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Cardiotocography in preterm birth

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ABSTRACT

Premature birth is a public health problem worldwide. Due to the lack of cardiotocography (CTG) guidelines in preterm infants according to different gestational ages, we analyzed literature and synthesized the physiological and pathological characteristics of the CTG route of different gestational age preterm infants. The purpose of this analysis is to summarize the most relevant information regarding CTG monitoring in pregnancies with preterm infants and to provide CTG follow-up criteria for the daily practice of these conditions. In conclusion, the baseline of the fetal heart rate it is slightly increased in premature fetuses and decreases with increasing gestational age. During the second trimester of pregnancy, low-amplitude transitory decelerations occur more frequently and with a much lower frequency by the end of the third trimester. Cycling of variability is reduced due to incomplete development of the parasympathetic component of the autonomic nervous system in premature fetuses.

Keywords: cardiotocography, premature fetus, decelerations, premature birth

INTRODUCTION

Premature birth is a public health issue especially when premature infants are involved. Medical personal should be aware about the differences in both normal and abnormal characteristics of premature and mature fetuses. Also, we should take into consideration the patterns of monitoring and the decision of pregnancy termination depending on gestational age. Cardiotocographic monitoring is routinely performed as an important part of monitoring fetal heart rate and antepartum and intrapartum contractions [1]. Fetal heart rate (FHR) and fetal circulation determine the supply of the fetus. According to the International Federation of Gynecology and Obstetrics (FIGO) score, a duration of FHR recording of 30 minutes is required, with an extension of the duration if the recording seems suspicious. The maximum time for an Oxford CTG is 60 minutes.

MATERIALS AND METHODS

PubMed, NCBI and Medical Journals were searched for studies written in English that analyzed and synthesized the physiological and pathological characteristics of the CTG. Also, correlations were done in order to summarize the most relevant information regarding CTG monitoring in pregnancies with preterm infants and to provide CTG follow-up criteria for the daily practice of these conditions. Our goal was to define the normal heart rate in pregnancies with preterm fetus compared to full-term pregnancies and the correct choice of "alarm limits".

We included cohort studies, case-control studies, systematic reviews, and meta-analyses. The reference lists of the included studies were also screened for additional literature.

CTG MONITORING

Cardiotocographic monitoring is one of the most important methods in order to evaluate fetus's condition, both routinely, in the last trimester, and when considered pathological or within labor. Fetal heart rate is regulated by the nerve field through the medullary center controlled by baroreceptors, chemoreceptors, and local metabolic processes that influence regulation [2].

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SOGR recommendations for antepartum CTG monitoring are: maternal anemia (Hg < 10 g/dl), fetal arrhythmia (especially tachyarrhythmias) on ultrasound, late bleeding during pregnancy, blood group incompatibility, hypertension, type I and II diabetes, suspected or pathological fetal Doppler findings, drug abuse (ex. nicotine abuse), polyhydramnios (AFI > 25 cm), viral (parvovirus B19) or bacterial infections, decreased fetal movements, unstable maternal blood pressure (orthostatic hypotension), multiple pregnancy, oligohydramnios (single bag less than 2 cm), accident with abdominal trauma or severe maternal injuries, premature contractions (tocolysis) / threat of premature birth, intrauterine growth restriction 10th percentile [3].

Indications for intrapartum CTG monitoring if it is a risk-free pregnancy can range from once every 30 minutes to a maximum of two hours. Continuous monitoring should begin later in the first part of labor and in the expulsion stage. It can be increased if oxytocin is administered during labor or if complications such as fever, bleeding or green amniotic fluid occur. Continuous CTG monitoring should be done in the last stage of labor and in expulsion. Severe fetal bradycardia, prolonged decelerations over 3 minutes and other pathological patterns (sinusoidal model) requires immediate intervention in order to terminate the pregnancy [4,5].

The normal heart rate range is between 120 and 160 bpm, what is below 120 bpm is bradycardia and above 120 bpm is tachycardia. The frequency between 100 and 110 (moderate bradycardia) and 150-170 (moderate tachycardia) can be considered variations of normal or can occur in physiological brady or tachycardia [1]. Cardiotocographic monitoring in premature pregnancies under 36 weeks involves the interpretation of the FHR model of the fetus with the respective gestational age. A normal model is not yet clearly established before 34 weeks [1]. The nonstress test (NST) is used internationally and involves recording FHR at rest. FHR accelerations with fetal movements are assessed. An NST is considered reactive if two FHR accelerations associated with fetal movements occur over a 20-minute period. Decreased or complete lack of acceleration may indicate fetal hypoxia [6,7]. The stress test evaluates FHR during spontaneous or induced uterine contractions [8].

FHR CHARACTERISTICS IN PREMATURE FETUSES

To assess the well-being of a term fetus during labor, four features of the CTG pathway are evaluated, these include fetal heart rate, variability and the presence of accelerations and decelerations [9].

The characteristics of FHR antepartum and intrapartum follow-up differ in the preterm fetus compared to a full-term fetus (FHR of the fetus between 20 and 24 weeks is on average 155 bpm compared to 140 bpm in the full-term fetus). As the fetus exceeds the age of 30 weeks, there is a gradual decrease in FHR as a result of the progressive increase in parasympathetic influence. Accelerations occur for the first time in the second trimester of pregnancy, as a result of fetal somatic activity. The frequency of accelerations and their amplitude increases with advanced gestational age. Variability may be reduced due to incomplete development of the autonomic nervous system and interaction between the sympathetic and parasympathetic systems, but also as a result of fetal tachycardia present in premature fetuses. FHR "cycling" is considered a sign of fetal well-being, referring to periods of activity and break characterized by segments of increased variability, interspersed with an apparent reduction in variability. In preterm fetus, "cycling" may be lacking due to functional immaturity of the CNS, rather than fetal hypoxia [10,11].

INTERPRETATION OF INTRAPARTUM CTG

The onset of labor between 24 and 26 weeks is determined in most cases by a basic infectious process and is a high-risk group. At this gestational age the risk of neonatal morbidity and mortality is increased and survival depends very much on fetal weight and maturity. FHR is above normal (150-160 bpm), any rate above 160 bpm should be considered tachycardia. Persistent tachycardia may occur secondary to tocolytic administration. Variability and cycling may be reduced due to incomplete development of the parasympathetic component of the autonomic nervous system. Reduced variability of FHR has been associated with the administration of drugs such as pethidine, magnesium sulfate and steroids. Accelerations may be absent or significantly reduced and to a lesser extent. Decelerations are common at this age of gestation, representing the normal development of cardio regulatory mechanisms, they should not be considered hypoxia, and interventions should not be established solely on the basis of this parameter [9].

The characteristics of fetal heart rate models are dependent on gestational age, as they reflect the development and maturity of the cardiac centers and central nervous system as well as the cardiovascular system. Normal physiology is essential in the correct interpretation of fetal heart rate. In case of labor onset between 2 and 28 weeks of gestation, FHR recording will be similar to that between 24 and 26 weeks. The frequency of variable decelerations decreases after 27 weeks of gestation. Variability is close to normal with the development of the autonomic nervous system. The frequency of accel-

erations may increase, but the amplitude may remain low. Survival between 26 and 28 weeks is significantly higher than in the previous group, but half of them may develop neurological defects. An increased FHR or reduced variability should not be considered indications for operative interventions. At this gestational age the physiological reserves for combating hypoxia are not the same as those of a full-term fetus, especially as the onset of labor occurs secondary to an infectious process. A combination of abnormalities or an observed deterioration in the characteristics of the CTG should raise the suspicion of hypoxia and acidosis [12].

Labor started between 28 and 32 weeks of gestation develops a variability greater than 5 bpm, with cycling similarity between 30 and 32 weeks, and the FHR rate falls below the upper limits of normal. Initially variable decelerations should decrease and disappear after 30 weeks, due to the development of the fetal myocardium and increased glycogen storage levels, with fetal maturation. Persistence of decelerations may represent ongoing uteroplacental insufficiency with subsequent fetal hypertension are the result of intermittent hypoxia. The persistence of hypoxia leads in time to acidosis and can cause permanent fetal brain damage. install much faster, therefore the intervention should take place faster [4,11].

In the case of labor started between 32 and 34 weeks of gestation, the risk of mortality and neonatal morbidity is significantly reduced and continuous monitoring of FHR is recommended. During this gestational period, the physiological maturity of the cardiovascular system and the neural control of FHR are similar to those of a full-term fetus. Heart rate and fetal variability should be compared with those of a full-term fetus. Accelerations with an amplitude greater than 15 bpm are normal and correspond to fetal well-being. Variable and late decelerations should be classified according to National Institute for Health and Care Excellence (NICE) guidelines and appropriate measures taken. Monitoring aims to identify intrapartum hypoxia and whether intervention is necessary [9].

CONCLUSIONS

CTG monitoring of preterm fetus is a clinical dilemma for physicians caring for these patients during labor. Tachycardia and reduced variability are common in preterm fetus, a major source of confusion in the diagnosis of fetal hypoxia. The sleep-activity cycle has a short duration (10 minutes) in preterm fetus as opposed to term ones (30-40 minutes maximum especially during sleep). Fetal movements in preterm fetus, although they are in greater numbers, are less strong and with a shorter duration, which explains the lower reactivity. Between 25 and 30 weeks the decelerations are higher being concordant with the fetal movements, of an amplitude between 15 and 30 bpm and a duration of 15-30 seconds. As the fetus approaches the term the decelerations disappear and the accelerations begin. Variable deceleration occurs in 70% of preterm fetus between 28 and 30 weeks and in 50% of those over 30 weeks.

Abnormal patterns such as tachycardia and reduced variability are difficult to assess because in preterm fetuses they are not associated with fetal acidosis. “Absent” variability, late deceleration and combined deceleration are more commonly associated with acidosis even in preterm fetuses. Particular attention should be paid to tasks with intrauterine growth restriction, oligohydramnios, hypertension, diabetes. It is important to realize that the physiological resources available to combat hypoxia are smaller than to the term fetus.

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