UK. However, it has continued to use the same "normal, suspicious, pathological" classification system without incorporating the features of different types of fetal hypoxia, the features of fetal compensatory responses and the features of fetal neuroinflammation seen in chorioamnionitis which is an important pathway of fetal neurological injury. Therefore, in the author's opinion, the NICE CTG Guideline (2022), similar to its precursors from 2001, 2007, 2014 and 2017, continues to remain suspicious with regard to the knowledge of fetal physiology, with the potential to cause pathological outcomes for women and babies.

Declarations

Conflict of interest: The author has conducted several Master classes on CTG and fetal ECG in the UK, Europe, Asia, and Australia and has been the co-organizer of the Intrapartum Foetal Surveillance Course at the Royal College of Obstetricians and Gynaecologists (RCOG) and he was a member of the Editorial Board for NHS e-learning on CTG. He pioneered physiological interpretation of CTG in 2006, including the concept of "How is THIS Fetus?" to determine fetal central organ oxygenation. He was the Course lead for the Neoventa Academy and the Baby Lifeline Physiological CTG Masterclasses. Organizers and hospitals of some of these Masterclasses have received sponsorships from Philips, Neoventa, Euroking, Huntleigh, K2, Cardiac Services, and other industry to support these Masterclasses. However, the author does not have any financial or managerial interests in any of these organisations. The author was one of the 3-member guideline development group which revised the international FIGO Guidelines on CTG in 2015, and he was on the Editorial Board which produced the first International Consensus Guidelines on Physiological Interpretation of CTGs in conjunction with 44 CTG Experts from 14 countries.

References

1. Gunaratne SA, Panditharatne SD, & Chandraharan E. Prediction of Neonatal Acidosis Based on the Type of Fetal Hypoxia Observed on the Cardiotocograph (CTG). European Journal of Medical and Health Sciences. 2022; 4(2): 8-18.
2. Chandraharan E, Evans SA, Krueger D, Pereira S, Skivens S, et al. Physiological CTG interpretation. Intrapartum Fetal Monitoring Guideline. 2018. https://physiological-ctg.com/guideline.html.
3. Evidence-based Clinical Guideline No. 8 The use and interpretation of CTG in intrapartum fetal surveillance. RCOG Press. 2001.
4. Maternal and Child Health Research Consortium. Confidential Enquiry into Stillbirths and Deaths in Infancy: 4th Annual Report, 1 January–31 December 1995. London: Maternal and Child Health Research Consortium. 1997.
5. Intrapartum-related Deaths: 500 Missed Opportunities. Chapter In: 2006 Annual Report of the Chief Medical Officer. On the State of Public Health. Department of Health. 2007. (https://webarchive.nationalarchives.gov.uk/ukgwa/20130105021748/http:/www.dh.gov.uk/en/Publicationsandstatistics/Publications/AnnualReports/DH_076817).
6. Study of Stillbirth Claims by NHSLA. https://silo.tips/download/study-of-stillbirth-claims-published-by-nhs-litigation-authority. 2009.
7. National Institute of Clinical Excellence. Intrapartum care: care of healthy women and their babies during labour. NICE Clinical Guideline. 2014. https://www.nice.org.uk/guidance/cg190/resources/intrapartum-care-for-healthy-women-and-babies-pdf-35109866447557.

8. NHSLA. Ten years of maternity claims: An analysis of NHS litigation authority data. 2012. www.nhsla.com/ safety/Documents/Ten%20Years%20of%20Maternity%20Claims%20%20An%20Analysis%20of%20the%20NHS %20LA%20Data%20- %20October%202012.pdf.

9. Chandraharan E. Should national guidelines continue to recommend fetal scalp blood sampling during labor? J Matern Fetal Neonatal Med. 2016; 29(22): 3682-5.

10. Royal College of Obstetricans and Gynaecologists. Each baby Counts: key messages from 2015. London: RCOG. 2016. (https://www.rcog.org.uk/media/3fopwy41/each-baby-counts-2015-full-report.pdf).

11. https://www.ncbi.nlm.nih.gov/books/NBK550639/

12. Royal College of Obstetricians and Gynaecologists. Each Baby Counts: 2018 Progress Report. London: RCOG. 2018. (https://www.rcog.org.uk/media/dswjqyin/each-baby-counts-report-2018-11-12.pdf).

13. Alfirevic Z, Devane D and Gyte G. Continuous cardio- tocography (CTG) as a form of electronic fetal monitor- ing (EFM) for fetal assessment during labour. Cochrane Database Syst Rev. 2013; 5: CD006066.

14. Chandraharan E. Fetal scalp blood sampling during labour: is it a useful diagnostic test or a historical test that no longer has a place in modern clinical obstetrics?. BJOG. 2014;121(9):1056-1062.

15. Chandraharan E. Fetal scalp blood sampling should be abandoned: FOR: FBS does not fulfil the principle of first do no harm. BJOG. 2016;123(11):1770.

16. Royal College of Obstetricians and Gynaecologists. Each Baby Counts: 2019 Progress Report. London: RCOG. 2020. (https://www.rcog.org.uk/media/qhzlelnc/each-baby-counts-2019-progress-report.pdf).

17. Royal College of Obstetricians and Gynaecologists. Each Baby Counts: 2020 Final Progress Report. London: RCOG. 2021. (https://www.rcog.org.uk/media/a4eg2xnm/ebc-2020-final-progress-report.pdf).

18. Holzmann M, Wretler S, Cnattingius S, Nordström L. Neonatal outcome and delivery mode in labors with repetitive fetal scalp blood sampling. Eur J Obstet Gynecol Reprod Biol. 2015; 184: 97-102.

19. Al Wattar BH, Lakhiani A, Sacco A, et al; AB-FAB Study Group. Evaluating the value of intrapartum fetal scalp blood sampling to predict adverse neonatal out- comes: a UK multicentre observational study. Eur J Obstet Gynecol Reprod Biol. 2019; 240: 62-67.

20. Fetal monitoring in labour. NICE Guideline [NG 229]. 2022. (https://www.nice.org.uk/guidance/ng229).

21. NHS Resolution, The Early Notification scheme progress report: collaboration and improved experience for families. 2019. https://resolution.nhs.uk/wp-content/uploads/2019/09/NHS-Resolution-Early-Notification-report.pdf.

22. Chandraharan E. Maternal "Oxygen and Fluids Therapy" to Correct Abnormalities in the Cardiotocograph (CT G): Scientific Principles vs Historical (Mal) Practices. J Adv Med Med Res. 2020; 32: 10-6.

23. Gracia-Perez-Bonfils A, Vigneswaran K, Cuadras D, Chandraharan E. Does the saltatory pattern on cardiotocograph (CT G) trace really exist? The ZigZag pattern as an alternative definition and its correlation with perinatal outcomes. J Matern Fetal Neonatal Med 2019; 1-9.

24. Tarvonen M, Hovi P, Sainio S, Vuorela P, Andersson S, Teramo K. Intrapartum zigzag pattern of fetal heart rate is an early sign of fetal hypoxia: A large obstetric retrospective cohort study. Acta Obstet Gynecol Scand. 2021; 100(2): 252-262.

25. Williams KP, Galerneau F. Intrapartum fetal heart rate patterns in the prediction of neonatal acidemia. Am J Obstet Gynecol. 2003; 188(3): 820-3.

26. Jia YJ, Chen X, Cui HY, Whelehan V, Archer A, Chandraharan E. Physiological CTG interpretation: the significance of baseline fetal heart rate changes after the onset of decelerations and associated perinatal outcomes. J Matern Fetal Neonatal Med. 2019; 1-6.

27. Galli L, Dall'Asta A, Whelehan V, Archer A, Chandraharan E. Intrapartum cardiotocography patterns observed in suspected clinical and subclinical chorioamnionitis in term fetuses. J Obstet Gynaecol Res. 2019; 45: 2343-50.

28. Sukumaran S, Pereira V, Mallur S, Chandraharan E. Cardiotocograph (CTG) changes and maternal and neonatal outcomes in chorioamnionitis and/or funisitis confirmed on histopathology. Eur J Obstet Gynecol Reprod Biol. 2021; 260: 183-188.

29. Chandraharan E, Tahan ME, Pereira S Each Fetus Matters: An Urgent Paradigm Shift is needed to Move away from the Rigid "CTG Guideline Stickers" so as to Individualize Intrapartum Fetal Heart Rate Monitoring and to improve Perinatal Outcomes. Obstet Gynecol Int J. 2016; 5(4): 00168.

30. Chandraharan E Intrapartum care: An urgent need to question historical practices and 'non-evidence'-based, illogical foetal monitoring guidelines to avoid patient harm. Journal of Patient Safety and Risk Management. 2019; 24(5): 210-217.

31. Pereira S, Chandraharan E. Recognition of chronic hypoxia and pre-existing foetal injury on the cardiotocograph (CT G): urgent need to think beyond the guidelines. Porto Biomed J. 2017; 2: 124-9.

32. Physiological interpretation of CTG: From Knowledge to Practice. Volumes 1-3. KDP. 2022. (https://www.amazon.co.uk/s?k=chandraharan&crid=1VKO4VCPZJ5IV&sprefix=chandraharan%2Caps%2C310&ref=nb_sb_noss_1).

33. O'Heney J, McAllister S, Maresh M, Blott M. Fetal monitoring in labour: summary and update of NICE guidance. BMJ. 2022; 379: 2854.

34. Oikonomou M, Chandraharan E. Fetal heart rate monitoring in labor: from pattern recognition to fetal physiology. Minerva Obstet Gynecol. 2021; 73(1): 19-33. doi: 10.23736/S0026-4784.20.04666-3.

35. Mcdonnell, S, Chandraharan E. Fetal heart rate interpretation in the second stage of labour: pearls and pitfalls. British journal of medicine and medical research 7. 2015; 957-970.

36. Preti M, Chandraharan E. Importance of fetal heart rate cycling during the interpretation of the cardiotocograph (CTG). Int J Gynecol Reprod Sci. 2018; 1(1): 10-12.

37. McDonnell S, Chandraharan E, The Pathophysiology of CTGs and Types of Intrapartum Hypoxia, Current Women`s Health Reviews. 2013; 9(3).

38. Al Fahdi B & Chandraharan E. True vs Spurious Intrapartum Fetal Heart Rate Accelerations on the Cardiotocograph (CTG): An Urgent Need for Caution. Glob J Reprod Med. 2020; 7(5): 5556722.

39. Saeed F, Abeysuriya S, Chandraharan E. Erroneous Recording of Maternal Heart Rate as Fetal Heart Rate During Second Stage of Labour: Isn't it Time to Stop this? J Biomed Res Environ Sci. 2021; 2(5): 315-319. doi: 10.37871/jbres1233.

40. Nurani R, Chandraharan E, Lowe V, Ugwumadu A, Arulkumaran S. Misidentification of maternal heart rate as fetal on cardiotocography during the second stage of labor: the role of the fetal electrocardiograph. Acta Obstet Gynecol Scand. 2012; 91(12): 1428-32.

41. Yanamandra N, Chandraharan E. Saltatory and sinusoidal fetal heart rate (FHR) patterns and significance of FHR 'overshoots'. Curr Wom Health Rev. 2013; 9: 18.

42. Albertson A, Amer-Wåhlin I, Lowe V, Archer A, Chandraharan E. Incidence of subacute hypoxia during active maternal pushing during labour. Book of Abstracts. World Congress of the Royal College of Obstetricians and Gynaecologists (RCOG). 2016.

43. Bolten M, Chandraharan E. The Significance of 'Non-Significant' Meconium Stained Amniotic Fluid (MSAF): Colour versus Contents. J Adv Med Med Res. 2019.

44. Griffiths K, Gupta N, Chandraharan E. Intrapartum fetal surveillance: a physiological approach,Obstetrics, Gynaecology & Reproductive Medicine. 2022; 32(8):179-187.

45. Chandraharan E. Handbook of CTG interpretation: From patterns to Physiology. First Edition, Cambridge, Cambridge University Press. 2017.

46. Physiological interpretation of CTG: From Knowledge to Practice. Volumes 1-3. KDP. 2022.

47. Pereira S, Lau K, Modestini C, Wertheim D, Chandraharan E. Absence of fetal heart rate cycling on the intrapartum cardiotocograph (CTG) is associated with intrapartum pyrexia and lower Apgar scores. J Matern Fetal Neonatal Med. 2021; 22: 1-6.

48. Xie W, Archer A, Li C, Cui H, Chandraharan E. Fetal heart rate changes observed on the CTG trace during instrumental vaginal delivery. J Matern Fetal Neonatal Med. 2017; 1-8.

49. Gracia-Perez-Bonfils A, Martinez-Perez O, Llurba E, Chandraharan E. Fetal heart rate changes on the cardiotocograph trace secondary to maternal COVID-19 infection. Eur J Obstet Gynecol Reprod Biol. 2020 ; 252: 286-293.

50. di Pasquo E, Commare A, Masturzo B, Paolucci S, Cromi A, Montersino B, Germano CM, Attini R, Perrone S, Pisani F, Dall'Asta A, Fieni S, Frusca T, Ghi T. Short-term morbidity and types of intrapartum hypoxia in the newborn with metabolic acidaemia: a retrospective cohort study. BJOG. 2022; 129(11): 1916-1925.

51. Descourvieres L, Ghesquiere L, Drumez E, Martin C, Sauvage A, Subtil D, Houfflin-Debarge V, Garabedian C. Types of intrapartum hypoxia in the newborn at term with metabolic acidemia: A retrospective study. Acta Obstet Gynecol Scand. 2022; 101(11): 1276-1281.

52. Yatham SS, Whelehan V, Archer A, Chandraharan E. Types of intrapartum hypoxia on the cardiotocograph (CTG): do they have any relationship with the type of brain injury in the MRI scan in term babies? J Obstet Gynaecol. 2020; 40(5): 688-693.

53. Pereira S, Patel R, Zaima A, Tvarozkova K, Chisholm P, Kappelou O, Evanson J, Chandraharan E, Wertheim D, Shah DK. Physiological CTG categorization in types of hypoxia compared with MRI and neurodevelopmental outcome in infants with HIE. J Matern Fetal Neonatal Med. 2022; 35(25): 9675-9683.

54. Zhu LA, Blanc J, Heckenroth H, Peyronel C, Graesslin B, Marcot M, Tardieu S, Bretelle F. Fetal physiology cardiotocography training, a regional evaluation. J Gynecol Obstet Hum Reprod. 2021; 50(6): 102039.

55. Chandraharan E, Preti M, Lowe V, Archer A, Ugwumadu A, Arulkumaran S. Effectiveness of 'George's Intrapartum Monitoring Strategy' on OperativeDelivery and Perinatal Outcomes at a Teaching Hospital in London:a 5 Year Experience. Book of Abstracts. COGI Conference, Vienna. 2013.

56. Lowe, V., & Chandraharan, E. Ensuring Competency in Intrapartum Fetal Monitoring: The Role of GIMS. In E. Chandraharan (Ed.), Handbook of CTG Interpretation: From Patterns to Physiology. Cambridge: Cambridge University Press. 2017; 180-184. doi:10.1017/9781316161715.033.

57. Ingram C, Gupta N, Mustafa S, Singh M, Chandraharan E. Impact of Physiological CTG Guidelines on Intrapartum Hypoxic injuries and brain cooling. World Congress of the Royal College of Obstetricians and Gynaecologists (RCOG). 2021.

58. Reeves K, Scully R, Dutta A, Bullen-Bull R, Singh M, Chandraharan E. Training and support on Physiological CTG Interpretation: Does it reduce the hypoxic encephalopathy (HIE) rate? European Congress on Intrapartum Care. 2021.

59. Zamora Del Pozo C, Chóliz Ezquerro M, Mejía I, Díaz de Terán Martínez-Berganza E, Esteban LM, Rivero Alonso A, Castán Larraz B, Andeyro García M, Savirón Cornudella R. Diagnostic capacity and interobserver variability in FIGO, ACOG, NICE and Chandraharan cardiotocographic guidelines to predict neonatal acidemia. J Matern Fetal Neonatal Med. 2022; 35(25): 8498-8506.

60. Samyraju M, Ledger S, Chandraharan E. Introduction of the Physiological CTG Interpretation & Hypoxia in Labour (HIL) Tool, and its Incorporation into a Software Programme: Impact on Perinatal Outcomes. Glob J Reprod Med. 2021; 8(3): 5556737.

61. Pasquo E, Ricciardi P, Valenti A, Fieni S, Ghi T, Frusca T. Achieving an appropriate cesarean birth (CB) rate and analyzing the changes using the Robson Ten-Group Classification System (TGCS): Lessons from a Tertiary Care Hospital in Italy. BIRTH-ISS PERINAT C. 2022; 49(3): 430-439.

62. Chandraharan E. Physiological Interpretation of Cardiotocograph: Does the Emerging Scientific Evidence Suggest a Reversal in the "Thunder and Lightning" Phenomenon? J Clin Med Surgery. 2023; 3(1): 1098.

63. Jia YJ, Ghi T, Pereira S, Gracia Perez-Bonfils A, Chandraharan E. Pathophysiological interpretation of fetal heart rate tracings in clinical practice. Am J Obstet Gynecol. 2023; 228(6): 622-644.