OPEN ACCESS

Ology Press

Mini Review

International Journal of Gynecology and Reproductive Sciences

Importance of fetal heart rate cycling during the interpretation of the cardiotocograph (CTG)

Marianna Preti,[1] Edwin Chandraharan[2]

[1]CT1 Psychiatry, Forth Valley Royal Hospital, Scotland

[2]Lead Consultant Labour Ward &Consultant Obstetrician & Gynaecologist, St George's University Hospitals NHS Foundation Trust, London

Correspondence: Edwin Chandraharan, Lead Consultant Labour Ward &Consultant Obstetrician & Gynaecologist, St George's University Hospitals NHS Foundation Trust, London, Email Edwin.c@sky.com

Received: July 17, 2018 | **Published:** July 23, 2018

Introduction

Baseline fetal heart rate variability is assessed by determining the 'bandwidth' of the baseline due to the opposing activity of the sympathetic and parasympathetic nervous systems. Therefore, it reflects the integrity of the fetal autonomic nervous system, and if the central nervous system is depressed during sleep, medications (e.g. opiates) or acidosis, this bandwidth would be reduced, resulting in a reduction in baseline fetal heart rate variability on the CTG Trace.

It is important to appreciate that similar to adult 'rapid eye movement' (REM) and 'Non-REM' sleep cycles, a healthy fetus at term would also demonstrates such alternating active and quiescent sleep cycles. This phenomenon of alternative quiet and active sleep epochs is termed 'cycling' and is a hallmark of fetal wellbeing in labour[1] and the presence of cycling indicates a non-depressed central nervous system (Figure 1). Current international consensus guidelines on CTG interpretation suggest that a reduction of baseline fetal heart rate variability for more than 50minutes should be considered as a pathological.[2] However, it is important to appreciate that the presence of a 'normal variability' (5-25bpm), in itself, does not indicate fetal wellbeing, if cycling of the fetal heart rate is absent. This is because fetal neurological damage is not solely determined by the presence of metabolic acidosis (i.e. loss of variability), and several non-hypoxic pathways may damage the fetal brain without the presence of metabolic acidosis. In such cases, the baseline variability may continue to remain normal, however, the fetus may demonstrate the loss of cycling phenomenon.

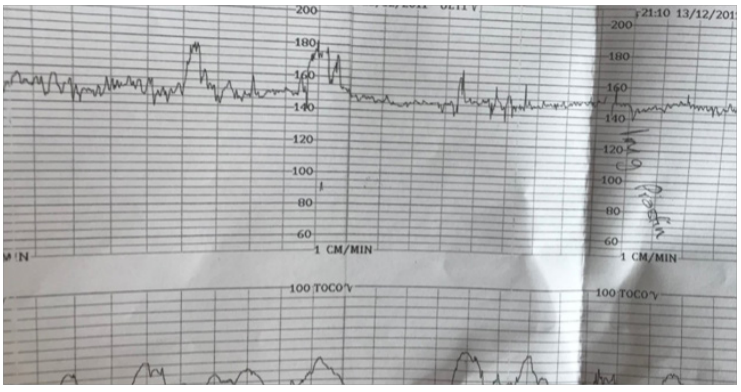

Figure 1 Note alternative periods of normal and reduced baseline variability demonstrating the phenomenon of cycling, reflecting fetal active and quiet sleep cycle.

What is the clinical application of cycling?

It is important to recognise that CTG was introduced into clinical practice to timely diagnose the onset of intrapartum fetal hypoxia so that corrective actions can be taken to reverse hypoxic stress (i.e. changing maternal position to relieve umbilical cord compression or reducing or stopping oxytocin) or to accomplish an urgent delivery, if corrective measures are not possible, to avoid hypoxic ischaemic encephalopathy (HIE) or perinatal death. However, there are several non-hypoxic pathways fetal neurological injury and perinatal deaths. These include inflammatory (e.g. maternal infection or chorioamnionitis with fetal systemic inflammatory response), metabolic (diabetic ketoacidosis), endocrine, biochemical (severe hyperglycemia or hypoglycaemia), immunological (congenital heart blocks), haematological (thrombocytopenia and fetal strokes) and fetal brain malformations. In such 'non-hypoxic' injuries, the abnormal features on the CTG trace (decelerations and loss of baseline variability) stipulated by the guidelines produced to recognise

intrapartum fetal hypoxic stress may not be present. On the contrary, depression of the central nervous system due to hypoxic and non-hypoxic insults may adversely affect the normal physiological fetal behavioural states, leading to the loss of the phenomenon of cycling.

Is there scientific evidence on cycling?

Longitudinal studies have suggested that the phenomenon of cycling develops after 36weeks of gestation due to progressive maturity of the fetal autonomic nervous system.[3] Animal studies have shown that a progressive reduction in placental oxygenation is associated with loss of acceleration and a reduction in baseline FHR variability.[4] Both animal[5,6] and human studies[7] have suggested that when exposed an acute intrapartum hypoxic stress, the baseline variability actually increases (i.e. a saltatory pattern) and it does not decrease. Therefore, it is vital to determine cycling of the FHR in addition to determining the baseline variability.[8] We analysed 202 CTG traces at term (between 36 completed weeks and 41 completed weeks) and found that 3.2% of "pathological" CTG traces had absence of cycling in the beginning of labour.[9] However, absence of fetal heart rate cycling at the end of labour was associated with a metabolic acidosis rate of 80%.[9]Therefore, cycling appears to be an important hallmark of fetal wellbeing during human labour, which denotes the optimal functioning of the fetal autonomic nervous system during human labour.

Clinical applications of 'cycling' phenomenon during labour

The aim of intrapartum fetal heart rate monitoring is to avoid hypoxic-ischaemic brain injury and perinatal deaths by timely recognising features suggestive of intrapartum hypoxic stress taking appropriate action prior to the decompensation and resultant fetal neurological injury and death. The use of fetal heart rate cycling may aid to recognise ongoing hypoxic and non-hypoxic causes of fetal injury (Table 1).

Table 1 Conditions which may be associated with loss of cycling

Hypoxic causes
Chronic hypoxia
Evolving hypoxia with syntocinon augmentation
Chronic fetal anaemia and acidosis
Non-hypoxic causes
Fetal infections (viral encephalitis, bacterial chorioamnionitis)
Feto-maternal haemorrhage
Intra-uterine fetal stroke
Diabetic keto-acidosis

Normal cycling

Presence of normal cycling indicates absence of depression of the central nervous system as the fetus has a normal behavioural state exhibiting active and quiet sleep epochs. Therefore, even in the presence of a 'pathological' CTG, if the fetus shows are assuring baseline fetal heart rate variability, the risk of acidosis is low.[10] In addition to normal variability, the presence of cycling would provide further reassurance regarding fetal wellbeing.

Absence of cycling in hypoxia

Catecholamine surge associated with hypoxia may obliviate the normal, phenomenon of active and quiet epochs. This is because the fetus goes into a deep sleep to reduce cerebral metabolism and to reduce oxygen requirements within the brain to avoid the onset of anaerobic metabolism and production of lactic acid. Although, the variability may be reduced, the fetus may continue to show cycling until the development of metabolic acidosis, when the central nervous system may be depressed. Therefore, urgent action should be taken (i.e. reducing or stopping oxytocin or administration of a tocolytic) if there is any evidence of reduced variability and/or absence of cycling following decelerations and a catecholamine-induced increase in the baseline FHR. In chronic hypoxia, it has been shown that there is a delayed maturation of the fetal autonomic nervous system[11] which may affect the phenomenon of cycling.

Absence of cycling in chorioamnionitis

Chorioamnionitis can cause fetal neurological damage by multitude pathways because a fetus has a relatively poorly developed blood brain barrier. These include the damage to developing neurons by inflammatory mediators due to fetal inflammatory response syndrome (FSIRS)[12] actual infection by the infective organisms (e.g. group B-Streptococcus or E.coli)[13] leading fetal meningitis or encephalitis, increased metabolic demand leading to a relative tissue hypoxia and acidosis and potentiating the damaging action of hypoxia on neurons.[14] In fetal brain inflammation, loss of the normal, physiological active and quiet sleep phases may result in the absence of cycling. One may not observe any other CTG abnormalities except a rise in the baseline FHR which is higher than expected for the given gestational age. In the absence of cycling, continuation of uterine contractions may potentiate the synergistic effect of hypoxia and inflammation on the fetal brain.

Other 'non-hypoxic' causes of absence of cycling

Intrauterine fetal strokes (i.e. cerebrovascular accidents) and hypovolemia (e.g. feto-maternal haemorrhage) and fetal metabolic disorders may also present with absence of cycling. Rarely, fetal viral encephalitis may also present with absence of cycling. It has been shown that fetuses of women with insulin dependent diabetes also have poor behavioural states.[15] We have proposed an Algorithm to incorporate the phenomenon of cycling whilst interpreting CTG traces during labour (Figure 2).

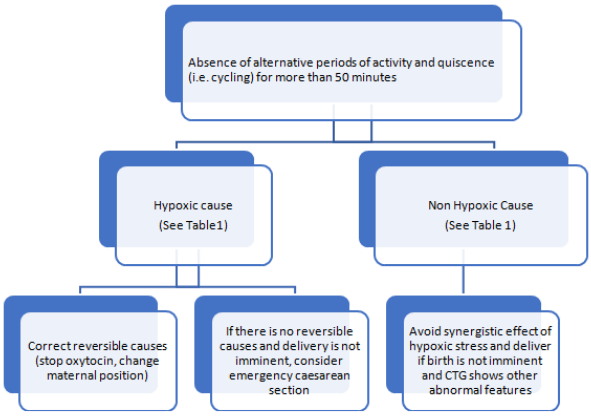

Figure 2 Suggested management algorithm for absence of cycling.

Conclusion

Understanding fetal behavioural states is crucial to interpreting CTG traces during labour to avoid hypoxic-ischaemic encephalopathy

(HIE) and perinatal deaths. Alternating periods of active and quiet sleep, called 'cycling' may help in differentiating a fetus who is well compensated from the fetus experiencing hypoxic and non-hypoxic insults to the central nervous system. The presence of a stable baseline which is appropriate for the gestational age, a reassuring variability, presence of accelerations and absence of repetitive, significant decelerations indicate absence of ongoing fetal hypoxia. However, presence of cycling (i.e. alternative periods of active and quiet epochs, approximately once in every 50minutes in a term fetus) is crucial to exclude depression of the brain secondary to hypoxic and non-hypoxic causes.

Acknowledgements

None.

Conflict of interest

Author declares that there is no conflict of interest.

References

1. Pinas A, Chandraharan E. Continuous cardiotocography during labour: Analysis, classification and management. *Best Pract Res Clin Obstet Gynaecol*. 2016;30:33–47.
2. Ayres-de-Campos D, Spong CY, Chandraharan E. FIGO consensus guidelines on intrapartum fetal monitoring: Cardiotocography. *Int J Gynaecol Obstet.* 2015;131(1):13–24.
3. Arduini D, Rizzo G, Giorlandino C, et al. The development of fetal behavioural states: a longitudinal study. *Prenat Diagn*. 1986;6(2):117–124.
4. Pulgar VM, Zhang J, Massmann GA, et al. Mild chronic hypoxia modifies the fetal sheep neural and cardiovascular responses to repeated umbilical cord occlusion. *Brain Res.* 2007;1176:18–26.
5. Westgate JA, Bennet L, Gunn AJ. Fetal heart rate variability changes during brief repeated umbilical cord occlusion in near term fetal sheep. *Br J Obstet Gynaecol*. 1999;106(7):664–671.
6. George S, Gunn AJ, Westgate JA, et al. Fetal heart rate variability and brain stem injury after asphyxia in preterm fetal sheep. *Am J Physiol Regul Integr Comp Physiol*. 2004;287(4):R925–933.
7. Kurahashi H, Okumura A, Kubota T, et al. Increased fetal heart rate variability in periventricular leukomalacia. *Brain Dev*. 2016;38(2):196–203.
8. Gracia Perez-Bonfils A, Chandraharan E. Physiology of fetal heart rate control. *Chapter In: Handbook of CTG Interpretation from Patterns to Physiology.* In: Chandraharan E, editor. Cambridge University Press; 2017.
9. M Preti, E Chandraharan, A Archer, et al. *Fetal Heart Rate 'Cycling' During Labour: Does It Really Matter*? Book of Abstracts. European Conference of Perinatal Medicine, Florence; 2014.
10. Chandraharan E, Arulkumaran S. Prevention of birth asphyxia: responding appropriately to cardiotocograph (CTG) traces. *Best Pract Res Clin Obstet Gynaecol*. 2007;21(4):609–624.
11. Murotsuki J, Bocking AD, Gagnon R. Fetal heart rate patterns in growth-restricted fetal sheep induced by chronic fetal placental embolization. *Am J Obstet Gynecol*. 1997;176(2):282–290.
12. Alexander BT. Intracerebral lipopolysaccharide induces neuro inflammatory change and augmented brain injury in growth-restricted neonatal rats. *Pediatr Res.* 2012;71(6):645–652.
13. Patrick LA, Gaudet LM, Farley AE, et al. Development of a guinea pig model of chorioamnionitis and fetal brain injury. *Am J Obstet Gynecol.* 2004;191(4):1205–1211.
14. McAdams RM, Juul SE. The role of cytokines and inflammatory cells in perinatal brain injury. *Neurol Res Int.* 2012:61494.
15. Vindla S, James D. Fetal behaviour as a test of fetal wellbeing. *Br J Obstet Gynaecol*. 1995;102(8):597–600.