MASTER CLASS

Fetal Heart Rate Monitoring

BY E. ALBERT REECE, M.D., M.B.A.

Over the years, we have endeavored to assess fetal well-being by a number of electronic and nonelectronic means with varying degrees of success. Of all these methods, fetal heart rate monitoring has withstood the test of time.

Our continued use of fetal heart rate monitoring as a means of assessing the fetus's biochemical and biophysical status has contributed much to our understanding of fetal well-being, or lack thereof. More recently, efforts have been made to better correlate variations in fetal heart rate to fetal well-being.

It is well known that the fetus is the final arbiter of intrauterine stress and may respond with compensatory mechanisms that may thwart various types of stresses. In such a scenario, the fetus may not manifest a compromised state, despite potentially harmful stress conditions. On the other hand, another fetus facing similarly stressful intrauterine conditions may struggle, exhibiting fetal distress or worse.

In reality, what matters most is the response of the fetus and not the stressful condition per se. Every attempt to monitor fetal well-being has been focused, therefore, on the response of the fetus to various types of stress. Because we're unable to conduct biochemical testing on a real-time or continuous basis, fetal heart rate monitoring often has been used as a surrogate for the biochemical adaptations by the fetus to intrauterine stress conditions.

Fetal heart rate monitoring, thus, becomes a very important diagnostic tool because the decisions that physicians make and the interventions that they undertake often are based on their interpretation of the fetal heart rate tracings. Such decisions are critical to the overall outcome of the fetus.

It is in this light that we are dedicating a Master Class to the subject of fetal acidosis and fetal heart rate assessment and have invited Dr. Michael G. Ross to serve as our guest professor this month. Dr. Ross is the chair of obstetrics and gynecology at the Harbor-UCLA Medical Center in Torrance, Calif., and professor and vice chair of obstetrics and gynecology at the David Geffen School of Medicine at the University of California, Los Angeles.

Dr. Ross's exceptional article on this topic delineates the mechanisms of fetal metabolic acidosis and its effects on fetal well-being. He also offers valuable insights on how fetal heart rate tracings might be better utilized as a powerful tool for detecting and predicting where a fetus may lie along the acidosis spectrum during various stages of labor so that interventions may be implemented to prevent severe acidosis and associated injury to the fetus. ■

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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 benefits 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."

BY MICHAEL G. ROSS, M.D., M.P.H.

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 acidotic 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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Classification of Intrapartum Fetal Asphyxia

Asphyxia	Metabolic acidosis at delivery*	Encephalopathy			Cardiovascular, respiratory, and renal complications	
		Minor	Moderate	Severe	Minor	Moderate/severe
Mild	+	+/–			+/–	
Moderate	+		+			+/–
Severe	+			+		+

*Umbilical artery base deficit ≥12 mmol/L.
Source: J. Obstet. Gynaecol. Res. 2004;30:276-86

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has provided a window of knowledge into the changes in fetal acidosis in relation to particular fetal heart rate patterns.

Early decelerations are generally well tolerated by the fetus and probably do not result in any additional acidosis. These are believed to result from fetal head compression and a subsequent hormonal or vagal response.

Similarly, mild or moderate variable fetal heart rate decelerations, which are due to modest cord compression, are well tolerated if they occur at a reasonable frequency (such as every 3 minutes). The frequency of variable decelerations is critical as lactic acid generated during the variable deceleration may be cleared across the placenta during the periods between decelerations.

When variable heart rate decelerations are severe and of increasing frequency, however, the fetus can accumulate lactic acid—sometimes rapidly—depending on the frequency. A severe variable deceleration results from complete or near-complete umbilical cord occlusion and is typically defined as one that lasts for at least 60 seconds, during which the heart rate drops below 70 beats per minute

In a study we published this year with colleagues in Canada, we found that severe variable decelerations result in an increase in base deficit of 0.5 mmol/L per minute of cord occlusion. We also found, however, that metabolic acidosis is cleared at a rate of 0.1 mmol/L per minute of recovery, when fetal heart rate is normal and stress is reduced (Am. J. Obstet. Gynecol. 2009;200:200.e1-7).

Given these rates of acid accumulation and normalization, one can understand how acidosis may develop when repetitive, severe variable decelerations occur every 3 minutes, for instance. Past a certain frequency and severity, there simply isn't enough recovery time to allow the fetus to sufficiently correct the base deficit.

Although most of us will not actually be using these acid accumulation and recovery rates to calculate specific base deficits, an awareness of the principles can aid us in assessing fetal acidosis. The concept of recovery time is an important one. Again, knowing where your patient is on the spectrum of acidosis tells you how much "buffer time" you have if something goes wrong.

There is policy, sometimes attributed to midwives, that advocates letting the patient push only during every other contraction. Given what we've learned about the development of acidosis, when pushing is associated with severe variable decelerations, there may be an advantage to pushing every other contraction in order to permit sufficient recovery time and clearance of acid between the decelerations.

Prevalence of Intrapartum Fetal Asphyxia

	Rate per 1,000 live births	
	Preterm	Term
Overall prevalence	73	25
Mild	38	21
Moderate/severe	35	4

Source: J. Obstet. Gynaecol. Res. 2004;30:276-86

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One of the signals that acidosis is progressing to a moderate level (approximately 8 mmol/L) is the change in severe variable decelerations from typical (having shoulders, a sharp drop, and a sharp rise) to atypical (a loss of shoulders, a U-shaped variable deceleration, or a slow return to baseline).

Another sign of moderate acidosis is a loss of variability between contractions. These heart rate patterns need more research, but in my experience a prolonged loss of variability between contractions (unrelated to the fetal sleep state) typically does not occur until the base deficit approaches 8 mmol/L or greater. Given that the risk of brain injury begins with a base deficit of 12 mmol/L, an observation of this change provides a buffer zone during which the patient can be even more closely monitored.

It used to be thought that late decelerations were extremely worrisome, but we have learned that these patterns are usually less threatening to the fetus's accumulation of metabolic acidosis than are severe variable decelerations.

When there is good baseline variability between the decelerations, the late deceleration often reflects a vagal-mediated response and probably involves no change in the level of acidosis. When there is a loss of variability between contractions, however, the late deceleration may reflect a hypoxia-induced response. Still, the rate of acidosis accumulation is typically less than it is with severe variable decelerations.

In general, the amount of acid accumulation with late decelerations is dependent on the frequency and severity of the decelerations; the accumulation of acidosis may range from a base deficit increase of 1 mmol/L every 5 minutes to an increase of just 1 mmol/L every 15 minutes.

One of the weak links in our understanding of fetal acidosis today is our inability to recognize preexisting acidosis or preexisting injury. We have little experience in identifying fetal heart rate patterns associated with preexisting hypoxic injuries.

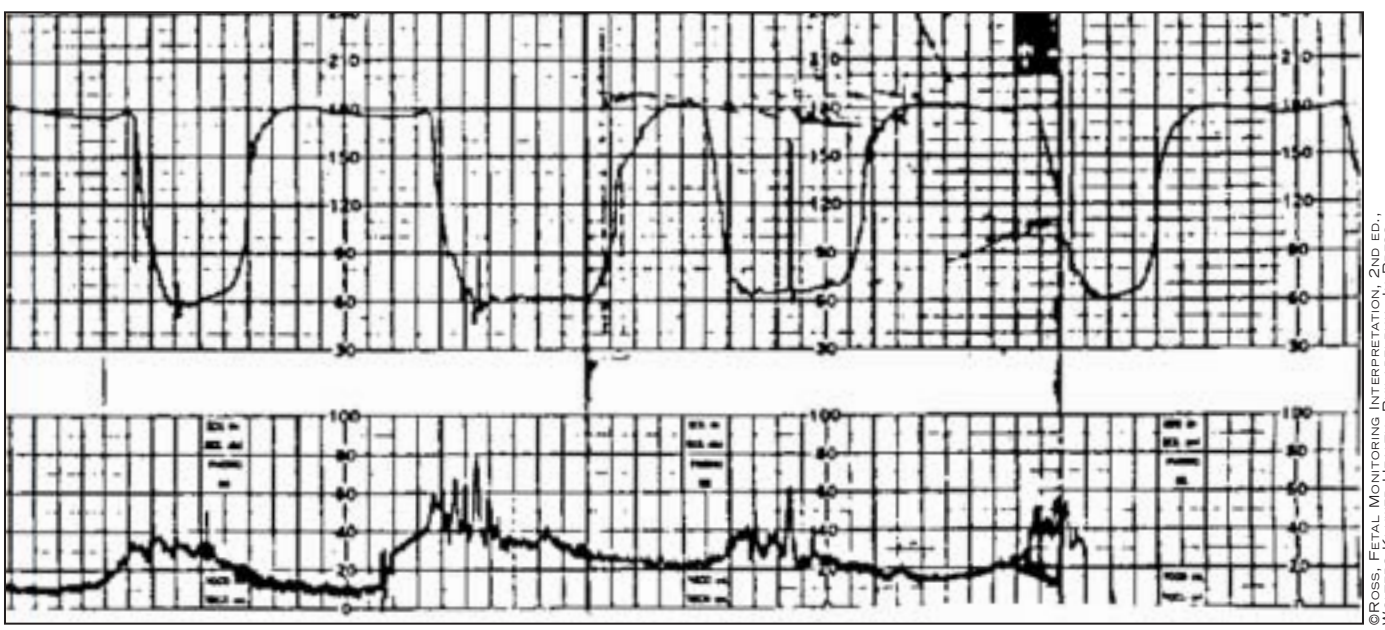
Adding to the challenge is the knowledge that a post-term fetus or one with intrauterine growth restriction may begin labor with a slightly greater level of acidosis. Furthermore, fetuses with true sepsis or severe anemia may accumulate acid at an increased rate compared with normal fetuses.

Where We Stand

Practically, attempting to avoid injury by recognizing mild, moderate, and potentially severe levels of fetal acidosis means that one must carefully examine fetal heart rate tracings, not only for the time we are present in the room, but at least back to the time of our previous assessment. As much as is possible, we should understand what the entirety of the monitoring has shown.

We should attempt to factor in the known changes in fetal acidosis associated with normal stages of labor together with estimated changes in acidosis related to superimposed fetal heart rate decelerations. With an understanding of the progress and stage of labor, the current fetal heart rate pattern, and the approximate level of fetal metabolic acidosis, we will be best prepared to manage the pregnancy for an optimal outcome. ■

DR. ROSS *said he has no disclosures relevant to the content of this article. Send comments to Dr. Ross at mikeross@ucla.edu.*



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

Pregnant Teens: Look For STIs

BY DAMIAN MCNAMARA

SAN ANTONIO — Routine repeat screening for sexually transmitted infections is worthwhile for pregnant adolescents during the third trimester, a Canadian study showed.

"We screen all adolescent pregnancies at baseline and again during the third trimester. This is different from adults, who we only screen at baseline," said Dr. Anjali Aggarwal of the Hospital for Sick Children in Toronto.

Part of the concern is that teenage women tend to use condoms less often once they become pregnant (Am. J. Public Health 2009 April 16 [doi:10.2105/AJPH.2007.131870]; J. Natl. Med. Assoc. 2008;100:929-35). Some discontinue condom use because they consider them primarily for pregnancy prevention and much less so for avoidance of sexually transmitted infections (STIs), Dr. Aggarwal said in a poster session at the annual meeting of the North American Society for Pediatric and Adolescent Gynecology.

She and her associates at the University of Toronto hospital assessed 89 pregnant adolescents with a median age of 16 years (range, 13-17 years) who were screened both at baseline and during the third trimester. They also screened 77 of the same participants again during the postpartum period.

Overall, 26 patients (29%) were diagnosed with an STI during or after pregnancy. "I was surprised it was that high," Dr. Aggarwal said in an interview. Specifically, STIs were detected in 17 patients during the first trimester, 7 in the third trimester, and 1 in the postpartum period. An additional patient was diagnosed in the first trimester, treated, and then treated again during the third trimester, based on symptoms later in her pregnancy.

The finding that more than 25% of the patients identified as having an STI were diagnosed in the third trimester justifies routine rescreening, said Dr. Aggarwal.

Only one statistically significant risk factor was associated with an STI in pregnancy: a history of not using contraception, other than a condom. There was no significant association with patient age, previous pregnancy, or previous STI. Women who lived with a partner, lived with the baby's father, or reported only one previous sexual partner versus more than one were at lower risk of an STI during pregnancy. These factors only trended toward statistical significance.

Dr. Aggarwal said she plans to compare computer-based patient interviews with those done by clinicians. The goal would be to determine if pregnant teenagers are more forthright when interfacing with a computer, she said. ■