Using Fetal Heart Rate Tracings to Assess Acidosis

Electronic fetal monitoring lies at the crux of our efforts to assess fetal well-being and detect intrapartum fetal compromise. Yet making the most of this tool—using it meaningfully to quantify or assess fetal well-being by the heart rate tracing—has been and remains a struggle.

To understand the challenges, one only has to look at the number of groups and individuals who have proposed—and continue to propose—various systems, definitions, and recommendations for assessing fetal heart rate tracings. Finding the best assessment strategies remains a key goal in obstetrics as we work toward realizing the full potential of electronic fetal monitoring.

The report issued last year by a panel convened by the National Institute of Child Health and Human Development, the American College of Obstetricians and Gynecologists, and the Society for Maternal-Fetal Medicine took us a step forward by initiating a consistent nomenclature of normal, abnormal, and indeterminate fetal well-being. This three-tiered system for fetal heart rate interpretation is limited, however, in that it assesses the fetal heart rate only during a discrete window of time, and provides no discrimination as to the degree of "normal." We need to think more broadly as we assess fetal heart rate tracings to understand where a fetus is on the spectrum of acidosis. The overall change in fetal metabolic acidosis during labor is what best reflects the risk of hypoxemia-induced organ injury. Although it's not a perfect criterion for predicting fetal well-being, the estimated degree of fetal metabolic acidosis is a much more meaningful predictor than is an estimate of the acute oxygenation status.

When seeing any normal fetal heart rate tracing at a snapshot in time, for instance, we could be dealing with a perfectly normal fetus (that is, one with a low level of acidosis) on the one hand, or we could have a fetus that is precariously close to entering severe acidosis. An abnormal fetal heart rate tracing, similarly, is not in-and-of-itself predictive of fetal metabolic acidosis. Knowing whether a fetus has only mild acidosis, or severe acidosis, has important implications. A fetus struck with bradycardia, for instance, will tolerate the complication much better if it has mild or no significant acidosis at the start than if it is on the precipice of shifting into severe acidosis. Knowledge of the degree of acidosis equips us to better predict and manage fetal compromise and avoid unnecessary operative deliveries.

Indeed, more research is needed to better understand the change in the level of fetal metabolic acidosis with both the progression of labor and with induced fetal heart rate changes. Yet even as we work to advance our knowledge, we have learned enough about fetal acidosis to be able to seek answers to several questions: Is what’s happening to the heart rate affected by hypoxia? Does the tracing reflect the degree of acidosis? Where are we on the spectrum of acidosis?

Changes in Base Deficit

The values termed “base excess” or “base deficit” are used to quantify the magnitude of metabolic acidosis during normal stages of labor. A large positive base deficit—or a large negative base excess—indicates that the body’s base buffers have been used up to buffer acids and that metabolic acidosis is present. A base deficit of 12 mmol/L—or alternatively a base excess of −12 mmol/L—is widely accepted as the threshold for risk of acute brain injury. When we’re looking at a tracing, our monitoring and management plans will differ significantly, therefore, for a fetus with a normal tracing and a base deficit of 2 mmol/L compared with a fetus who has a normal tracing and a base deficit of 8 mmol/L.

The average fetus enters labor slightly acidic with a base deficit of approximately 2 mmol/L. During the latent phase of labor, which typically represents minimal stress, the fetus incurs no real change in base deficit. During the active phase, however, the stress of the labor causes the base deficit to increase by approximately 1 mmol/L every 3-6 hours, and during the second stage, the base deficit increases by approximately 1 mmol/L every hour. This means that by the end of the first stage of labor, the fetus has a base deficit of 4 mmol/L, on average. At the end of the second stage, the average baby is born with a base deficit of approximately 5 mmol/L.

The development of mild acidosis through the stages of normal labor is analogous to an adult walking, jogging, and then sprinting. Most of us would progressively use more oxygen than we can provide as we pick up the pace, spurring a conversion from aerobic to anaerobic metabolism that results in the production of lactic acid and consequent soreness—even aching pain—in our legs. For the fetus, the latent phase of labor is the equivalent of our walking; the active phase represents jogging; and the second stage is equivalent to a sprint.

During labor, lactic acid accumulation can lead to metabolic acidosis and a blunting of the vagal regulation of the fetal heart rate and consequent loss of accelerations, loss of variability between contractions, and other changes with possible long-term sequelae.

In monitoring labor, we want to know where we are on the spectrum of acidosis. Have we gone through the active phase, for example? Where are we in the second stage? Understanding where the fetus is on this spectrum prepares us to manage any changes—any additional acidosis related to fetal heart rate decelerations—that are superimposed on the background stress of the labor process.

Acidosis and Heart Rate Patterns

Research has confirmed not only degrees of hypoxemia and fetal base deficit values during the normal course of labor; it also

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This fetal heart rate tracing shows repetitive, severe variable decelerations.