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Case Study

Fetal Monitoring by Cardiotocography in the Intensive Care Unit

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Abstract

A 25-year-old woman was admitted to the Intensive Care Unit for a 34.5-week gestation pregnancy and diabetic ketoacidosis associated with pyelonephritis. Laboratory tests: Hemoglobin 12.3 gr/dL Hematocrit 37.2%, Leukocytes 20,000 × 10^9/L, Platelets 210,000 × 10^9/L, Glucose 252 mg/dL, Urea 50 mg/dL, Creatinine 1.7 mg/dL, Uric Acid 7.2 mg/dL. The arterial blood gas shows a pH 7.1, base deficit -20, HCO3 4 mmol/L, lactate 1.5 mmol/L, PaO_2 110 mmHg, $PaCO_2$; 21 mmHg and Oxygen Saturation 96%, ketonemia 50 mmol/L. General Urine Examination with proteins +++, abundant bacteria, positive nitrites, abundant leukocytes, ketonuria +++, glucosuria >1000.

Obstetrics confirms the diagnosis of Intrauterine Growth Restriction and rules out labor at this time. Blood Pressure is 80/62 mmHg, heart rate 110 beats per minute, Respiratory Rate of 25 breaths per minute, Temperature 36.8º Celsius, uterine fundus of 26 cm, does not perceive fetal movements, Fetal Heart rate of 170 beats per minute.

Hartmann's solution 1,000 ml intravenously, Ceftriaxone 1,000 mg intravenously every 8 hours and insulin infusion are administered. After 6 hours of driving, he found the following biochemical parameters: Glucose 180 mg/dL, Sodium 135 mEq/L, Potassium 3.4 mEq/L, Chlorine 102 mEq/L, Ketonemia 15 mmol/L, urea 30 mg/dL, creatinine 1.3 mg/dL, arterial blood gas pH 7.25, 12 mmol/L HCO_3, -12 base deficit, 0.9 mmol/L lactate, 95% oxygen saturation.

A new obstetric evaluation is requested to assess fetal well-being, the cardiotocographic record shows a silent trace, which suggests an immediate interruption of pregnancy via the abdominal route, a cesarean section is performed and death is found.

Keywords: Ketonemia; Intensive Care Unit (ICU); RCTG

Introduction

Maternal and fetal pathophysiological processes that result in fetal death include; decreased uteroplacental flow due to maternal hypotension, maternal hypoxemia with decreased gas exchange in the trophoblastic membrane, metabolic alterations, maternal-fetal sepsis, fetal anemia, fetal heart failure and umbilical cord accidents, all these mechanisms can develop in a pregnant woman attended in the intensive care unit (ICU). It is estimated that a pregnant woman may require an ICU between 0.1% and 0.9% [7]. Fetal mortality in these women can be alarmingly high; A study that included 14 ICUs in England, fetal mortality was 75%, another study with 93 women and an average gestational age of 31 weeks, that is, at an average age of high viability, and yet the incidence of death was 15%. (Cartin-Ceba 2008) [7]. Fetal death is an event that can be prevented since there are premonitory data that can be identifiable. In critically ill women, the pathology and therapeutic measures applied to the mother affect the fetus. Generally, the intensivist does not perform continuous monitoring of fetal status as a complementary resource to multisystem monitoring.

The objective of this review is to show the benefit of the cardiotocographic registry (RCTG) as a tool for monitoring fetal well-being in the intensive care unit to avoid fetal death as an indicator of adequate intensive care in pregnant women in critical condition.

Fetal well-being follow-up by cardiotocography

Electronic fetal heart rate monitoring and was initially introduced for clinical use in the late 1960 as an alternative to the very labor-intensive auscultation of the fetal heart. The aim of such monitoring, which is generally continuous, is to enable clinicians to accurately identify hypoxic fetuses at risk for deterioration and who might benefit from expedited or immediate delivery either vaginally or by Cesarean section is an accepted component of labor management and fetal assessment, the true positive predictive value for metabolic acidosis [8].

Monitoring and RCTG are the first-line strategies for assessing fetal status. Monitoring is aimed at the early detection of fetal hypoxia, before irreversible effects occur, such as hypoxic-ischemic encephalopathy (O'neill 2012; Cochrane 2015) [9,10].

The American College of Gynecology and Obstetrics (ACOG), the International Federation of Gynecology and Obstetrics (FIGO 2015) and the National Institute for Health and Care Excellence (NICE 2014) recommend the RCTG as an assessment tool in various maternal and fetal pathologies such as; arterial hypertension, sepsis, diabetes, chronic kidney disease, growth restriction, preeclampsia, history of stillbirth, among others (ACOG 2014; FIGO 2015; NICE 2014) [10-12].

Cardiotocography

The CTG is a real-time, paper recording of the baby's heart rate and mother's contractions. Whilst a continuous CTG gives a written record, this is done by using a Doppler ultrasound transducer to monitor the baby's heart rate and a pressure transducer to monitor uterine contractions, both of which are linked to a recording machine. The patient should be placed in a semi fowler or left lateral decubitus position. The FHR is monitored with the transducer that is placed in the area that corresponds to the location of the fetal heartbeat, while the tocodynamometer is placed in the uterine fundus [17].

The goals of fetal testing are to identify fetuses at risk for stillbirth, assess for uterine asphyxia, and intervene to prevent adverse outcomes while minimizing maternal and neonatal morbidity.

In the CTG, the reactivity of the fetal heart rate is considered as an indicator of adequate fetal autonomic function; since a healthy fetus, without acidosis or neurological depression, temporarily accelerates its heart rate in response to fetal movements [17]. For a correct interpretation, the physician must follow a checklist of steps. First of all Corroborate the gestational age, investigate about fetal or maternal previous pathologies (CIUR, Preeclampsia, history of medications), then evaluate the current clinical situation, the indication of the CTG, and finally determine the acceptable limits of normality for this CTG before starting the assessment.

Evaluation of basic CTG features

- Baseline
- Variability
- Accelerations

- Decelerations
- Sinusoidal pattern

Baseline

This is the mean level of the most horizontal and less oscillatory FHR segments. It is estimated in time periods of 10 minutes and expressed in beats per minute (bpm). The baseline value may vary between subsequent 10 minute sections. A normal baseline is considered between 110 and 160 bpm. Tachycardia is valued above 160 bpm lasting more than 10 minutes (the most frequent cause may be of extra-uterine origin such as the administration of beta-agonist drugs, parasympathetic blockers or associated with intrauterine infection or whatever cause of a rise in maternal temperature. Bradycardia a baseline value below 110 bpm lasting more than 10 minutes; however, values between 100 and 110 bpm may occur in normal fetuses, especially in postdate pregnancies [18]. Continuous repetitive late decelerations with the onset of bradycardia after the peak of uterine contraction and persisting after contraction may be a sign of fetal compromise, particularly if associated with decreased variability. This abnormal fetal physiology in the critically ill patient is usually the result of hypoxia or reduced uterine oxygen availability. The presence of tachycardia is associated with acidemia with AUC 0.80 [19,20].

Figure 1

Tachycardia - Cardiotocographic record with fetal tachycardia, baseline at 230 beats per minute.

Figure 2

Bradycardia - Cardiotocographic record showing a decrease in the fetal heart rate and a baseline between 100 and 110 beats per minute in the presence of regular uterine activity.

Variability

Refers to the oscillations in the FHR signal, evaluated as the average bandwidth amplitude of the signal in one minute segments [18].

Variability can be categorized as absent, minimal, moderate, or marked. Moderate variability reliably predicts the absence of metabolic fetal acidaemia [7]. When amplitude range is undetectable is considered absent variability. Variability of less than 5 bpm is called a silent rhythm and is usually the result of fetal acidosis. The decreased variability for more than 30 minutes, the incidence of fetal acidosis is 30% [21,22].

Recurrent or sustained disruption of oxygen transfer to the fetus can lead to progressive deterioration of fetal oxygenation, resulting in loss of regulation of the fetal heart rate. When finding absence of variability, an evaluation should be carried out to rule out fetal metabolic acidemia.

A bandwidth amplitude of 5-25 bpm is considered a moderate (normal) variability. A bandwidth amplitude below 5 bpm for more than 50 minutes or for more than 3 minutes during decelerations in baseline segments is considered a reduced variability. It can occur due to central nervous system hypoxia/acidosis and resulting decreased sympathetic and parasympathetic activity, but it can also be due to previous cerebral injury, infection, administration of central nervous system depressants or parasympathetic blockers. And an increased variability (saltatory pattern) is valued exceeding 25 bpm lasting more than 30 minutes, it may be seen linked with recurrent decelerations, when hypoxia/acidosis evolves very rapidly and it is presumed to be caused by fetal autonomic instability/hyperactive autonomic system.

Figure 3

Minimum variability

Cardiotocographic record with variability less than 5 bpm, without decreases, without ascents.

Silent rhythm

A cardiotocographic record with a baseline at 160 bpm is observed, with absence of variability, without ascents or descents, in the presence of uterine contractions.

Figure 4

Accelerations

Accelerations are a sudden rise in the fetal heart rate, above the baseline. The increment must be at least 15 beats per minute in amplitude above baseline and lasting more than 15 seconds but less than 10 minutes in duration. Registration must be done for a minimum of 20 minutes [18]. The existence of transient accelerations of the FHR during the test is a phenomenon known as reactivity. This fact is universally considered as a sign of fetal health or well-being (Gallo 2014) [13]. And is a sign of a neurologically responsive fetus that does not have hypoxia/acidosis. Before 32 weeks' gestation, their amplitude and frequency may be lower (10 seconds and 10 bpm of amplitude). After 32-34 weeks, with the establishment of fetal behavioural states, accelerations rarely occur during periods of deep sleep, which can last up to 50 minutes [8]. The presence of FCF accelerations (spontaneous or stimulated) predicts the absence of fetal metabolic acidemia. Accelerations due to fetal movements are the most frequent alterations in CTG recordings, being present in almost 100% of normal fetuses (J Parer 2006) [23].

Decelerations

Decelerations are a decrease in the fetal heart rate on the CTG recording below the baseline. All decelerations can be measured in depth in beats per minute below the baseline. In studies carried out to evaluate the clinical significance of late decelerations to detect pH in the umbilical artery less than 7.1, a positive predictive value has been found that changes exponentially from 0% in the absence of decelerations to 1% in occasional late decelerations and > 50% in recurrent late decelerations [24]. The total area of deceleration demonstrates a superior predictive capacity for acidemia with an area under the curve of 0.83 [25].

The decelerations are classified in early, variable, late and prolonged decelerations. The early decelerations are shallow, short-lasting and are coincident with contractions. They are believed to be caused by fetal head compression, and do not indicate fetal hypoxia/acidosis. The variable decelerations are (V-shaped) that exhibit a rapid drop (onset to nadir in less than 30 seconds), there is good variability within the deceleration, rapid recovery to the baseline, varying size, shape and relationship to uterine contractions and constitute the majority of decelerations during labour, as occurs with umbilical cord compression. And the late decelerations are the U-shaped that have gradual onset and/or a gradual return to the baseline and/or reduced variability within the deceleration, These are indicative of a chemoreceptor-mediated response to fetal hypoxemia [12]. And the prolonged decelerations that last more than 3 minutes and indicate hypoxemia and require emergent intervention.

Figure 5

Decelerations

Cardiotocographic record with multiple descents in the presence of uterine activity.

Sinusoidal pattern

This pattern is characterized by an oscillating FHR baseline, with regular amplitude and frequency, minimal variability, and total absence of fetal movements or FHR accelerations, or both, resembling a sine wave. Its duration is at least ten minutes with an amplitude of 5-15 beats per minute. It indicates a very compromised and possibly terminal fetal state, it was observed for the first time in hydropic fetuses in feto-maternal incompatibility due to Rh factor. Although the pathophysiological mechanism of this pattern is unknown, it has been classically associated with fetal anemia, as is found in anti-D allo-immunisation, fetal-maternal hemorrhage, twin-to-twin transfusion syndrome and ruptured vasa praevia. It has also been described in cases of acute fetal hypoxia, infection, cardiac malformations, hydrocephalus and gastroschisis [26].

The existence of a sinusoidal pattern will be related to a perinatal mortality of between 50% and 55%, with a perinatal morbidity and mortality that reaches 92.5%. This pattern is also considered one of serious fetal deterioration and therefore will also condition the termination of pregnancy [26].

Figure 6

Undulatory or sinusoidal

Cardiotocographic record with a sinusoidal pattern. It is characterized by a fetal heart rate in the form of waves, with regular amplitude and frequency.

Reactive or non-reactive cardiotocographic recording

The most common definition of reactive recording is to identify two or more FHR accelerations in a 20-minute period, each lasting at least 15 seconds [27]. A non-reactive study lacks sufficient accelerations of FHR over a 40-minute period. The normal preterm fetus record (between 22.0 and 36.6 weeks of gestation) is often nonreactive. Between 24 and 28 weeks of gestation, up to 50% of the records may be non-reactive and 15% from weeks 28 and 32 [28,29]. Loss of fetal reactivity is commonly associated with fetal sleep cycles but can also result from causes of central nervous system depression including fetal acidemia [11]. It is for this reason that a nonreactive study warrants further evaluations to confirm fetal well-being.

The presence of an abnormal fetal heart rate pattern should alert the physician to fetal impairment. The ominous or terminal pattern refers to that trace in which there is no transitory acceleration, late-type decelerations appear in all contractions, and the variability of the baseline is reduced or silent undulatory. These changes in the cardiotocographic record warrant a reassessment of mean arterial pressure, data on hypoxia and maternal acidemia. An ominous pattern is considered indicative of severe fetal deterioration, which is seen in fetuses with subsequent intrauterine death, in the case of non-intervention, and with a high percentage of acidotic newborns after cesarean section intervention.

Figure 7

Reactive CTG

A cardiotocographic record is observed without alterations, adequate variability.

Fetal monitoring in intensive care unit

Diabetic ketoacidosis is a serious acute metabolic complication of diabetes. It is characterized by the triad of hyperglycemia, metabolic acidosis, and increased ketone bodies concentration. This is a rare complication of diabetic women during pregnancy, with an incidence rate of 1-2%, Nonetheless, represents a leading cause of fetal loss [9].

Septic shock is a life-threatening clinical syndrome that, despite its rare occurrence in obstetrics, remains a leading cause of maternal mortality.

Its pathophysiology is explained by a profound systemic response to a complex variety of host cellular and humoral mediators elaborated after exposure to microbial toxins.

Need diagnostic studies should never be withheld because of “pregnancy concerns”. With critically ill patients, the risk-to-benefit ratio supports the use of these diagnostic studies in

almost all circumstances. Standard therapy is directed principally at restoring tissue perfusion by intravascular volume expansion and in some instance's vasoactive pharmacological intervention. Simultaneously, identification of the source of infection and commencement of appropriate empiric antibiotic treatment are critical.

In an observational study conducted in China to analyze the perinatal prognosis and related factors of bacterial sepsis in pregnant women admitted to a tertiary care center in western China between 2011 and 2015, urinary tract infections were found to be the Most common source of infection in 47.7%, also showing that fetal deaths were 10.5% and are associated with maternal sepsis in general [10].

Pregnancy-specific considerations include the following: 1) initial signs or symptoms of septic shock may be masked by normal physiologic alterations of pregnancy; 2) a mixed polymicrobial group of organisms, consistent with lower genital tract flora, should be anticipated; and 3) initial therapy should be directed at maternal concerns since adverse fetal effects are most likely the result of maternal decompensation.

Fetal concerns

Maternal hyperglycemia results in fetal hyperglycemia and fetal osmotic diuresis. The fetus can also become acidotic from ketoacids that cross the placenta.

Acidemia decreases uterine blood flow, reduces tissue perfusion, and leads to decreased oxygenation of the fetoplacental unit. Furthermore, a leftward shift of the maternal oxyhemoglobin dissociation curve with decreased 2,3-diphosphoglycerate increases hemoglobin affinity for oxygen, decreasing fetal oxygen delivery [1-3].

Ketoacids dissociate into hydrogen ions and organic anions, both of which are transported across the placenta. Thus, with increasing maternal ketonemia, fetal metabolic acidosis may develop [4].

Because the fetus is not directly accessible, inferences regarding fetal status are often made from the external recording of the fetal heart rate. Often, decreased or absent variability, absent accelerations, and late decelerations are observed on external fetal heart rate tracings with decompensated maternal diabetic ketoacidosis.

Doppler ultrasound has also been used to look at blood flow in fetal vessels of mothers with diabetic ketoacidosis [3]. Transient fetal blood flow redistribution was demonstrated in the umbilical and middle cerebral arteries, as measured by pulsatility index. Reversal of the abnormal flow was demonstrated after treatment of the diabetic ketoacidosis maternal.

A single episode of diabetic ketoacidosis poses considerable risk to the fetus. Several retrospective studies have reported a perinatal mortality rate of 9-35% (1-5). Early recognition and prompt treatment of diabetic ketoacidosis might well avoid adverse fetal outcome [4].

Maternal hemodynamic responses in shock of any etiology do not include protective mechanisms for the fetus, but on the contrary, they protect the maternal vital organs, changing cardiac output from the uterine and placental circulation to the maternal brain and heart. Under these conditions, fetal oxygenation decreases as placental perfusion decreases. Maternal hypoxia causes fetal-placental vasoconstriction, reducing placental flow and fetal oxygen transfer causing a significant drop in fetal oxygen transport [14].

The Intensivist physician must recognize the risk to the fetus taking into account the underlying maternal pathophysiological process and the fetal state [15]. Since the fetal heart rate can vary according to maternal systemic changes.

In the context of a pregnant woman in critical condition, the monitoring of the fetal heart rate should be directed to the detection of abnormal patterns, mainly those that reflect fetal deterioration and risk of imminent death called "ominous data of the cardiotocography registry" [16].

Conclusions

Fetal heart rate monitoring represents a useful tool for the acute evaluation of the fetus and helps the intensivist to seek obstetric assistance in the context of a critically ill woman during viable

pregnancy. In general, critically ill women with respiratory or hemodynamic decompensation are accompanied by subsequent fetal deterioration. The identification of ominous traits can predict fetal death. Several factors, including gestational age and medication administered to the mother, can affect FHR features, so CTG analysis needs to be integrated with other clinical information for a comprehensive interpretation and adequate management. However, the general principles that guide clinical management are a good tool that can be used by the intensivist to reduce the high rates of stillbirth in intensive care units.

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