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REVIEW ARTICLE

Physiological Interpretation of Cardiotocograph (CTG): The role of the intrapartum "FIT- CAT"

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ABSTRACT

Physiological interpretation of cardiotocograph (CTG) involves determining the combination or sequence of features of the fetal heart rate to recognise different types of fetal hypoxic or non-hypoxic (e.g., chorioamnionitis) stresses, as well as scrutinising the CTG trace to differentiate features suggestive of fetal compensatory responses from decompensation. The Fetal Monitoring Checklist was recommended in 2017, and this has been recently endorsed by the International Expert Consensus Statement on Physiological Interpretation of CTG produced by more than 50 CTG experts from over 20 countries. The aim of the *Fetal Monitoring Checklist* is to exclude or to identify features suggestive of pre-existing fetal compromise by assessing the oxygenation of fetal central organs, placental function and the wider clinical context. This enables the frontline clinicians to ask the question *"Is THIS fetus FIT to undertake the progressively hypoxic journey of human labour?"* However, some pre-existing risk factors may manifest after the onset of established labour due to the super-imposed hypoxic stress. Similarly, intra-amniotic infection following a recent spontaneous prelabour rupture of membranes may be silent during the incubation period, but it may manifest during labour. In such cases, the use of the *Fetal Monitoring Checklist* may lead to the clinicians concluding that the "Fetus is FIT for Labour", although, there is increased risk of fetal compromise with the passage of time and intrapartum super-imposed hypoxic stress. The intrapartum **"FIT-CAT"** (**F**IT for labour -**C**linical **A**nticipation **T**ool) is designed to aid frontline clinicians to anticipate the changes on the CTG which are likely to occur during labour, based on the identified antenatal or intrapartum risk factors. This intrapartum anticipation tool may enable the timely recognition of features on the CTG which heralds the onset of fetal decompensation in fetuses who are deemed "FIT" to commence labour with pre-existing risk factors and/or develop intrapartum risk factors. Prompt recognition and appropriate management based on individualisation of care and the understanding of the impact of clinical context on the CTG trace may help improve perinatal outcomes.

Key Words: Physiological Interpretation of CTG, Chorio Duck Score, FIT for Labour, "How is THIS Fetus?', FIT-CAT, Tone Enhancers.

Introduction

Intrauterine fetal well-being depends on good placental function for gaseous exchange as well as transfer of essential nutrients and removal of fetal metabolic waste products. Optimum intrauterine fetal environment with clear and copious amniotic fluid without any microbial invasion as well as a conducive maternal environment that facilitates fetal growth and wellbeing are equally important. This is because in addition to normal maternal oxygen saturation and normal functioning of the maternal liver, kidneys, lungs, cardiac function with sufficient perfusion pressure to the placenta, the fetus also depends on appropriate "concentration-gradients" for transfer of metabolic byproducts and body heat generated during growth, body movements and metabolism. Therefore, if there is maternal acidosis or pyrexia, this essential maternal-fetal concentration gradient will be blunted leading to accumulation of metabolic waste products and overheating in the fetal compartment, which may lead to fetal neurological damage.

The intrauterine life consists of approximately 40 weeks during the antenatal period and 6-12 hours, if labour commences spontaneously and progresses normally. In addition to chronic utero-placental insufficiency, several metabolic, haematological, cardiovascular, neurological, endocrine and inflammatory disorders may compromise fetal well-being during the antenatal period. Such a fetus with an ongoing, pre-existing, antenatal compromise may not be able withstand additional, super-imposed hypoxic stress caused by intermittent and progressively increasing frequency, duration and strength of uterine contractions during established labour. Moreover, their physiological reserves may have been already exhausted with depletion of the buffering system leading to a shortened "stress to neurological damage" interval. Guidelines which use arbitrary parameters and unscientific time limits may miss such fetuses with pre-existing fetal compromise with blunted protective reflex responses[1,2]. In 2017, Pereira & Chandraharan developed the *"Fetal Monitoring Checklist"*[3] to identify CTG features which were suggestive of pre-existing fetal compromise at the onset of CTG monitoring. This "Fetal Monitoring Checklist" was aimed to enable the frontline clinicians to answer the question "*Is THIS Fetus FIT to undertake the progressively hypoxic journey of labour?"*[4]. This checklist (Table 1) was also recently recommended by the International Expert Consensus Guidelines on Physiological Interpretation of CTG produced by more than 50 CTG experts from over 20 countries[5]. This approach of applying the "Fetal Monitoring Checklist" as a screening tool at the time of initial admission in labour (or prior to an induction of labour) to exclude the features of ongoing fetal compromise due to pre-existing pathology (antenatal causes) will help avoid subjecting these fetuses to additional, super-imposed hypoxic stress and help prevent further injury.

Table 1. Fetal Monitoring Checklist: Is THIS Fetus Fit for Labour? Pereira&Chandraharan 2017

	CTG Features / Risk Factors	Assessment	
1	Baseline fetal heart rate stable and appropriate for the gestational age .	Yes	No
2	Normal variability and cycling	Yes	No
3	Presence of TRUE 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.

However, even the fetuses which are deemed to be "FIT for labour" may have antenatal or de novo intrapartum risks factors which may increase the likelihood of fetal compromise. If there is a delay in recognising these features suggestive of fetal compromise, fetal neurological injury or a terminal myocardial failure may occur. For example, a fetus with the background history of spontaneous prelabour rupture of membranes (SROM) may display a "normal" CTG at the onset of labour and therefore, this fetus will be deemed "FIT for labour". However, the bacteria which had entered the amniotic cavity may be multiplying at this stage and fetal inflammation/ infection due to the entry of bacteria and toxins into lungs during fetal breathing movements resulting in congenital pneumonia, which may occur several hours later resulting in increased fetal metabolic rate. This initial change on the CTG trace (>10% increase in the baseline FHR from 120 bpm to 135 bpm) may be easily missed because several CTG guidelines continue to provide a large "population-based" ranges (110-160 bpm) without individualising care[1,2,6]. A clinical management decision must be made based on the wider clinical context which include the parity, cervical dilatation and the rate of progress of labour. If a timely delivery is not accomplished when features of increased fetal metabolic rate is observed (i.e., > 10% increase in the baseline FHR), then, continued bacterial multiplication and production of bacterial toxins such as lipopolysaccharides (LPS) and inflammatory cytokines such as interleukin-6 (IL-6) may lead to the development of fetal systemic inflammatory response syndrome (FSIRS) resulting in multi-organ failure, neonatal encephalopathy (NNE) and terminal myocardial failure. Recently, it has been shown that features suggestive of fetal inflammation (SOFI) on the CTG trace are associated with approximately four-fold increase in the levels of IL-6 in the umbilical artery at birth compared to the control group and had increased risk of composite adverse perinatal outcomes[7].

Similarly, a fetus with a pre-existing *subclinical* placental failure may not display any abnormalities when screened using the "Fetal Monitoring Checklist" because of the absence of any mismatch between oxygen demand and supply prior to the onset of established uterine contractions with sufficient intensity. However, as the intensity and frequency of uterine contractions increase, due to this episodic reduction of placental perfusion due to compression of the branches of uterine arteries supplying the placental bed, the fetal metabolic and oxygen demands may outstrip the supply. Such a fetus is like to mount compensatory responses to ensure adequate perfusion to the central organs, to deal with these episodes of "relative" utero-placental insufficiency. The cardiotocograph (CTG) features suggestive of an ongoing relative utero-placental insufficiency labour (RUPI-L) may be easily missed if the attending clinicians are not aware of these specific changes[5,8] and may fail to anticipate their occurrence. Similarly, a macrosomic fetus and a fetus with likely placental malfunction (e.g., gestational diabetes mellitus) may experience progressive hypoxic stress after the onset of established labour as the oxygen delivery via the placenta may not be sufficient to meet the metabolic requirements.

In addition to such antenatal pathology which may manifest their detrimental effects on the fetus after the onset of established labour, several intrapartum risk factors may also contribute to or hasten fetal compromise. Passage of meconium during labour leads to reduced phagocytosis because the bile salts and bile acids inhibit the activity of the neutrophils within the amniotic cavity[9], leading to increased risk of chorioamnionitis[10]. Therefore, based on the clinical context (meconium staining of amniotic fluid) it is essential to anticipate and predict specific cardiotocograph (CTG) changes which are likely to occur to ensure timely and appropriate actions are taken to optimise perinatal outcomes.

Conversely, changes in the cardiotocograph (CTG) trace suggestive of fetal compromise may occur secondary to certain medications administered to the mother. For example, administration of opioids

for pain relief or antenatal corticosteroids for fetal lung maturity may stimulate the fetal brain leading to a persistent reduction in the baseline FHR variability[11-14]. Therefore, if these changes are not anticipated and predicted on the CTG after the administration of these medications, based on the knowledge of fetal pathophysiology, then, it may lead to increased likelihood of unnecessary interventions and their undesirable consequences.

Robust Intrapartum Fetal Surveillance (RIFS) does not solely depend on the use and the correct interpretation of the cardiotocograph. It involves a careful scrutiny of the wider clinical context, which includes both antepartum and intrapartum risk factors. In addition, the anticipation and prediction of CTG changes based on the observed clinical context are crucial not only to institute timely and appropriate interventions but also to avoid unnecessary obstetric interventions. This should be done after the fetus has been deemed to be "FIT" for labour, and should continue throughout the intrapartum period, and when additional risk factors are observed, or medications are administered to the parturient.

Aim & Scope

The aim and scope of this expert review is to provide a practical tool to aid frontline clinicians to anticipate the CTG changes which are likely to occur based on the identified antepartum and intrapartum risk factors.

What is FIT-CAT?

It is well recognised that a fetus who is deemed to be "FIT" for labour at the time of admission may not remain "FIT" throughout labour. This is because some of the antepartum risk factors such as entry of bacteria into the amniotic cavity due to spontaneous prelabour rupture of membranes (SROM) may have a delayed manifestation after several hours, and CTG may show changes suggestive of chorioamnionitis in late first stage of labour. Similarly, passage of meconium in early labour may be associated with a normal CTG on admission (i.e., "FIT for labour"), however, with passage of time due to the blunting of the antibacterial effect of the amniotic fluid by the inactivation of neutrophil phagocytosis, the CTG trace may show changes suggestive of chorioamnionitis in late first stage of labour. Moreover, a fetus who was previously deemed "FIT for labour" may be exposed to an intrapartum risk factor such as commencement of oxytocin and resultant reduction in utero-placental oxygenation. However, the features of a gradually evolving hypoxic stress may not be apparent on the CTG trace until a few hours later. Therefore, anticipation of future CTG changes based on the observed risk factors is essential to timely recognition of the significance of the observed CTG changes so that prompt actions can be taken to mitigate the risks of hypoxic-ischaemic encephalopathy (HIE) and/or perinatal death.

The **F**IT for Labour – **C**linical **A**nticipation **T**ool **(FIT-CAT)** has been developed to aid frontline clinicians to anticipate the expected CTG changes (Table 2) based on the observed clinical context. It recognises that the human fetus is exposed to a continuum of risk posed by several underlying pathophysiological processes capable of causing neurological or myocardial damage, both during the antenatal period and as well as extending throughout the intrapartum period until birth. It must be appreciated that not all underlying risk factors will immediately manifest the expected changes on the CTG, and some may start their detrimental effects on the fetal metabolic, biochemical, cardiovascular neurological systems hours later. Some risk factors (e.g., subclinical or relative utero-placental insufficiency) may lead to CTG changes only after the onset of super-imposed uterine contractions with sufficient intensity and/or duration. Therefore, the knowledge of specific CTG changes associated with risk factors which are associated with fetal compromise (Table 1) is essential to anticipate and respond to these changes in a timely manner to optimise perinatal outcomes. The key parameters of the FIT-CAT are given below:

Table 2. FIT for labour-Clinical Anticipation Tool (FIT-CAT)

Risk Factor	Impact on the Fetal pathophysiology	Expected CTG changes and likely underlying mechanisms
F- Fetal disorders	Fetal Growth Restriction	Tardy decelerations or increased baseline with the onset of uterine contractions.
	Oligohydramnios	"Quicklie" deceleration which are often deeper and/or wider. Multiple "single prolonged decelerations", subacute hypoxic pattern during first and second stages of labor.
	Relative uteroplacental insufficiency of labour (RUPI-L) / Late onset placental failure	An abrupt and sustained increase in the baseline with or immediately following uterine contractions, large amplitude "spurious accelerations" coinciding with contractions, ZigZag pattern, subacute hypoxic pattern during the first stage of labour
	Polyhydramnios	Sudden and prolonged deceleration immediately after rupture of membranes (occult or frank cord prolapse)
	Macrosomia	Same changes as RUPI-L (see above)
	Gastrochisis	Reduced baseline FHR variability / lower than expected baseline FHR due to sustained vagal stimulation.
	Post-term	Same changes as RUPI-L
	Spontaneous Rupture of membranes (SROM)	>10% increase in the baseline compared to the initial recording and/or absence of cycling, ZigZag or sinusoidal pattern suggestive of ongoing chorioamnionitis. "Low voltage complexes" suggestive of "myometrial irritability" on the tocograph. Quicklie" deceleration which are often deeper and/or wider. Multiple "single prolonged decelerations", subacute hypoxic pattern during first and second stages of labor due to reduced cushioning effect to protect the umbilcal cord from compression.
I- Intrapartum Risk Factors	Fresh meconium staining of amniotic fluid	>10% increase in the baseline compared to the initial recording and/or absence of cycling, ZigZag or sinusoidal pattern suggestive of ongoing chorioamnionitis. "Low voltage complexes" suggestive of "myometrial irritability" on the tocograph by bile salts and bile acids and/or inflammatory mediators and bacterial toxins.
	Fresh vaginal bleeding	Poole Shark Teeth Pattern due to fetal hypovolemia and hypotension (feto-maternal haemorrhage including ruptured vasa praevia and placental abruption). "Low voltage complexes" suggestive of "myometrial irritability" on the tocograph due to blood seeping into the myometrial fibres giving the classical "Teeth on Teeth" pattern.
	Epidural analgesia	Sudden and prolonged deceleration due to maternal hypotension resulting in an acute reduction in placental perfusion. Reduced baseline FHR variability secondary to fentanyl (opioids) entering the maternal circulation and reaching the fetus via the placenta. An increase in the baseline without preceding decelerations approximately 4-6 hours later due to maternal pyrexia.

Risk Factor	Impact on the Fetal pathophysiology	Expected CTG changes and likely underlying mechanisms
	Occipito-posterior (OP) position	A sudden and sustained drop in the baseline FHR in late first stage or second stage of labour as the deflexed fetal head attempts to enter the maternal bony pelvis compressing the eyeballs/ anterior fontanelle which have rich innervation of parasympathetic fibres. In the absence of hypoxic stress, the baseline FHR variability remains normal during ongoing baseline bradycardia/ drop in the baseline. Often, there will be spontaneous normalisation of baseline bradycardia with flexion of the fetal head.
	Commencement of active maternal pushing	Subacute hypoxic pattern and/or ZigZag pattern due to an increase in fetal hypoxic stress with maternal Valsalva manoeuvre either de novo or following the previously observed features of gradually evolving hypoxic stress
	Vacuum birth	A sudden increase in the baseline FHR immediately after the creation of chignon with the vacuum cup most likely secondary to the activation of the sympathetic nervous system in response to pain/pressure
	Forceps Birth	A sudden and prolonged deceleration and/or ZigZag pattern most likely due to the increased intracranial pressure when the forceps blades are locked.
T- Tone enhancers	Prostaglandins	Recurrent decelerations / a prolonged deceleration/ subacute hypoxic/ZigZag pattern due to increased uterine activity (frequency, duration, strength and basal tone). Tocograph shows evidence of excessive uterine activity and/or reduced inter-contraction interval.
	Oxytocin	Evidence of gradually evolving hypoxic stress, a prolonged deceleration/ subacute hypoxic pattern/ZigZag pattern even during the first stage of labour due to increased uterine activity (frequency, duration, strength and basal tone). Tocograph shows evidence of excessive uterine activity and/or reduced inter-contraction interval
	Blood	Low voltage complexes or increased duration / frequency of contractions on the tocograph due to the irritation of myometrium by blood.
	Infection	Low voltage complexes or increased duration / frequency of contractions on the tocograph due to the irritation of myometrium by bacterial toxins, inflammatory cytokines or bile acids (meconium). In late stages of chorioamnionitis, due to the inflammation mediated membrane instability / onset of lactic acidosis within the myometrium, there may be cessation of uterine activity.
C-Chorioamnionitis	History of Group -B Streptococcal (GBS) colonization	>10% increase in the baseline compared to the initial recording and/or absence of cycling, ZigZag or sinusoidal pattern suggestive of ongoing chorioamnionitis.

Risk Factor	Impact on the Fetal pathophysiology	Expected CTG changes and likely underlying mechanisms
	History of insertion of cervical balloon catheter/membrane sweep / prelabour SROM History of gestational diabetes or medications which impair immunity to infections Presence of "old" or fresh meconium with impaired amniotic fluid phagocytic activity	FHR overshoots due to spasm of umbilical blood vessels due to funisitis. "Low voltage complexes" suggestive of "myometrial irritability" on the tocograph inflammatory cytokines such as interleukin-6 (IL-6) and bacterial toxins and/or by bile salts and bile acids in meconium seeping into the choriodecidual space. In clinical chorioamnionitis and transplacental transfer of bacterial toxins and inflammatory cytokines, a reactive fetal tachycardia may occur with absence of FHR cycling.
A-Adverse Maternal Environment	Pyrexia – epidural/ infection / inflammation	Reactive increase in the baseline FHR, > 10% higher than the previously recorded rate or frank fetal tachycardia (>160 bpm) without preceding repetitive decelerations. The increase in the baseline is often accompanied by absence of FHR cycling. In intra-amniotic inflammation / infection, evidence of myometrial irritability may be present.
	Dehydration	Reactive increase in the baseline FHR, > 10% higher than the previously recorded rate or frank fetal tachycardia (>160 bpm) without preceding repetitive decelerations. Normal FHR cycling is seen
	Diabetes Mellitus	An abrupt and sustained increase in the baseline with or immediately following uterine contractions, large amplitude "spurious accelerations" coinciding with contractions, ZigZag pattern, subacute hypoxic pattern during the first stage of labour due to RUPI-L. There is a mismatch between oxygen supply (abnormal, immature villi with placental infarction, thrombosis) and increased demand (fetal macrosomia) which results in poor tolerance to superimposed hypoxic stress. If there is fetal diabetic cardiomyopathy, an unstable baseline may be observed. Features of chorioamnionitis may be observed in cases of prolonged labour
	Keto-acidosis	Reactive increase in the baseline FHR, > 10% higher than the previously recorded rate or frank fetal tachycardia (>160 bpm) without preceding repetitive decelerations. Reduced baseline FHR is often observed due to the transplacental passage of maternal ketoacids and resultant depression of the fetal central nervous system.
	Immunological disorders (SLE)	Evidence of congenital heart block characterised by abrupt, and episodic fall in the fetal heart rate from the baseline with abrupt recovery with normal variability during these apparent "decelerations". This is episodic abrupt and recurrent decrease

Risk Factor	Impact on the Fetal pathophysiology	Expected CTG changes and likely underlying mechanisms
		in the FHR is due to maternal anti-Ro and anti-La antibodies crossing the placenta and then binding to fetal cardiac conduction tissues, leading to the disruption of electrical impulses reaching the ventricles. In long standing cases, fibrosis of the fetal cardiac conduction system may lead to a baseline fetal bradycardia.
	Obstetric Cholestasis	Spurious accelerations and FHR overshoots due to spasm of umbilical blood vessels due to the irritant effect of bile salts and bile acids on vascular smooth muscles. Sudden and profound deceleration due to sustained umbilical vascular spasm or fetal myocardial dysfunction due to the toxic effects of bile salts and bile acids.
	Renal/hepatic disorders	Reduced baseline FHR variability due to renal / hepatic acidosis depressing the fetal CNS.
	Parvoviral infection	Fetal bone marrow suppression leading to fetal anaemia and acidosis and the onset of the "typical" sinusoidal" pattern. Direct myocardial toxicity may result in reduced cardiac output and the onset of ZigZag patterns due to a rapid reduction in carotid perfusion and/or a baseline bradycardia.
T-Treatment (effect of medications & intrapartum procedures)	**Mechanism of Action (effects on the fetal heart/brain)**	**Expected CTG changes**
Betamimetics	Stimulation of the myocardium by exerting inotropic and chronotropic effects through the Beta-1 adrenoceptors.	Increased baseline FHR / fetal tachycardia
Atropine	Blocks the muscarinic acetyl cholinergic receptors, and attenuates the effect of the parasympathetic nervous system	Increased Baseline FHR due to the unopposed activity of the sympathetic nervous system and reduced baseline FHR variability. Decelerations, which are vagally mediated, may not occur despite ongoing hypoxic stress.
Labetalol	Nonspecific alpha- and beta-adrenergic receptor blocker. Therefore, it blocks the effect on the sympathetic nervous system on the heart ($\beta 1$ receptors), smooth muscles of blood vessels ($\alpha 1$ receptors), and glycogenolysis from the liver and myocardium ($\beta 2$ receptors).	Unable to increase baseline FHR when exposed to hypoxic stress Unable to re-distribute and centralise blood flow (alpha-1 receptor blockage) leading to loss of baseline FHRV without the preceding increase in the baseline FHR. Unable to breakdown cardiac glycogen to glucose thorough the process of glycogenolysis, which is mediated through $\beta 2$ receptors. Therefore, due to the lack of sufficient energy substrate (availability of glucose) the ability to deal with hypoxic stress may be blunted, especially in fetuses with FGR with reduced glycogen reserves.

Risk Factor	Impact on the Fetal pathophysiology	Expected CTG changes and likely underlying mechanisms
Propranolol	Nonspecific beta adrenergic blocker	Reduced baseline FHR variability / fetal bradycardia due tot the blockage of $\beta 1$ receptors in the myocardium. Unable to breakdown cardiac glycogen to glucose thorough the process of glycogenolysis, which is mediated through $\beta 2$ receptors.
Opiates	Depression of the fetal central nervous system	Reduced baseline FHR variability, and due to the depression of somatic nervous system, reduced or absence of true fetal heart rate accelerations.
Alpha Methyl Dopa	Centrally acting sympatholytic drug which reduces central sympathetic outflow CNS depressant	Reduction in the baseline FHR due to sympathetic blockage and reduced baseline variability due to CNS depression
Antenatal Corticosteroids	Depression of the fetal autonomic nervous system (brain stem)	Reduced baseline FHR variability without preceding repetitive decelerations and/or an increase in the baseline FHR
Magnesium sulphate	Depression of the Fetal Brain	Reduced baseline FHR variability, and loss of accelerations due to the depression of the fetal autonomic nervous system
Vacuum extractor	Initial stimulation of the vagus when the vacuum cup is applied likely due to the stimulation of parasympathetic free nerve endings in the skin of the scalp, followed by stimulation of the sympathetic nervous system when the scalp tissue is sucked into the vacuum cup (i.e., creation of chignon) due to pain /pressure	Initial single prolonged deceleration and/or an abrupt increase in the fetal heart rate
Forceps Birth	Increased intracranial pressure and stimulation of the autonomic nervous system as the forceps blades are locked.	ZigZag pattern associated with a deceleration/ increased baseline FHR depending on which component of the autonomic system is predominantly stimulated after the locking of the forceps blades

a. FETAL DISORDERS

Several pathophysiological processes affecting the fetus may lead to specific changes in the fetal heart rate, based on the underlying pathology, as the labour progresses. These changes may manifest at any time during labour. For example, a fetus with growth restriction with mild to moderate utero-placental insufficiency may tolerate ongoing relative milder uterine contractions during the latent phase and early active phase of labour due to the availability of sufficient reserves. CTG changes suggestive of a gradually evolving hypoxic stress may be observed during late first stage or second stage of labour. Conversely, a fetus SGA with moderate to severe loss of placental function secondary to placental infarction or thrombosis

during the antenatal period may show features suggestive of hypoxic stress even during the latent phase of labour. Well grown fetuses with subclinical placental insufficiency (e.g., loss of approximately 10-30% of placental function) or those with late onset placental failure may not demonstrate any abnormalities in the fetal heart rate at the onset of labour. However, if attempts are made to induce labour or in the presence of established uterine contractions, these fetuses may show CTG changes suggestive of a relative uteroplacental insufficiency of labour (RUPI-L).

Fetuses who are "large for dates" (LFD) or "macrosomic" may have a critical balance between their oxygen supply from the placenta and their (increased) metabolic demands during the antenatal period. This is because relatively larger organs, increased muscle mass and adipose tissue require extra oxygen to meet their metabolic requirements to avoid anaerobic metabolism and development of lactic acidosis. However, this "critical balance" may be lost with the onset of uterine contractions with sufficient intensity and duration to reduce placental oxygenation leading to the ongoing increase in the fetal metabolic demands outstripping the blood supply. Therefore, features suggestive of a gradually evolving hypoxic stress may be seen on the CTG trace. However, if there is a rapid evolution of hypoxic stress, then, a ZigZag pattern[15,16] may be seen. Post term fetuses may also demonstrate similar CTG features due to the relative larger size of the fetus as the gestation advances associated with a progressive decrease in the placental function.

Oligohydramnios usually indicates a severe chronic utero-placental insufficiency with renal shutdown (i.e., leading to a reduced fetal urine output). Therefore, most fetuses would show features of "chronic hypoxia" on the CTG trace and would be deemed "Not FIT" for labour when the Fetal Monitoring Checklist is used. However, if uterine contractions are allowed to continue in fetus with oligohydramnios, then, due to the lack of sufficient amniotic fluid "cushion" to protect the blood vessels within the umbilical cord from being compressed during uterine contractions, then features of a gradually evolving hypoxic stress may be observed. However, in late first stage of labour or if uterotonic agents are used, then, a subacute or acute hypoxic patten.

Fetuses with abdominal wall defects would have exposure and irritation of the contents of the peritoneal cavity, especially the intestines, which are richly supplied by the parasympathetic nervous system. Ensuing sustained vagal stimulation may lead to a persistent and sustained drop in the baseline FHR and/or a reduction in the baseline variability. In modern obstetric practice, these fetuses are likely to be diagnosed during antenatal ultrasound scans leading an elective caesarean section. However, if these fetuses are exposed to uterine contractions during labour, then, "deep and ugly" decelerations may occur due the ongoing vagal stimulation and compression of the exposed abdominal contents as well as the parietal peritoneum during uterine contractions.

b. INTRAPARTUM RISK FACTORS

Unfortunately, despite the superficial attempts to consider the "wider clinical picture" whilst interpreting intrapartum CTG traces and introducing acronyms such as "DR C BRAVADO", the same CTG classification tool has been used in all human fetuses[17]. This illogical practice of "defining the risk" (DR) and then illogically using exactly the same number of contractions and CTG features with the same arbitrarily pre-defined time duration (C BRAVADO) has resulted in disastrous consequences to babies and their families[18]. One cannot use the same cut off for the upper limit of the baseline FHR (>160 bpm) with the same range of 110-160 bpm in all human fetuses because a post-term or a fetus with reduced physiological reserves who increases the baseline from 120 bpm to 140 bpm due to ongoing hypoxic or inflammatory stress will be missed. Classification these fetuses by using the same range for the baseline (110-160 bpm) will miss these fetuses who have increased the baseline from 120 to 140 bpm,

leading to disastrous consequences[19]. It is regrettable that some CTG guidelines even experimented with human physiology by artificially increasing the upper limit of the baseline FHR to > 180 bpm[20,21] making it impossible for fetuses with poor physiological reserves to ever become "abnormal". This unscientific, illogical and avoidable-harm-inducing action would lead to an increase in avoidable poor perinatal outcomes[22]. The continued use of such illogical CTG guidelines which failed to consider specific CTG changes induced by the intrapartum clinical risk factors has been associated with repetitive publications highlighting avoidable poor perinatal outcomes in approximately 70% of cases[23-28]. In addition, the medico-legal costs due to settling the clinical negligence claims involving avoidable harm in the maternity service have been eye-wateringly high, dwarfing other "high-risk" clinical specialties[22,29.] According to the latest report from NHS Resolution, a body responsible for indemnifying the National Health Service from clinical negligence claims has highlighted that claims due to cerebral palsy and severe brain damage, the vast majority of which are caused by CTG misinterpretation, cost the tax payer in the United Kingdom approximately £6.7 million, *every day*[30].

Therefore, a deeper understanding of the intrapartum clinical risk factors and the specific anticipated changes in the CTG trace in response to these risk factors is essential to avoid poor perinatal outcomes (Table 1). A thorough scrutiny of both cardiac (fetal heart rate changes) and toco (uterine activity) parts of the CTG trace are essential whenever an intrapartum risk factor is identified. For example, intrapartum passage or detection of meconium may lead to the obliteration of the antibacterial activity of the amniotic fluid resulting in an intraamniotic infection[31,32] characterised >10% increase in the baseline compared to the initial recording and/or absence of cycling, ZigZag or sinusoidal pattern. However, in early stages, bile salts and bile acids present within meconium may cause "low voltage complexes" suggestive of "myometrial irritability" on the tocograph (Table 1). Therefore, lack of knowledge to anticipate these changes based on the understanding of fetal pathophysiology may lead to delays in appropriate action and poor perinatal outcomes.

c. TONE ENHANCERS

It is important to appreciate that intrapartum fetal wellbeing depends on the ability of a fetus to mount an effective compensatory response to slow and a progressive increase in hypoxic stress, with sufficient relaxation time, in between the contractions, to obtain fresh oxygen and to eliminate carbon dioxide and other metabolic by-products from the fetal compartment. Therefore, any agent which increases the uterine tone would significantly increase ongoing hypoxic stress, and impairment of fetal oxygenation and may cause rapid decompensation. Unfortunately, several CTG guidelines erroneously defined uterine hyperstimulation based on the frequency of uterine contractions alone[19-21] due to the apparent lack of knowledge of muscle physiology. Properties of any muscle contraction include frequency, duration, strength and resting tone, and therefore, focussing solely on the frequency > 6 in 10 minutes would lead to adverse outcomes in fetus exposed to < 5 contractions in 10 minutes. If these uterine contractions had longer duration, increased intensity or an enhanced resting tone in between uterine contractions, then, rapid fetal decompensation may occur culminating in hypoxic-ischaemic encephalopathy (HIE). Therefore, it is not surprising that repetitive Each Baby Counts Reports published by the Royal College of Obstetricians & Gynaecologists (RCOG) has highlighted the association of oxytocin use in cases of intrapartum and early neonatal deaths as well as severe hypoxic-ischaemic brain injuries[25-28]. Approximately 70% of medico-legal cases involving cerebral palsy have been reported to be associated with injudicious use of oxytocin[33]. Therefore, it is essential to use the correct definition of uterine hyperstimulation whilst using uterotonic agents, and any increase in the uterine activity (frequency, duration, strength and resting tone) associated

with changes in the fetal heart rate should be considered as uterine hyperstimulation[34].

In addition to the use of prostaglandins and oxytocin infusion to induce and augment labour, respectively, both blood (e.g., placental abruption), as well as inflammatory cytokines and bacterial toxins (chorioamnionitis) can irritate and stimulate the myometrium. Irrespective of the underlying pathophysiology, increased uterine tone over time will result in progressive reduction in fetal oxygenation. Therefore, anticipation of specific changes (Table 1) is crucial to optimise perinatal outcomes, and if a spontaneous vaginal birth is not imminent, and the underlying pathology is irreversible (e.g., placental abruption or chorioamnionitis), then, birth should be expedited.

d. CHORIOAMNIONITIS

The spectrum of intraamniotic inflammation, intraamniotic infection and chorioamnionitis occurs in approximately 10% of labour and it is an important cause of fetal compromise[35,36]. It has been reported that chorioamnionitis increases the risk of neonatal encephalopathy (NNE) five-fold and approximately 10% of all cases of cerebral palsy were due to chorioamnionitis[37,38]. Several animal experimental studies have confirmed that injection of bacterial toxins such as the lipopolysaccharides (LPS) are associated with perinatal brain damage[39-41]. Galli et al[42] analysed 2105 cases of both subclinical and clinical chorioamnionitis and reported specific CTG features associated with chorioamnionitis such as > 10% increase in the baseline FHR, absence of FHR cycling, the ZigZag and sinusoidal patterns[42]. These were subsequently confirmed by Sukumaran et al, who analysed histologically confirmed cases of chorioamnionitis and funisitis[43]. Similar features were reported in fetuses exposed to maternal cytokine storm in Covid-19 infection[44].

Therefore, if these features suggestive of an ongoing fetal inflammation are present, then, co-existing or superimposed hypoxic stress should be avoided and delivery should be expedited. This is because several animal experimental studies have confirmed that in the presence of fetal inflammation and consequent sensitization of the developing fetal brain, the threshold for hypoxic-ischaemic injury is lowered[45-49]. In other words, if uterine contractions (super-imposed hypoxic stress) are continued in a fetus with ongoing chorioamnionitis, then, due to the synergistic effect whereby ongoing fetal neuroinflammation reduces the threshold at which hypoxic-ischaemic brain injury occurs, the risk of brain injury and cerebral palsy will be potentiated[50].

Despite this scientific knowledge several CTG guidelines had failed to include the features of chorioamnionitis in their classification system. For example, repetitive NICE CTG guidelines, did not include any of the features suggestive of chorioamnionitis, but also erroneously increased the upper threshold of abnormal FHR baseline from the internationally accepted 160 bpm to 180 bpm[20,21]. Moreover, they had continued to recommend fetal scalp blood sampling (FBS) until 2022, despite the physiological knowledge that in chorioamnionitis, due to peripheral vasodilation, there would be excessive blood flow to the skin, which is a peripheral non-essential tissue, leading to falsely reassuring, false negative result. Despite these risks being highlighted by scientific publications[2,51,53], FBS was continued to be recommended in the UK contrary to available scientific evidence until 2022. Therefore, it is not surprising that due to the lack of due diligence regarding the specific features of chorioamnionitis by the NICE CTG Guideline, NHS Resolution Report in 2019 has highlighted that more than 15% of neonates who required brain cooling for severe brain injury at birth had evidence of infection [30]. Recent scientific evidence has confirmed that fetuses with CTG features suggestive of fetal inflammation (SOFI) such as > 10% increase in the baseline FHR, absence of FHR cycling, the ZigZag and sinusoidal patterns are associated with approximately a four-fold increase in the levels of interleukin-6 (IL-6) which is a marker of fetal inflammation [54]. Chandraharan & Bolten published a "Chorio Duck Score" (CDS), and

recommended that if the CDS > 5, the overall clinical context should be considered and delivery must be expedited if a spontaneous vaginal birth is not imminent[55]. Table 3 highlights the rationale for the parameters included in the Chorio Duck Score.

Table 3. Clinical Rationale of the Chorio-Duck Score (CDS)

Parameter	Clinical Rationale: Why do these changes occur?
Increase in the Baseline FHR	An increased demand of oxygen in the presence of infection in a rise in the baseline heart rate. This helps to maintain aerobic metabolism by increasing oxygen delivery to tissues and organs to prevent metabolic acidosis, however, it comes at the expense of increased myocardial workload and augmented cardiac oxygen demand.
Features of Neuroinflammation	Reduced variability at a higher-than-expected baseline and absence of accelerations and cycling are a result of neuroinflammation which occurs early in FIRS due to the absence of a blood-brain barrier in the fetus.
Meconium	The presence of Meconium can alter antibacterial effect of the amniotic fluid by inhibiting the phagocytotic function of neutrophiles and phagocytes. Meconium impairs the normal protective effect of cells which fight bacterial infections, and therefore predisposes the fetus to ascending infections. Likewise, chorioamnionitis can cause the passage of meconium by fetal gastroenteritis caused by ingestion of infected amniotic fluid as well as through increasing acidosis in the presence of FIRS. The developing acidosis when the higher oxygen demand in sepsis is not met triggers the chemoreceptors which alongside other reactions trigger a strong vagal response that activates peristalsis and relaxes the muscle of the anal sphincter.
Myometrial Irritability	The majority of women with subclinical acute chorioamnionitis demonstrate extension of the inflammation into the choriodecidual space and the myometrium, which can result in tissue damage and uncontrolled spasms /irritability of the myometrium.
Maternal Parameters	In the vast majority of cases, chorioamnionitis occurs as a result of an ascending infection through the cervical canal. Only in late stages of FIRS does the infection extend to the mother directly through the umbilical arteries or via the decidual vessels. The mother will then start showing signs of sepsis with an increase in heartbeat and pyrexia which, if untreated, may to low blood pressure.

It is indeed very regrettable that even the recently updated NICE CTG Guideline has chosen to disregard the features suggestive of inflammation (SOFI) in the classification tool[56]. This has serious consequences for fetuses with intraamniotic inflammation or infection and is very likely that NHS Resolution Reports and the Each Baby Counts Reports in the future would continue to report avoidable brain damage due to fetal infection. Lack of knowledge and disregarding scientific evidence should have a "Zero tolerance" approach in obstetric practice. Therefore, the authors strongly recommend that chorioamnionitis should be considered as an important intrapartum risk factors which increase the likelihood of fetal compromise. And any features suggestive of SOFI or myometrial

irritability requires an immediate, careful review and the most appropriate obstetric intervention based on the progress of labour. Figure 1 illustrates > 10% increase in the baseline FHR without repetitive decelerations, absence of cycling and presence of the "Poole Shark Teeth Pattern" six hours after spontaneous rupture or membranes. The use of traditional CTG guidelines classifying CTG traces into "Normal, suspicious and Pathological", focussing on the morphology of decelerations would miss such fetuses. However, the use of the intrapartum FIT-CAT may help frontline clinicians to anticipate such changes, facilitating timely intervention to avoid super-imposed hypoxic stress.

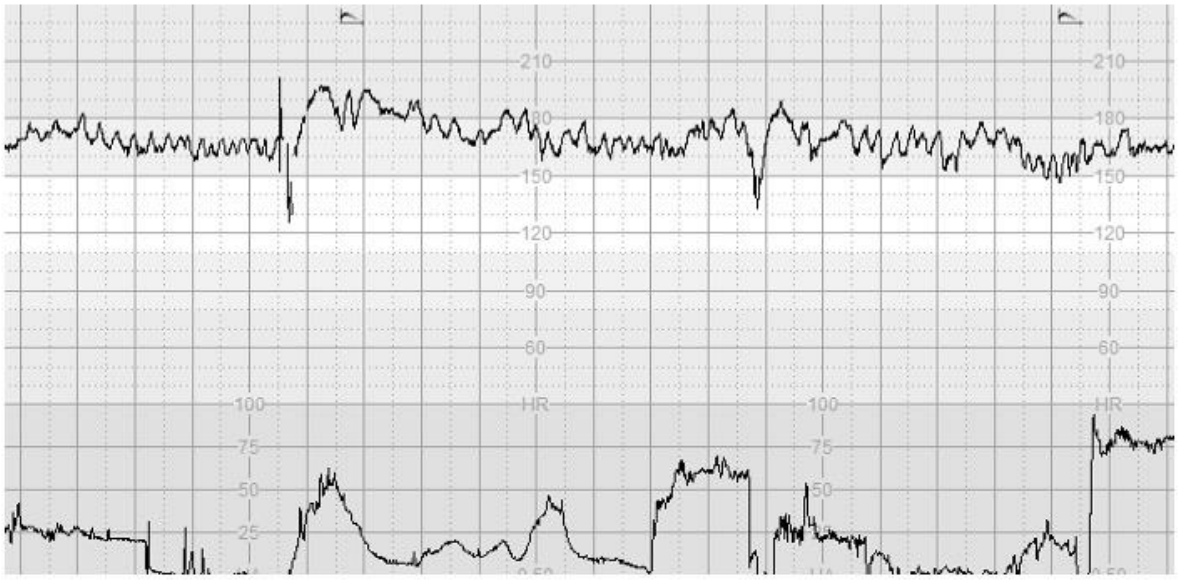

Figure 1. CTG trace illustrates > 10% increase in the baseline FHR without repetitive decelerations, absence of cycling and presence of the "Poole Shark Teeth Pattern", which are the features of SOFI, following SROM

e. ADVERSE MATERNAL ENVIRONMENT

The immediate "external" environment of a fetus is the maternal milieu comprising of haematological, endocrine, metabolic, inflammatory, cardio-respiratory, excretory and immunological components. Therefore, derangements in any of the above systems can adversely affect intrauterine fetal wellbeing and may result in changes in the CTG (Table 1). For example, a mother with systemic lupus erythematosus (SLE) may transfer the Anti-Ro and Anti-La (or SS-A and SS-B) antibodies via the placenta and the umbilical cord into the fetal compartment[57]. These abnormal antibodies have a special affinity to the fetal cardiac conductive tissue (Bundle of His, Bundle Branches and Purkinje Fibres) leading to a reduction in transmission of electrical impulses from the SA node to the ventricles resulting in congenital heart block (1:1, 1:2 or 1:3). However, in some cases, due to the prolonged exposure and resulting fibrosis and calcification of the cardiac conducting tissue following the initial inflammatory response can result in a baseline bradycardia. Similarly, maternal parvovirus infection may lead to the transplacental passage of the parvovirus into the fetal compartment. Parvovirus preferentially attacks two fetal organs: the bone marrow and the myocardium leading to bone marrow suppression causing chronic fetal anaemia and acidosis culminating in fetal cardiac failure, and direct myocardial suppression[58]. Therefore, one should carefully scrutinise the CTG trace for the "typical sinusoidal" pattern (chronic fetal anaemia and acidosis) or a progressive reduction in the baseline FHR (direct myocardial depression). Simply listing "Parvovirus" as the "defined risk" of the illogical "DR C BRAVADO" methodology and then ignoring the specific features due to this clinical risk factor, due to the lack of knowledge of fetal pathophysiology should no longer be acceptable

in modern obstetric practice. Recently, progressive fetal encephalopathy has been reported, and therefore, it is essential to exclude absence of fetal heart rare cycling[59].

It is also important to appreciate that the fetus depends on the concentration gradient for the transfer of essential nutrients from the mother and metabolic byproducts to the mother via to the placenta. Therefore, in maternal pyrexia and maternal acidosis, this essential concentration gradient would be adversely affected, resulting in accumulation of heat and metabolic acids and other waste products within the fetal compartment. The CTG trace is likely to show reduced variability in fetal acidosis and absence of cycling due to the disruption of the temperature gradient in maternal pyrexia[60,61]. Table 4 illustrates the negative impacts of an adverse maternal environment.

Table 4. Impact of persistent adverse maternal environment with super-imposed intrapartum hypoxic stress

Observed Abnormality	Expected impact on the fetus	Likely outcome
Persistent fever	Increased metabolic rate and tissue oxygen demands Neuronal dysfunction/ denaturation of proteins	Increased predisposition for hypoxic-ischaemic brain injury and multi-organ failure. Neonatal convulsions and neonatal encephalopathy (NNE)
Persistent tachycardia	Increased oxygen requirement of the myocardium due to increased myocardial workload. Reduced coronary perfusion due to reduced diastole leading to progressive myocardial ischaemia	Fetal "high output" heart failure leading to terminal bradycardia.
Persistent hypoxia (bronchial asthma, Covid-pneumonia)	Reduced placental oxygen transfer leading to hypoxia to fetal tissues and organs	Increased predisposition for hypoxic-ischaemic brain injury and multi-organ failure
Persistent maternal hypotension	Reduced perfusion pressure to the placental bed resulting in rapid development of hypoxia-ischaemia to fetal central organs	Increased predisposition for hypoxic-ischaemic brain injury and multi-organ failure
Persistent maternal hepatic/ renal acidosis	Disruption in the materno-fetal concentration gradient required for transfer across the placenta, leading to accumulation of acid in the fetal compartment	Neonatal encephalopathy and terminal myocardial failure due to acidosis
Persistent obstetric cholestasis	Toxicity to smooth muscles leading to spasm of the blood vessels within the umbilical cord and/or depression of fetal myocardium	Terminal bradycardia and intrauterine fetal death

f. TREATMENT (EFFECT OF MEDICATIONS & INTRAPARTUM PROCEDURES)

Several medications which are administered to the mother may cross the placenta and affect the fetal cardiovascular and central nervous systems resulting changes in the CTG trace (Table 2). Failure to understand the specific CTG features secondary to the medications administered to the mother may lead to over-reaction to observed patterns resulting in unnecessary operative interventions. For example, reduced baseline FHR variability due to the depression of fetal CNS by antenatal corticosteroids may lead to the classification of the cardiotocograph (CTG) trace as "pathological" due to the illogical, arbitrary time limit of 90 minutes stipulated by some CTG

guideline. This may result in an unnecessary, iatrogenic premature delivery with resultant complications.

Conversely, it must be appreciated that some medications may blunt the anticipated fetal heart rate changes in s gradually evolving hypoxia, and failure to appreciate this may lead to disastrous consequences. For example, nonspecific beta-blockers such as propranolol and labetalol may blunt the fetal catecholamine response and resultant increase in the baseline FHR due to the blockage of cardiac β1 receptors. Therefore, the baseline FHR variability may be reduced immediately following repetitive decelerations without an increase in the baseline FHR. Labetalol also blocks α-1 receptors which are responsible for peripheral vasoconstriction and centralisation of blood flow. Therefore, blunting of redistribution due to labetalol may lead to rapid onset of decompensation with the onset of reduced baseline FHR variability without a compensatory increase in the baseline FHR.

Management

Individualisation care is the cornerstone of clinical practice, and in a medical ward, a physician or a nurse would never treat all adults with tachycardia with the same medication. This is because baseline tachycardia in adults may be caused by thyrotoxicosis, sepsis, haemorrhage, stress, anxiety, anaemia and dehydration. Therefore, it would be illogical to administer fluids to all adults with tachycardia, and the treatment must be aimed at rectifying the underlying pathology which caused tachycardia. Unfortunately, several CTG guidelines overlooked this very basic tenet of individualisation of care taught in medical schools, and arbitrarily grouped FHR features into different categories, without any scientific evidence with the overall classification of CTG traces into "Normal, suspicious, Pathological". Illogically, these guidelines recommended oral or intravenous fluids for all suspicious CTG traces[20,21], which increased the risk of fetuses with chorioamnionitis sustaining brain damage or dying due to the delay in definitive treatment. Therefore, it is not surprising that repetitive Each Baby Counts Reports[25-28] concluded that in more than 70% of babies who sustained severe brain damaged and/or died following intrapartum hypoxia, a different care would have given rise to a different outcome. Similarly, the NHS Litigation Authority (NHSLA) Study on Stillbirths had concluded that 34% of stillbirths were solely due to CTG misinterpretation[28]. The same poor outcomes would be expected in an adult or in a paediatric ward, if the doctors and nurses had treated all cases of tachycardia with intravenous or oral fluids, disregarding the underlying pathology.

Similarly, some CTG guidelines illogically recommended fetal scalp blood sampling (FBS) for all pathological CTGs which resulted in arbitrarily grouping random features into different categories, irrespective of underlying pathology and despite the serious concerns raised by several publications[51-53,62]. This resulted in fetuses who were well compensating to ongoing hypoxic stress by effectively re-distributing oxygenated blood from the scalp, due to catecholamine-mediated peripheral vasoconstriction being misclassified as "fetal distress", leading to an increase in the rate of unnecessary intrapartum operative interventions to the mother and resultant potentially serious complications. Despite FBS being stopped in the USA approximately 30 years ago due to lack of scientific evidence to support the use, and obstetricians from most countries around the world not doing FBS due to basic knowledge of fetal physiology, FBS was continued to be used in the UK until December 2022[63]. This was despite repetitive Cochrane Systematic Reviews concluding no beneficial effects of FBS[64], and several publications highlighting increased rate of emergency caesarean sections without improving perinatal outcomes[65,66], including a multicentre trial from the UK concluding lack of improvement in perinatal outcomes, and an approximately 60% increase in the perinatal outcomes[67]. These poor maternal and perinatal outcomes for treating all fetal heart rate changes with the same treatment

(fluids/ FBS/ caesarean sections) should no longer accepted in contemporary obstetric practice. The medico-legal and ethical pitfalls of treating all human fetuses with the same pre-defined parameters have been repetitively highlighted[1-3,68]. However, no action was taken to remove the error-producing CTG guidelines which were causing avoidable poor perinatal outcomes, resulting in women and their families calling for criminal prosecutions in 2022 ((https://www.independent.co.uk/news/health/east-kent-maternity-baby-deaths-b2206143.html) as well as a public enquiry ((https://www.theguardian.com/society/2023/oct/31/parents-of-babies-who-died-or-were-harmed-in-nhs-care demand-inquiry). The unscientific CTG guidelines which failed to individualise care were continued despite of continuing poor perinatal outcomes, and 100 cases have been referred to the police in 2024 (https://www.bbc.com/news/articles/cpwre51d22yo) .

Therefore, frontline clinicians must individualise care based on the observed clinical context during labour because each fetus is different with different physiological reserves and different frequency, strength, duration and the basal tone of ongoing uterine contractions. Moreover, the intrauterine risk factors and maternal environment are also different. Therefore, in additional to the classification and management recommended by the International Expert Consensus Statement on Physiological Interpretation of CTG [5], the expected fetal heart rate changes based on the observed clinical context should be anticipated so that prompt recognition and timely action will help improve outcomes. Table 5 provides examples of how management can be tailor-made to the identified clinical context, and this should be used in conjunction with the recommended intrapartum fetal assessment tool (Table 6).

Table 5. Immediate recommended actions by obstetricians and midwives

Clinical Context	Immediate Actions	Rationale
Prolonged deceleration following the administration of epidural analgesia	**Call for help** – emergency bell by the end of 2 minutes Turn to the left lateral position Maternal observations every 5 minutes **SBAR your concern:** **Request anaesthetist** attendance to consider ephedrine bolus Correct any blood pressure drop through drugs and fluids Ensure close observation of fetal heart and maternal blood pressure **Senior Obstetric attendance:** Observe for improvement If no improvement at 6 minutes plan for delivery If no recovery by 9 minutes, commence operative birth by 12 minutes.	90% of the decelerations usually recover because they are secondary to maternal hypotension secondary to epidural (or spinal) analgesia. This could be due to the position (supine hypotension and/or compression of the uterine blood vessels by the fetal head as the woman bends forwards during siting of the epidural catheter or due to sympathetic neural blockage leading to maternal vasodilation and relative hypovolemia and hypotension. If no recovery may be due to other factors. This is a reversible cause resolved by rapid fluid administration ± I.V. ephedrine bolus (by the anaesthetic team). (Chandraharan et al (2024) https://www.sciencedirect.com/science/article/pii/S0301211524005281#t0005 If the observed prolonged deceleration persists despite the restoration of the circulating volume and normalisation of maternal blood pressure, other co-existing pathology such as umbilical cord prolapse, placental abruptio and uterine rupture must be considered. Provide an overall clinical review and actions Consider other predisposing factors/causes: SGA/Reduced growth restriction GDM Reduced movements

Clinical Context	Immediate Actions	Rationale
A sudden and prolonged decelerations following spontaneous or artificial rupture of membranes in polyhydramnios	Emergency bell/ buzzer: Request obstetric and anaesthetic team SBAR situation Check for cord prolapse and relieve head off the cord Expediate delivery and ensure timely delivery within 15 minutes.	Cord prolapse is an irreversible cause. Umbilical arterial pH is expected to drop by 0.01 every minute. (Neonatal outcome will be determine by the condition of the fetus/ depletion of buffers prior to the onset of prolonged decelerations, and fetal reserves) Scientific evidence suggests that delivery beyond 17 minutes in acute hypoxia is associated with poor perinatal outcomes.
Poole Shark Teeth Pattern observed following spontaneous or artificial rupture of membranes	Deliver immediately Immediate senior obstetrician review Senior Midwife review	Suspected sudden loss of blood due to ruptured vasa praevia. This leads to an acute fetal haemorrhage, acute fetal anaemia, hypovolemia and hypotension. Failure to accomplish birth immediately may increase the risk of hypoxic-ischaemic encephalopathy (HIE) and perinatal death.
A prolonged deceleration observed when the mother lies on her left side	Change position Stop oxytocin Observe for the 3 accidents Expediate delivery if no improvement at 6 minutes Deliver by 12 minutes	Sustained cord compression Unknown aetiology
>10% increase in the baseline compared to the initial recording and/or absence of cycling, ZigZag or sinusoidal pattern suggestive of ongoing chorioamnionitis. "Low voltage complexes" suggestive of "myometrial irritability" on the tocograph following a history of spontaneous, prelabour rupture of membranes > 6 hours	Maternal observations Reduce the superimposed hypoxic stress due to ongoing uterine contractions STOP oxytocin infusion Check for other signs of Chorioamnionitis: Liquor colour Offensive liquor Obstetric review Senior midwife review Management plan: Expediate delivery immediately	Risk of cerebral palsy increases with infection: J.K. Grether, K.B. Nelson Maternal infection and cerebral palsy in infants of normal birth weight. (Published erratum appears in JAMA 1998; 279: 118)
Fetal tachycardia and reduced variability in a case of diabetic ketoacidosis	Administer IV fluids Correct the Ketoacidosis	To correct ketoacidosis and ensure maternal stabilisation. No operative interventions are required because with normalisation of the maternal environment, reactive fetal tachycardia and reduced variability will revert back to normal.
A sudden & prolonged deceleration observed one hour after insertion of the prostaglandin pessary	Emergency bell Change position Immediate senior obstetric review Administer terbutaline (tocolytic) immediately to relieve uterine hypertonus. Follow the 3 -6 -8 -10 -12 minutes rule If there is no improvement, and other causes of acute	Suspected hyperstimulation / hypertonus The prostaglandins within the pessary/ tablet/ gel may have been absorbed rapidly as the rate of absorption depends on many factors including maternal pharmacodynamics and pharmacokinetics as well as the pH of the vagina.

Clinical Context	Immediate Actions	Rationale
	hypoxia is excluded and uterine hypertonus persists on the tocograph / on clinical examination, consider a second dose of terbutaline, in the absence of maternal tachycardia / contraindications.	
Subacute pattern with episodes of ZigZag pattern observed within 20 minutes of active maternal pushing with the fetal head at 0 station in an occipito-posterior position	Immediate obstetric review Stop pushing Stop oxytocin If no improvement Discuss and offer birth by the safest and fastest based on the clinical context and the station of the fetal head.	Sign of fetal autonomic irritability indicating metabolic acidosis. Avoid continued hypoxic stress and take immediate measures to reduce the ongoing hypoxic stress, unless an immediate spontaneous or operative vaginal birth as imminent (fetal head visible at the perineum).

Table 6:

Intrapartum Fetal Assessment Tool			
Mat Pulse:	Temp:	Initial Baseline FHR	Induced / Augmented labour? Y/N
Risk Factors.			
Current Baseline FHR **Paper speed**	Variability	Accelerations	Decelerations Quicklies/Tardies/Both
Rise in Baseline (≥10%)	No	Yes	
Inter-contraction interval <90 seconds	No	Yes	
Abnormal Variability (<5 or >25)	No	Yes	
No Cycling / Loss of Cycling	No	Yes	
Features of Hypoxia	No	Yes	
TYPE of Hypoxia	Gradually Evolving/Sub-acute/ Combination/ Acute /None		
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			

Conclusion

A fetus who is *deemed FIT for labour* at the commencement of labour may not remain *FIT throughout labour*. This is because several antepartum and intrapartum risk factors may cause fetal compromise during labour, and it is essential to anticipate and timely recognise the specific changes on the CTG trace caused by this underlying pathology. The blind use of "toolkits" without changing the "Normal, suspicious, Pathological" (or White, Amber, Green) CTG guidelines without considering the types of fetal hypoxia, fetal response to hypoxic and inflammatory tress and the impact of the identified risk factor on the fetal heart rate is likely to worsen maternal and perinatal outcomes.

It is hoped that the use of FIT-CAT will aid frontline clinicians to predict the expected changes based on the observed clinical context.

Conflict of Interest:

EC, ME and MB are members of the International Expert Consensus Guidelines on Physiological Interpretation of CTG. However, they have no financial interests which conflict with this publication.

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Contribution of Authorship

EC conceived the concept of "FIT-CAT". SG, YK, ME and MB contributed to the drafting of the manuscript, reviewing and editing the manuscript.

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