Seminars in Fetal & Neonatal Medicine 20 (2015) 144–148

Contents lists available at ScienceDirect

Seminars in Fetal & Neonatal Medicine

journal homepage: www.elsevier.com/locate/siny

Review

Fetal heart rate monitoring

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Keywords:
Electronic fetal monitoring
Category I, II and III fetal tracings
Late decelerations
Variable decelerations
Sinusoidal

SUMMARY

Electronic fetal heart rate monitoring is a widely utilized means of assessment of fetal status during labor. Whereas little evidence exists regarding efficacy, this modality continues to be used extensively in every modern labor and delivery unit in developed countries. It is of importance that all providers of health care to the woman in labor and her newborn have a clear understanding of the basic pathophysiology of fetal heart rate monitoring and an appreciation for labor course and concerns as they arise in order to optimize outcomes and patient safety.

1. Introduction

Electronic fetal heart rate monitoring (EFM) is widely used in nearly every modern labor and delivery unit in the developed world, and was initially introduced for clinical use in the late 1960s as an alternative to the very labor-intensive auscultation of the fetal heart. EFM uses either the direct electrical signal of the fetal heart rate captured through a fetal electrode (direct) or more usually employs ultrasound technology and Doppler physics to interpret the changes in frequency of sound waves reflected from pulsations within fetal vessels (indirect). 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. Whereas EFM remains an accepted component of labor management and fetal assessment, the true positive predictive value for metabolic acidosis is low. Unfortunately, this often results in unnecessary interventions and a significant increase in the Cesarean section rate for concerns regarding fetal intolerance of labor. Such an observation may be the result of appropriate early intervention before worsening of metabolic condition occurs or inappropriate early intervention from a misreading of the information generated from the fetal monitor. Whereas the negative predictive value is exceptionally high (a normal fetal heart rate is associated with a normally oxygenated and non-acidotic fetus) and the rate of unexpected intrapartum stillbirth approaches zero, nonetheless, despite the wide usage of EFM, unfortunately there has been no significant decrease in the incidence of long-term neurologic morbidity (including cerebral palsy) or several other measures of neonatal wellbeing for the term newborn [1]. Despite these factors, EFM will continue to be utilized and needs to be understood with clinical concerns clearly communicated to provide the best care to all patients during pregnancy and in labor. Every member of the health care team needs to have an understanding of the suspected fetal status, which is often directly related to findings of changes in the fetal heart monitor so care can be best coordinated and effectively rendered in both non-emergency and emergency situations.

2. Physiologic basis of fetal monitoring

Fetal heart rate monitoring is essentially an observation of ongoing human fetal physiology, most often during labor or prior to labor, in the assessment of patients with certain high-risk conditions for which antepartum fetal surveillance is indicated. The question asked in all situations is: what is the adequacy of fetal oxygenation at this point? Since there are certain characteristic changes in the fetal heart rate pattern resulting from various hypoxic and non-hypoxic influences, one needs to appreciate the basic physiology of fetal respiratory exchange and resultant control of the fetal heart rate.

The placenta functions as an extracorporeal support system for the human fetus, serving as the fetal lung (respiratory exchange), kidney (excretion), gastrointestinal tract (nutrition) and skin surface (heat exchange). In addition, it plays a critical role in protecting the fetus against certain substances in the maternal circulation, which may be harmful particularly during early gestation. Further, the placenta actively produces several steroid and protein

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http://dx.doi.org/10.1016/j.siny.2015.02.002
1744-165X/

hormones critical to successful maintenance of the gestation and initiation of parturition. The human placenta is termed a hemochorial type, since maternal blood is extravascular when it comes into contact with the fetal chorionic villus. Exchange of oxygen, carbon dioxide, nutrients, water, heat, and waste products occurs within the placenta and must cross two layers of fetal trophoblast, the fetal connective tissue within the villus and the fetal capillary wall. Maternal blood flow to the uterus, and in turn to the placenta, results principally from the uterine arteries as well as from the internal iliac and ovarian arteries, and it markedly increases over the course of pregnancy. The arteries feeding the intervillous space where fetal–maternal exchange occurs are termed spiral arteries, and they must traverse the maternal myometrium. Flow through these spiral arteries is directly affected by anything that influences maternal cardiac output (e.g., hypotension, hemorrhage, sepsis) with immediate changes noted in the fetal heart rate in response to the decrease in uterine blood flow. Further and most importantly, as the uterus contracts, the pressure generated within the myometrium may exceed the intra-arterial pressure of the spiral arteries, resulting in intermittent cessation of maternal blood flow into the intervillous space. If such stasis is prolonged or associated with a significantly compromised placenta (small, infarcted, inflamed or senescent), either acute or chronic episodes of hypoxia and potentially acidosis in the fetus may result. Such changes are reflected in specific pattern changes in the fetal heart rate. Further, these conditions may be mitigated by position change of the mother to a lateral position or by directly addressing and correcting the causes of the compromised maternal cardiac output. The fetus also responds to such challenges by redistributing blood flow to certain vital organs – such as the brain, the heart, and the adrenal glands – while diminishing blood flow to less vital organs including the lungs, liver, kidneys, intestine, and periphery. However, if such changes persist, there may be a loss of cerebral autoregulation as well as an eventual decrease in fetal cardiac output. Cerebral blood flow is maintained initially by a fall in fetal cerebral vascular resistance by as much as 50%. However, if the physiologic condition continues to be compromised or worsens, then a reduction of cerebral blood flow may result, leading to potential fetal neuronal injury or death.

3. Essentials of fetal heart rate regulation

Just as in the adult, the fetal heart rate results from the influences of various intrinsic and extrinsic factors that modify the rate. The heart rate is actually the reciprocal of the interval between successive heart contractions. EFM uses the peak of the fetal electrocardiogram (ECG) R-wave to signify the time of the beat. The rate is then calculated by the interval between consecutive fetal ECG R-waves when using direct fetal electrodes, while it is calculated between consecutive pulse waves generated by myocardial contractions when using external monitoring employing ultrasound technology. It is the change in the fetal heart rate that is observed in various conditions that constitutes the basis of EFM, and one must appreciate the various factors that influence or modify the fetal heart rate to be able to correctly interpret the patterns generated by the fetal monitor.

The baseline heart rate of the fetus must be established to correctly interpret fetal tracings. Baseline heart rate decreases with advancing gestational age, being higher in early pregnancy than later. Further, the normal rate for the baseline is between 110 and 160 beats per minute (bpm). Such a rate results normally from the atrial pacemaker with modulation by parasympathetic and sympathetic factors. Rarely seen are cases of complete fetal heart block (with the rate usually about 60 bpm) resulting from the intrinsic ventricular or nodal rate.

In addition to baseline heart rate, key to interpreting EFM is the variability of the baseline heart rate. Variability is the true variance in time between consecutive heart beats, and it is normally present in a healthy fetus. Variability results from direct sympathetic and parasympathetic effects on the heart, which originate either in the brain stem and are conducted to the heart via the vagus nerve, or may arise from direct humoral stimulation of cardiac receptors responding to the release of epinephrine from the fetal adrenal glands. It is thought that higher brain centers also play a role in variability of the heart rate. Factors that influence the central nervous system such as drugs, hypoxia, metabolic acidosis, or brain injury affect the baseline variability of the heart rate. The presence of moderate variability of the heart rate is one of the most reliable markers of normal oxygenation and absence of acidosis, and its presence or absence is key to the correct interpretation of EFM.

A normally oxygenated fetus has certain characteristics of its heart rate. After 32 weeks of gestation, it is well established that normal fetuses have episodes of acceleration of their heart rate associated with fetal movements. This is termed “reactivity”, and, when present, has a very high association with a normal fetal state of oxygenation. Whereas there is not necessarily an acceleration of the heart rate every time a fetus moves, the converse is true. A normally oxygenated fetus generally demonstrates accelerations at least every 60–80 min. Certain drugs that affect the central nervous system can suppress accelerations, as can certain types of primary central nervous system abnormalities. A basic tenet that has been demonstrated experimentally is that in a fetus undergoing progressive hypoxia, there will be the appearance of decelerations before the absence of accelerations.

Fetal monitoring is assessed both in the absence and in the presence of uterine contractions. As discussed, a uterine contraction may cause intermittent decreases in maternal blood flow into the intervillous space. This may result in stasis of blood with inadequate exchange between the mother and fetus of oxygen, carbon dioxide, and other products. If significant hypoxia of the fetus occurs, there may be a decrease in fetal cerebral blood flow, resulting in a change of the sympathetic and parasympathetic control of the fetal heart. This may lead to a slowing of the fetal heart rate or a deceleration. Another frequently occurring mechanism explaining the occurrence of the deceleration is intermittent occlusion of blood flow through the umbilical cord due to cord compression or stretching. Such changes, particularly when associated with uterine activity, are considered contraction-related or “periodic” changes in the fetal heart rate. Again, key to interpretation of these changes in the fetal heart rate is the understanding of their physiologic bases.

4. Early decelerations

An early deceleration is a uniformly shaped deceleration of gradual onset and gradual return to baseline associated with a uterine contraction. The degree of slowing generally is proportional to the strength of the associated uterine contraction, and the timing of the deceleration accompanying the uterine contraction generally mirrors the contraction with the early deceleration nadir at the peak of the contraction. Early decelerations are thought to result from fetal head compression altering cerebral blood flow and leading to cardiac slowing through a vagal reflex. This is supported by the experimental finding that early decelerations can be abolished or altered by the administration of atropine. Early decelerations are generally seen in active labor and are a pattern not associated with fetal hypoxia, acidosis, or subsequent depressed Apgar scores.