Recognition of Chorioamnionitis on the Cardiotocograph (CTG): The role of the “Chorio Duck Score”

Edwin Chandraharan1,* and Mareike Bolten2

ABSTRACT
Chorioamnionitis refers to the inflammation of the chorion and amnion by the bacteria and their toxins as well as by the inflammatory cytokines. Therefore, it is a fetal disease with the bacteria, their toxins as well as the fetal immunological responses involving inflammatory cytokines (interleukins, interferons, and tumour necrosis factor-alpha) exerting their detrimental effects within the fetal compartment (i.e., amniotic fluid, fetal membranes, the placenta, and fetal tissues and organs). The vast majority of fetal inflammation occurs as a result of an ascending infection (i.e., entry of the bacteria from the maternal genital tract through the cervix). Therefore, maternal signs (tachycardia and pyrexia) may not be observed until the late stages of the disease. Cardiotocograph (CTG) trace was introduced into clinical practice in the 1960s as a tool to timely recognise ongoing fetal hypoxic stress so that immediate action could be taken to avoid hypoxic ischaemic encephalopathy (HIE) and/or intrapartum hypoxia-related perinatal deaths. Unfortunately, the CTG was introduced into clinical practice without any prior randomised controlled trials, which resulted in panicking and reacting to the observed morphology of fetal heart rate decelerations, which reflect an ongoing fetal compensatory response to reduce the myocardial workload. A sudden and reflex reduction of the heart rate in response to ongoing intermittent hypoxic stress (i.e., repetitive compression of the umbilical cord or interruptions in uteroplacental oxygenation due to uterine contractions) would ensure a reduction in myocardial oxygen demand and maintenance of aerobic metabolism to avoid the onset of anaerobic metabolism and production of lactic acid within the myocardium. Lack of understanding of fetal compensatory responses resulted in 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”. The same parameters were used in fetuses with chorioamnionitis with an alternative, inflammatory pathway of neurological injury. The international consensus guidelines of physiological interpretation of CTG produced by 44 CTG experts from 14 countries in 2018 ensured a paradigm shift and recommended classification of CTG traces based on the type of fetal hypoxia and fetal response to stress, by applying the knowledge of fetal physiology whilst interpreting CTG traces. During the last 5 years, research has highlighted CTG features in fetal neuro-inflammation, which include an absence of fetal heart rate cycling, the ZigZag Pattern, and sinusoidal patterns. The proposed “Chorio Duck Score” is a scoring system based on recently published scientific evidence on CTG features in subclinical and clinical chorioamnionitis to enable timely diagnosis of fetal systemic inflammatory response syndrome (FIRS). This will help avoid the continuation of super-imposed hypoxic stress (i.e., progressive increasing frequency, duration, and strength of uterine contractions) on the background fetal inflammation, to prevent fetal neurological injury.

Keywords: Fetal Blood sampling, “How is THIS Fetus?”, Physiological CTG interpretation, ZigZag pattern.

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1. INTRODUCTION

Human fetuses are exposed to two main pathways of neurological damage during labour. The hypoxic pathway of neurological injury is usually due to a progressively worsening frequency, duration, and strength of uterine contractions resulting in repetitive compression of the umbilical cord and/or interruptions in utero-placental oxygenation. However, it may rarely occur due to a sudden interruption of oxygen (e.g., umbilical cord prolapses, uterine rupture, or placental abruption). It is no longer acceptable to use a “normal, suspicious, pathological” classification system to identify fetal hypoxia without incorporating fetal physiological responses because repetitive Cochrane Systematic Reviews have concluded that compared to intermittent auscultation, the use of CTG did not improve the rates of cerebral palsy or perinatal deaths [1]. In contrast, such non-physiological classification of CTG traces has contributed to an increase in the rate of caesarean sections and operative vaginal births [1]. It has been reported that this unphysiological approach of grouping random fetal heart rate (FHR) features into different categories does not improve the rate of metabolic acidosis resulting from intrapartum hypoxic stress [3].

In comparison to the hypoxic pathway for fetal neurological injury, the inflammatory pathway that causes neurological injury is more common, but often it is misdiagnosed or underdiagnosed. This is because of the use of the illogical CTG guidelines focussing on the morphology of decelerations, which is not suitable even to detect intrapartum hypoxic stress, in fetuses with ongoing chorioamnionitis. Fetal inflammation does not cause intermittent umbilical cord compression and/or intermittent reduction in the uteroplacental oxygenation leading to the absence of decelerations unless there is a superimposed hypoxic stress. In contrast, the entry of bacteria and their toxins into the fetal compartment and the resultant fetal inflammatory response results in continuous and progressive “inflammatory stress”. Similar to infection in adults, the disease would progress through the stages of increased tissue metabolic rate due to fetal pyrexia, dissemination of infection to fetal tissues and organs, and fetal inflammatory response to the invading bacteria and their toxins which finally results in fetal systemic inflammatory response syndrome (FIRS). The latter is similar to adult systemic inflammatory response syndrome (SIRS) which occurs in the late stages of an infection resulting in multi-organ involvement. As fetuses have a relatively poorly developed blood-brain barrier (BBB), the entry of bacterial toxins and inflammatory cytokines into the brain results in fetal neuroinflammation. This increases the metabolic rate of neurones and their oxygen requirement, predisposing them to hypoxia-ischaemic encephalopathy (HIE) because inflammation sensitizes the neurones to hypoxic injury. Moreover, the inflammatory cytokines and the bacterial toxins can directly attack and damage the neurones resulting in neonatal encephalopathy (NNE) and its long term sequelae.

2. AETIOPATHOGENESIS OF CHORIOAMNIONITIS

Bacteria can enter the fetal compartment from the maternal birth passage through the cervix and the amniotic membranes (ascending infection), or from the maternal bloodstream through the placenta and the umbilical vein (vertical transmission). The onset of fetal infection (congenital pneumonia in ascending infections) will result in CTG changes secondary to chorioamnionitis (Fig. 1). There will be progressive changes in the fetal heart rate commencing with the fetal response to elevated fetal core body temperature and the resultant increase in the metabolic rate. The severity of the fetal infection and resultant inflammatory response would depend on the virulence of the organism, the host immune system, and the bacterial load. Therefore, any risk factor that increases the bacterial load such as “membrane sweep” (transfer of bacteria which normally colonise the upper or lower vagina, through the cervical canal, adjacent to the membranes) or insertion of prostaglandins for induction of labour (i.e., transfer of bacteria from the lower vagina, adjacent to the cervical canal) may increase the risk of a severe fetal infection. As the bacteria and their toxins enter the fetal systemic circulation, there is an increased risk of multi-organ involvement, and fetal systemic inflammatory response (FIRS). Entry into the fetal brain across the poorly developed fetal blood-brain barrier (BBB) would lead to the onset of fetal neuroinflammation. If the bacterial infection commences in the maternal compartment (e.g., pyelonephritis, gastroenteritis), then, maternal pyrexia and maternal tachycardia would be the first signs. There will be >10% increase in the baseline FHR as the fetus responds to increased metabolic rate as a result of maternal pyrexia. As the bacteria and their toxins cross the placenta and reach the fetus via the umbilical vein, there will be evidence of fetal inflammatory response syndrome (FIRS).

In contrast, in an ascending infection where the bacteria and their toxins are confined within the fetal compartment, maternal symptoms and signs will be absent. Therefore, it is both illogical and futile to be falsely reassured by the absence of maternal pyrexia and tachycardia. It is only when the bacterial toxins and inflammatory cytokines enter the maternal bloodstream via the umbilical arteries and through the placental sinuses and the uterine vein, one would observe maternal pyrexia and maternal tachycardia. This usually happens very late when the fetus suffers from severe FIRS, and often, maternal signs occur immediately after birth when the bacteria, their toxins, and the inflammatory cytokines rapidly enter the maternal bloodstream through the large, vascular placental site, immediately after the expulsion of the placenta. Therefore, clinicians should not depend on maternal symptoms and signs to diagnose chorioamnionitis due to an ascending bacterial infection.

3. CARDIOTOCOGRAPH CHANGES IN CHORIOAMNIONITIS

The onset of fetal systemic inflammatory response (FIRS) is heralded by loss of accelerations, and entry of the
bacteria across the blood-brain barrier (BBB) would result in the onset of neuroinflammation. Increased metabolic rate of neurons will result in the loss of the fetal deep sleep cycle and evidence of autonomic instability. If no action is taken, continuing and progressively increasing hypoxic stress (uterine contractions) on the background of increased tissue metabolic and oxygen demands will result in the onset of metabolic acidosis. Myocarditis due to the inflammatory cytokines, and the onset of myocardial metabolic acidosis would result in a progressive decrease in the cardiac contractility, culminating in a terminal bradycardia. Therefore, randomly grouping features of the fetal heart rate into different “categories” to classify CTG traces into “normal, suspicious, pathological” (or “Category I, II and III”) will miss these features of neuroinflammation increasing the likelihood of poor perinatal outcomes.

Galli et al. first published the CTG changes in subclinical and clinical chorioamnionitis [3], and features of increased fetal metabolic rate (>10% compared to previously observed baseline FHR without preceding decelerations), fetal systemic inflammatory response syndrome (loss of accelerations without preceding decelerations), and neuroinflammation (absence of cycling, “ZigZag” and sinusoidal patterns) were associated with both subclinical and clinical chorioamnionitis. However, the magnitude of the difference was greater in clinical chorioamnionitis as compared to subclinical chorioamnionitis [3]. Sukumaran et al. correlated the CTG features based on physiological interpretation with placental histopathological findings [4] and concluded that the features reported by Galli et al. correlated with placental histopathology. In this study, all fetuses (100%) showed an increase in the baseline FHR >10%, and 43% did not cross the threshold of 160 bpm used by several CTG guidelines focussed on “pattern recognition”. The latter finding should not be surprising to anyone who understands fetal physiology because a 1 °C rise in the temperature increases the baseline FHR by approximately 10%. Therefore, whilst all fetuses with chorioamnionitis and high metabolic rate due to inflammatory response would show an increase in the baseline FHR >10%, and 43% did not cross the threshold of 160 bpm used by several CTG guidelines focussed on “pattern recognition”. The latter finding should not be surprising to anyone who understands fetal physiology because a 1 °C rise in the temperature increases the baseline FHR by approximately 10%. Therefore, whilst all fetuses with chorioamnionitis and high metabolic rate due to inflammatory response would show a 10% increase in the baseline FHR, term fetuses with a lower baseline FHR (<130 bpm), will not be able to breach the artificial, illogical upper threshold of 160 bpm to be identified as abnormal. This explains why many fetuses with chorioamnionitis are currently missed by the existing CTG Guidelines which are...
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4. ROLE OF MECONIUM STAINING OF AMNIOTIC FLUID

Presence of meconium in the amniotic fluid has been shown to increase the risk of chorioamnionitis, by blunting the protective effects of macrophages within the amniotic fluid [6]. It has been shown that the growth of both E. coli and Group B Streptococcus (GBS) are accelerated when the amniotic fluid is contaminated with meconium [6]. Although there is a dose-dependent increase in the growth of bacterial colonies, the authors concluded that even light (or “thin”) meconium staining of amniotic fluid is associated with fetal infection [6]. Therefore, the illogical and potentially dangerous practice of classifying meconium into “non-significant” and “significant” and recommending women with “non-significant” meconium to labour at “low-risk” midwifery units with intermittent auscultation should no longer accepted in modern obstetric practice. Intermittent auscultation will not be able to detect the features of fetal neuroinflammation, and therefore, continuation of uterine contractions in such fetuses with missed neuroinflammation may lead to poor perinatal outcomes.

Presence of meconium in the amniotic fluid occurs in about 5%–20% of pregnancies and this incidence increases post-term to approximately 27% [7]. The passage of meconium may reflect a physiological process of progressive maturation of the fetal gut and maturation of intestinal ganglion cells and synapses and the consequent initiation of peristalsis. However, the presence of meconium within the amniotic fluid blunts the antibacterial action of the neutrophils and phagocytes, increasing the risk of chorioamnionitis.

Conversely, bacteria may enter the amniotic cavity with the clear amniotic cavity, and due to fetal breathing movements, reach the lungs and cause congenital pneumonia, and due to fetal swallowing movements to reach the fetal gut, resulting in gastroenteritis. The resultant fetal diarrhoea due to the inflammation of the bowel mucosa by the
ingested bacteria (gastroenteritis) will result in the passage of meconium. Therefore, meconium is very closely associated with chorioamnionitis, Galli et al. reported that 38% of cases of chorioamnionitis had evidence of meconium staining of amniotic fluid, as opposed to approximately only 2% in the control group [3]. Bolten & Chandraharan highlighted the local and systemic detrimental effects of meconium staining of amniotic fluid (MSAF) on the fetus [8], and chorioamnionitis and intrapartum hypoxic stress increase the likelihood of meconium aspiration syndrome. Therefore, in the presence of MSAF, if there are CTG features of chorioamnionitis, one should not allow uterine superimposed hypoxic stress to continue as it increases the likelihood of poor perinatal outcomes. Sukumaran et al have found that myometrial irritability was detected in 91% of cases of histologically confirmed chorioamnionitis [4]. It has been shown that in the presence of meconium, if the baseline FHR is >160 bpm, then, the likelihood of fetal infection is increased by 51-fold, compared to fetuses without meconium staining of amniotic fluid and tachycardia [9]. Therefore, fetuses with chorioamnionitis have an inherent risk of hypoxic injury both due to increased tissue metabolic demands and oxygen consumption as well as reduced oxygen supply due to myometrial irritability and reduced oxygen supply to the placental bed through the spiral arterioles. In addition, increased placental metabolism due to inflammation increases placental oxygen consumption and may reduce the transfer of oxygen to the fetus. Continuation of labour (i.e., progressively worsening uterine contractions) in the presence of MSAF and features of neuroinflammation and myometrial irritability on the CTG trace may lead to myocardial decompensation and terminal bradycardia (Fig. 2).

It is important to note that fetuses >40 weeks of gestation who may have a lower baseline FHR (110–130 bpm) due to the strong vagal influence may not increase the baseline FHR >160 bpm (Fig. 3). Unfortunately, most CTG guidelines do not mention the “ZigZag” pattern and absence of cycling, and stipulate normal baseline as 110–160 bpm.

Therefore, in the absence of repetitive decelerations, most fetuses with ongoing chorioamnionitis with features of neuroinflammation (absence of FHR cycling, the ZigZag and sinusoidal patterns) and a >10% increase in the baseline FHR (Fig. 3) will be easily missed, resulting in avoidable poor perinatal outcomes.

Classification of such CTG traces (Fig. 3) as either “Normal” or Suspicious” by using population-based CTG guidelines with arbitrary parameters and an illogical emphasis on deceleration on fetuses with chorioamnionitis may lead to disastrous consequences.

5. Recognition of the CTG Features of Chorioamnionitis: The Concept of the “Chorio Duck”

There is a well-known saying in the English language to indicate something that is very obvious based on the observed features: “If it looks like a duck, walks like a duck, and quacks like a duck, then it is a duck”. Chandraharan first coined the term “Chorio Duck” in Physiological CTG Masterclasses in 2012 to highlight the expected CTG features in chorioamnionitis (Table I). This has been subsequently published to enable midwives and doctors to timely recognise the features of chorioamnionitis to improve maternal and perinatal outcomes [10]. Most of the CTG guidelines with “Normal, Suspicious, Pathological” (Or Category I, II, III) classification systems will miss fetuses with chorioamnionitis because not only do they provide a wide range of the baseline FHR (110–160 bpm) as “normal” and thereby miss fetuses increasing their baseline from 120–145 bpm, but they also do not include features of neuroinflammation (absence of cycling [11], and the ZigZag Pattern) in the classification table. Moreover, greater emphasises is placed on the morphology and the duration of decelerations which occur due to intermittent, hypoxic stress (repetitive compression of the umbilical cord, or uterine contractions-mediated reduction in utero-placental oxygenation). However, chorioamnionitis causes continuous, inflammatory stress with increased metabolic rate and multi-organ involvement. Therefore, clinicians should not blindly use CTG Guidelines which are developed to recognise intrapartum hypoxic stress on fetuses which experience an inflammatory response to avoid neonatal encephalopathy and perinatal deaths. Recently Pereira et al. concluded that the absence of FHR cycling is associated with a significantly increased risk of developing maternal pyrexia and perinatal outcomes [11]. Some earlier scientific studies have failed to show any correlation between CTG changes in chorioamnionitis and perinatal outcomes. This is because they used the “normal, suspicious, pathological” classification system and used maternal pyrexia and maternal tachycardia for the diagnosis of chorioamnionitis. Moreover, they used Apgar Scores and umbilical arterial pH as markers of neonatal outcome. This approach has several flaws. Firstly, it has been shown that 43% of fetuses did not increase the baseline FHR

<table>
<thead>
<tr>
<th>Comparison to the Duck</th>
<th>Underlying pathophysiology</th>
<th>Observed features</th>
</tr>
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<tbody>
<tr>
<td>Looks like a Duck</td>
<td>Risk factors which increase the risk of entry of bacteria into the fetal compartment or their multiplication</td>
<td>SROM/History of Membrane Sweep/presence of meconium staining of amniotic fluid</td>
</tr>
<tr>
<td>Walks like a Duck</td>
<td>Increased fetal metabolic rate</td>
<td>&gt;10% increase in the baseline compared to previous observed baseline FHR or what would be expected for the given gestational age</td>
</tr>
<tr>
<td>Quacks like a Duck</td>
<td>Onset of Fetal Systemic Inflammatory Response Syndrome (FIRS)</td>
<td>Loss of accelerations</td>
</tr>
<tr>
<td>Displays a large Banner saying “I am a Duck”</td>
<td>Onset of fetal neuroinflammation Spill over of bacterial toxins and inflammatory cytokines to the maternal environment</td>
<td>ZigZag Pattern Sinusoidal patterns Maternal Tachycardia Maternal Pyrexia</td>
</tr>
</tbody>
</table>

**TABLE I: The CTG Features of the “Chorio Duck”**

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above 160 bpm [4], and maternal pyrexia and tachycardia would not be universally present, unless in very late stages of the disease [3], [4]. Moreover, unless there is superimposed hypoxic stress, both Apgar Score and umbilical arterial pH will be normal in these fetuses. Most studies did not consider delayed admission to the neonatal unit with evidence of congenital pneumonia, neuroinflammation, and multi-organ involvement, which are the most common neonatal manifestations of fetal inflammation.

6. DETRIMENTAL EFFECTS OF SUPERIMPOSED HYPOXIC STRESS ON THE BACKGROUND OF FETAL INFLAMMATION

Several studies have concluded that chorioamnionitis significantly increases the risk of neonatal encephalopathy (NNE) and cerebral palsy [13], [14]. Experimental animal studies have highlighted that both lipopolysaccharides (LPS) and inflammatory cytokines sensitize the fetal brain to hypoxic insults [15]-[19], and a review of scientific evidence concluded that antenatal exposure to infection lowered the threshold for intrapartum hypoxic-ischaemic brain injury [20]. An experimental study in newborn rats that administration of LPS alone did not result in brain injury, however, superimposition of hypoxia ischaemia resulted in a dose-dependent in brain injury [21]. Moreover, if the LPS is administered intra-cisternal in newborn rats, it has been shown that there is an increased expression of tumour necrosis factor-alpha and an increase in the sensitization of the brain to hypoxic-ischaemic injury [22]. Observational human studies have suggested inflammatory activation and damage to the white matter of the brain in chorioamnionitis [23], and approximately a 78-fold increase in neonatal encephalopathy in cases of superimposed hypoxia on the background of chorioamnionitis [24]. Therefore, there is evidence both from experimental animal models and observational human studies that ongoing chorioamnionitis sensitizes the fetal brain to hypoxic injury and reduces the threshold for neurological injury. Therefore, translation of this scientific evidence in daily clinical practice would suggest that the superimposition of the hypoxic stress (i.e., uterine contractions that intermittently and progressively reduce fetal oxygenation) is very likely to worsen the perinatal outcomes and increase the likelihood of neurological injury.

7. THE USE OF THE “CHORIO DUCK SCORE” TO HELP OPTIMISE PERINATAL OUTCOMES

The dilemma that clinicians often face is when to intervene when there are features suggestive of chorioamnionitis, especially in the absence of maternal tachycardia and pyrexia. Unfortunately, focusing on the morphological appearance of decelerations to classify CTG traces as “pathological”, and the lack of awareness regarding features of neuroinflammation on the CTG trace may lead to the continuation of labour. A combination of ongoing inflammation and an additional, super-imposed hypoxic stress may lead to rapid deterioration of fetal condition, culminating in a poor perinatal outcome.

The “Chorio Duck Score” has been developed to aid clinicians in making the decision to intervene at the right time (Table II). A retrospective application of this score in settled medico-legal cases and risk management cases (Table III) suggests that if the Chorio Duck Score (CDS)
is >5, the likelihood of poor perinatal outcomes is greater. All cases were admitted to the neonatal unit after birth, and 50% of neonates had either severe hypoxic ischaemic injuries or died in the neonatal period (Table III). All cases (100%) had >10% increase in the baseline FHR (i.e., evidence of increased metabolic rate) and features of neuroinflammation (i.e., absence of cycling, and/or the ZigZag Pattern and/or sinusoidal patterns). 90% of women had evidence of myometrial irritability on the tocograph and meconium staining of amniotic fluid (MSAF) was noted in 50% of cases. Maternal tachycardia and pyrexia were observed only in 30% of cases.

8. CTG Features of Chorioamnionitis: Recommendations for Best Practice

Frontline clinicians must consider chorioamnionitis as a fetal disease with signs in the fetus, and they should not await the onset of maternal tachycardia and pyrexia to make management decisions. This is because changes in the maternal parameters may not occur until the late stages of the disease, and even in cases of poor perinatal outcomes, only 30% of women had tachycardia and pyrexia (Table III). In adult and paediatric medicine, once sepsis is diagnosed, high-flow oxygen is recommended to avoid the onset of anaerobic metabolism and production of lactate due to increased tissue metabolism. The same principles should be applied in cases of chorioamnionitis and therefore, one should not allow uterine contractions to continue (unless a spontaneous vaginal birth is imminent). Pereira & Chandraharan proposed a “Fetal Monitoring Checklist” to be used at the time of admission in labour to exclude pre-existing causes of fetal compromise [25] to determine whether the “Fetus is FIT to undertake the progressively hypoxic journey of labour”. If abnormalities are noted on the “Fetal Monitoring Checklist” with features suggestive of chorioamnionitis at the onset of labour or in the antenatal period, including evidence of myometrial irritability on the tocograph, then, delivery should be expedited by an emergency caesarean section.

This is because uterine contractions reduce uteroplacental oxygenation by compressing the spiral arterioles that supply the placental bed, and they may also cause compression of the umbilical cord. This intermittent reduction in fetal oxygenation which progressively worsens as the uterine contractions become more frequent, stronger, and last longer as labour progresses, is usually well tolerated by the vast majority of fetuses. However, fetuses with chorioamnionitis have an increased metabolic rate and oxygen demand, and therefore, any reduction in oxygenation for a prolonged period is likely to increase the risk of anaerobic metabolism and risk of neurological damage due to increased lactate levels. Current evidence suggests that fetal inflammation accounts for approximately 15% of all cases of neonatal encephalopathy [5], [26]. Moreover, animal experimental studies and observational human studies have suggested that chorioamnionitis sensitizes the fetal brain to hypoxic-ischaemic injury. Therefore, if a spontaneous vaginal birth is not imminent, then, in the presence of CTG features suggestive of neuroinflammation whether in early labour or in an established labour, an emergency caesarean section should be considered to optimise perinatal outcomes.

Population-based studies have suggested that maternal pyrexia, operative vaginal births and emergency caesarean sections increased the likelihood of cerebral palsy, whereas an elective caesarean section reduced this risk, most likely due to the detrimental effect of intrapartum hypoxic stress on fetal inflammation [26], [27].

9. Likely Impact of Earlier Intervention Based on the “Chorio Duck Score” on Intrapartum Caesarean Sections

Some clinicians may be concerned that performing an emergency caesarean section when features of neuroinflammation of the CTG trace and/or based on the “Chorio duck Score” of >5 may increase the rate of unnecessary intrapartum interventions. However, current scientific evidence suggests that if labour is continued in these fetuses with chorioamnionitis, then, approximately 90% would require an emergency caesarean section due to worsening abnormalities on the CTG trace [4]. Our case series (Table III) also suggests that 70% of women had an emergency caesarean section any way during late labour, and
these fetuses had poor perinatal outcomes. Moreover, two out of the three fetuses who were born vaginally had meconium aspiration syndrome, which corroborates the findings of the animal experimental studies which reported that a dose and time-dependent worsening of neurological injury when chorioamnionitis and intrapartum hypoxic stress are combined [16–21]. Therefore, it is very unlikely that the overall caesarean section rate will be increased by the presence of chorioamnionitis and/or meconium-stained amniotic fluid on the growth of Escherichia coli and group B streptococcus. J Perinatol. 2002 Sep;22(6):477–71.


Recognition of Chorioamnionitis on the Cardiotocograph (CTG): The role of the ‘Chorio Duck Score’ Chandraharan and Bolten 10. Conclusion

Chorioamnionitis is a fetal disease, and therefore, it is essential to recognise features of fetal inflammatory response and neuroinflammation on the CTG trace. Both animal experimental studies and observational human studies, including review of medico-legal and risk management cases suggest that perinatal outcomes worsen when chorioamnionitis and intrapartum hypoxic stress are combined. It is hoped that the “Chorio Duck Score” will aid clinicians to promptly recognise ongoing chorioamnionitis even in the absence of maternal tachycardia and pyrexia, so that birth can be expedited to avoid the detrimental effects of super-imposed hypoxic stress (uterine contractions) on ongoing fetal inflammation.

Conflict of Interest

The author Edwin Chandraharan has conducted several Physiological Masterclasses 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 was the Course lead for the Neoenta Academy and the Baby Lifeline CTG Masterclasses. Organizers and hospitals of some of these Masterclasses have received sponsorships from Philips, Neoventa, Eourkeing, Hutleigh, K2, Cardiac Services, and other industries 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 in 2018. Mareike Bolten has been a member of the faculty for Physiological CTG Masterclasses and does not have any conflict of interest.

References


