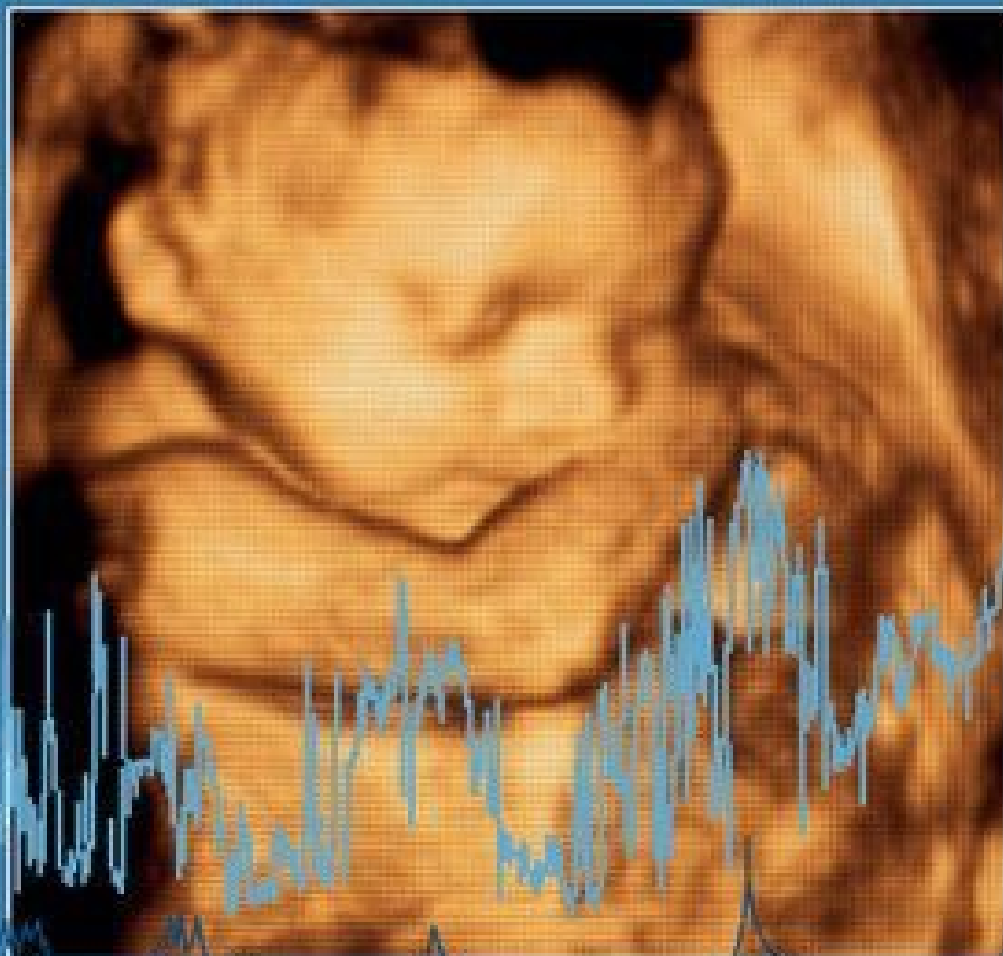


**HANDBOOK OF**

# **CTG Interpretation**

**From Patterns to Physiology**



**EDITED BY EDWIN CHANDRAHARAN**

**CAMBRIDGE**

**Medicine**

# Handbook of CTG Interpretation



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# Handbook of CTG Interpretation

From Patterns to Physiology

Edited by

**Edwin Chandraharan**

St George's University Hospitals NHS Foundation Trust, London, and St George's University of London, UK

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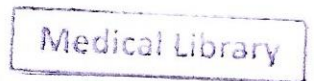
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# Contents

*List of Contributors* page ix

*Preface* xi

*Acknowledgements* xv

*Glossary* xvii

- 
- 1 **'An Eye Opener': Perils of CTG Misinterpretation: Lessons from Confidential Enquiries and Medicolegal Cases** 1  
Edwin Chandraharan
  - 2 **Fetal Oxygenation** 6  
Anna Gracia-Perez-Bonfils and Edwin Chandraharan
  - 3 **Physiology of Fetal Heart Rate Control and Types of Intrapartum Hypoxia** 13  
Anna Gracia-Perez-Bonfils and Edwin Chandraharan
  - 4 **Understanding the CTG: Technical Aspects** 26  
Harriet Stevenson and Edwin Chandraharan
  - 5 **Applying Fetal Physiology to Interpret CTG Traces: Predicting the NEXT Change** 32  
Edwin Chandraharan
  - 6 **Avoiding Errors: Maternal Heart Rate** 41  
Sophie Eleanor Kay and Edwin Chandraharan
  - 7 **Antenatal Cardiotocography** 45  
Francesco D'Antonio and Amar Bhide
  - 8 **Intermittent (Intelligent) Auscultation in the Low-Risk Setting** 55  
Virginia Lowe and Abigail Archer
  - 9 **Current Scientific Evidence on CTG** 59  
Ana Piñas Carrillo and Edwin Chandraharan
  - 10 **Role of Uterine Contractions and Intrapartum Reoxygenation Ratio** 62  
Sadia Muhammad and Edwin Chandraharan
  - 11 **Intrapartum Monitoring of a Preterm Fetus** 67  
Ana Piñas Carrillo and Edwin Chandraharan
  - 12 **Role of Chorioamnionitis and Infection** 71  
Jessica Moore and Edwin Chandraharan
  - 13 **Meconium: Why Is It Harmful?** 78  
Nirmala Chandrasekaran and Leonie Penna
  - 14 **Intrapartum Bleeding** 82  
Edwin Chandraharan
  - 15 **Labour with a Uterine Scar: The Role of CTG** 87  
Ana Piñas Carrillo and Edwin Chandraharan
  - 16 **Impact of Maternal Environment on Fetal Heart Rate** 91  
Ayona Wijemanne and Edwin Chandraharan



- 17 **Use of CTG with Induction and Augmentation of Labour** 96  
Ana Piñas Carrillo and Edwin Chandraharan
- 18 **Recognition of Chronic Hypoxia and the Preterminal Cardiotocograph** 101  
Austin Ugwumadu
- 19 **Unusual Fetal Heart Rate Patterns: Sinusoidal and Saltatory Patterns** 109  
Madhusree Ghosh and Edwin Chandraharan
- 20 **Intrauterine Resuscitation** 114  
Abigail Spring and Edwin Chandraharan
- 21 **Management of Prolonged Decelerations and Bradycardia** 118  
Rosemary Townsend and Edwin Chandraharan
- 22 **ST-Analyser (STAN): Principles and Physiology** 130  
Ana Piñas Carrillo and Edwin Chandraharan
- 23 **ST-Analyser: Case Examples and Pitfalls** 135  
Ana Piñas Carrillo and Edwin Chandraharan
- 24 **Role of a Computerized CTG** 142  
Sabrina Kuah and Geoff Matthews
- 25 **Peripheral Tests of Fetal Well-being** 147  
Charis Mills and Edwin Chandraharan
- 26 **Operative Interventions for Fetal Compromise** 151  
Mary Catherine Tolcher and Kyle D. Traynor
- 27 **Nonhypoxic Causes of CTG Changes** 155  
Dovilė Kalvinskaitė and Edwin Chandraharan
- 28 **Neonatal Implications of Intrapartum Fetal Hypoxia** 162  
Justin Richards
- 29 **Role of the Anaesthetist in the Management of Fetal Compromise during Labour** 167  
Anuji Amarasekara and Anthony Addei
- 30 **Medico-legal Issues with CTG** 171  
K. Muhunthan and Sabaratnam Arulkumaran
- 31 **Ensuring Competency in Intrapartum Fetal Monitoring: The Role of GIMS** 180  
Virginia Lowe and Edwin Chandraharan
- 32 **Physiology-Based CTG Training: Does It Really Matter?** 185  
Sara Ledger and Edwin Chandraharan
- 
- Appendix: Rational Use of FIGO Guidelines in Clinical Practice* 193  
*Answers to Exercises* 197  
*Index* 225

# Medico-legal Issues with CTG

K. Muhunthan and Sabaratnam Arulkumaran

## Background

- Cardiotocography refers to the recording of fetal heart rate (FHR) and contractions (tocography).
- Continuous electronic fetal monitoring (EFM) has become a standard practice in high-risk pregnancies and labour in the Western world.
- Despite severely abnormal CTG changes, failure of timely action and nonconsideration of the clinical situation leads to a compromised fetus.
- In-utero fetal death in labour, neonatal death and cerebral palsy associated with abnormal CTGs and asphyxia lead to medical litigation.

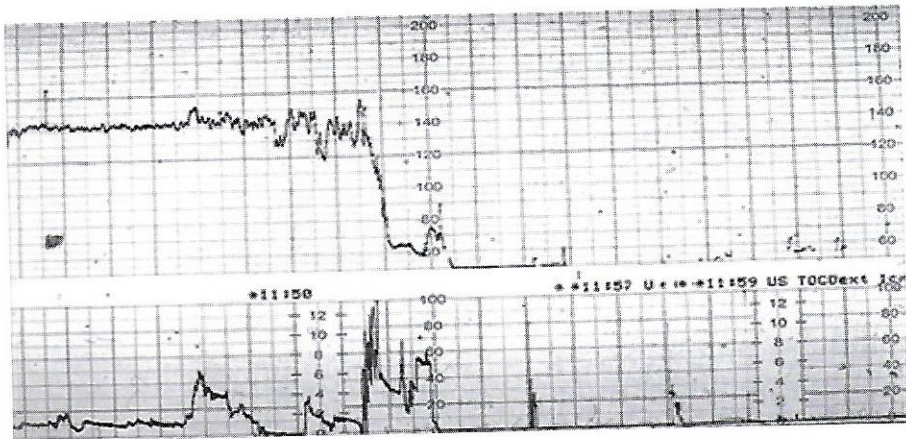
## Key Facts

Medical negligence involves establishing the causation and liability.

- Presence of abnormal CTG, low Apgar score, low cord arterial pH, assisted ventilation, admission to neonatal intensive care, moderate or severe neonatal encephalopathy and subsequent neurological damage point to asphyxia as a possible cause.
- However, several intrinsic fetal disorders (e.g. severe hypoglycaemia) cause neurological disability, and an abnormal CTG may have been coincidental.
- Causation is best determined by neuroradiologist and paediatric neurologist. The fetus born at term demonstrates certain areas of scarring within the brain on MRI. The thalamus, basal ganglia injury show scarring, reflecting acute profound hypoxia while prolonged partial hypoxia results in bilateral cortical atrophy.<sup>1</sup> Paediatric neurologist supports these findings by demonstrating that the baby has athetoid or dyskinetic cerebral palsy with acute profound hypoxia and spastic quadriplegia with prolonged partial hypoxia.<sup>2</sup>
- Liability is determined by demonstrating that appropriate and timely action was not taken in the presence of an abnormal CTG in that clinical situation.<sup>3</sup>
- Expert opinion is requested to judge whether care provided fell short of what was expected (Bolam principle).<sup>4</sup>

## Key Features on the CTG Trace

There are few key CTG patterns that are recognized to be associated with fetal compromise and are described below with example CTGs.<sup>5</sup>



**Figure 30.1** Acute hypoxia.

## Acute Hypoxia

- Presents with profound deceleration with a heart rate <80 bpm (Figure 30.1).
- The pH can drop on an average by 0.01 per minute.<sup>6,7</sup> The outcome of the fetus/newborn would depend on the physiological reserve of the fetus, actual heart rate (whether it is 40 or 60 bpm), duration of prolonged deceleration before delivery and cause for prolonged deceleration (e.g. abruption placentae, cord prolapse or scar rupture).
- An example of prolonged deceleration or bradycardia is given below. If prolonged, it can cause fetal death, or if born asphyxiated, it may lead to neurological injury associated with acute profound hypoxia.
- The thalamus and basal ganglia region gets affected and leads to athetoid or dyskinetic type of cerebral palsy.
- An example of such a trace is shown in Figure 30.1.

## Subacute Hypoxia

- Presents with prolonged decelerations (Figure 30.2).
- The FHR is below baseline rate for a longer time (e.g. >60 to 90 seconds) than at baseline rate (<30 seconds).<sup>8</sup>
- With such FHR, there is less than optimal circulation through the placenta over a given time, especially if the FHR drops to <80 bpm. With such a trace (Figure 30.2), some of the fetuses would get compromised with the progression of acidosis of approximately 0.01 every 2–3 minutes.

## Gradually Developing Hypoxia

The CTG trace usually starts with a normal baseline rate, normal baseline variability, accelerations and no decelerations.

Once decelerations start due to cord compression (variable decelerations) or reduced placental reserve (late decelerations), hypoxia can set in leading to catecholamine surge and rise in the baseline rate.