



EDITORIAL

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Antepartum Fetal Surveillance Today: The Case for Condition-Specific Fetal Testing

How would you characterize the practice of a doctor who always orders the same test, regardless of the patient's chief complaint? Unreasonable? Unheard of?

Historically, obstetricians have been challenged to choose a single "best" test from a battery of antepartum fetal surveillance tests that have surfaced during the past 3 decades. Such tests include maternal kick counts, contraction stress test, non-stress test (NST), vibroacoustic stimulation, computerized fetal heart rate (FHR) monitoring, biophysical profile, amniotic fluid index, Doppler velocimetry of umbilical and various fetal and uterine vessels, serial ultrasounds for fetal growth, etc. Quite frequently, obstetricians have been presented with literature comparing the diagnostic accuracies of these tests—among each other—without considering the different underlying pathophysiologies of the various high-risk conditions.

The primary purpose of antepartum fetal surveillance has been to avoid fetal death and perhaps neonatal morbidity due to fetal hypoxemia and acidosis. Based on more than 35 years of experience, as well as a critical appraisal of the litera-

ture, 2 conclusions are apparent: (1) There is no ideal test for every high-risk fetus, and (2) the predictive accuracy of each test depends on the underlying pathophysiology. Therefore, before choosing the ap-

propriate testing, one has to determine what specific pathophysiology places the fetus at risk. The known pathophysiologic processes leading to fetal death or in utero neurologic injury are decreased uteroplacental blood flow, decreased gas exchange (at the trophoblastic membrane level), metabolic causes, fetal sepsis, fetal anemia, fetal heart failure, and umbilical cord accident. Decreased uteroplacental blood flow is the primary pathophysiology in mothers with chronic hy-

pertension, preeclampsia, collagen, and renal or vascular disease, as well as in most cases of early (<32-34 weeks) idiopathic intrauterine growth restriction (IUGR). Here, the compromised fetus frequently

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presents with sequential hemodynamic changes, which are best detected by serial Doppler velocimetry abnormalities in the uterine arteries, umbilical artery, fetal middle cerebral artery, and ductus venosus. However, not all IUGR fetuses follow the same pattern, so it is important to also incorporate some biophysical assessment (NSTs or biophysical profiles), as well as serial ultrasounds for fetal growth. In this clinical setting, Doppler velocimetry evaluates the fetal cardiovascular system, whereas

the biophysical assessment evaluates fetal central nervous system function.

Decreased gas exchange is the primary pathophysiology in postdates and in some late-onset (>32-34 weeks) IUGR fetuses that present with decreased abdominal circumference measurement and decreased amniotic fluid volume. Here, Doppler velocimetry tests are often normal, and therefore, the best tests to follow these fetuses are amniotic fluid volume assessments, NSTs, and fetal biophysical profiles. One should also remember that the most effective “preventive strategy against postdates” is accurate dating by first trimester crown-rump length measurement.

The most frequent metabolic pathophysiologic processes that can place the fetus at risk are fetal hyperglycemia with secondary fetal hyperinsulinemia, as frequently seen in fetuses of diabetic mothers, or primary fetal hyperinsulinemia, as seen in Beckwith-Wiedemann syndrome. In pregestational diabetic mothers who have no vasculopathy (White classes B, C, and D), the main threat to the fetus is lactic acidosis. Unlike hypoxemic acidosis, lactic acidosis is frequently associated with false-negative biophysical assessments and also false-negative Doppler results. The reason for the false-negative biophysical profile assessments is that elevated maternal serum glucose levels can cause increases in fetal breathing, fetal movements, FHR reactivity, and amniotic fluid volume. In this clinical setting, the best tests are maternal glucose levels and ultrasounds to rule out fetal macrosomia, asymmetric fetal overgrowth, and polyhydramnios.

In the clinical setting of diabetic mothers with vasculopathy (White classes F and R), in addition to the threat of fetal lactic acidosis, there

is the threat of reduced uteroplacental blood flow. In such cases, both Doppler velocimetry and fetal biophysical assessments are the tests of choice, given their high sensitivity in detecting hypoxemic acidosis.

Fetal sepsis is the primary pathophysiologic process that should be suspected in women presenting with preterm premature rupture of the membranes (PROM), preterm labor, or maternal fever. Here, the most sensitive test in ruling out intra-amniotic infection is amniocentesis (for glucose, white blood cell count, and cultures). The best noninvasive tests are daily NSTs or biophysical profiles and amniotic fluid volume assessments. In patients with preterm PROM, the presence of severe oligohydramnios, persistent nonreactive NST, or abnormal biophysical profiles is associated with an approximately 70% to 90% risk for fetal infection.

Fetal anemia is another pathophysiologic process that can threaten the fetus in cases of maternal sensitization (to Rh or other irregular antibodies), fetomaternal hemorrhage, parvovirus B19 infection, fetal hydrops, or twin-twin transfusion syndrome. Here, the best noninvasive tests are ultrasounds for fetal liver length measurements (in the presence of fetal chronic hemolytic anemia) and middle cerebral artery peak systolic velocity measurements. However, these 2 tests are not reliable after 34 weeks. Placentomegaly, polyhydramnios, and abnormal biophysical profile are late findings. Of course, the “gold standard” test is cordocentesis for direct measurement of the fetal hemoglobin and hematocrit.

Fetal heart failure is the pathophysiology that should be suspected in the presence of sustained fetal arrhythmia, nonimmune hydrops, placental chorioangioma,

or aneurysm of the vein of Galen. Here, the best tests are M-mode fetal echocardiography (to diagnose the exact type of arrhythmia), continuous FHR monitoring (to determine time spent in sinus rhythm), Doppler of the fetal venous circulation, ultrasounds (to rule out placentomegaly, hepatomegaly, and hydrops), and biophysical profiles (in the presence of fetal hydrops).

Umbilical cord accident should not be a “diagnosis of exclusion” after an adverse pregnancy outcome. The possibility of umbilical cord accident should be suspected in the antepartum period in very specific circumstances such as umbilical cord entanglement, as seen in monoamniotic twins, velamentous cord insertion, funic presentation, oligohydramnios (especially when associated with IUGR), and noncoiled umbilical cord. Here, verification of any of the above diagnoses should be made by using color Doppler. However, there are no reliable antepartum fetal surveillance tests. Frequent NSTs to rule out persistent variable FHR decelerations and/or umbilical artery Doppler velocimetry to rule out a systolic notch have been proposed as means of fetal surveillance.

The time has come to reject the notion that there is a single “best” antepartum fetal surveillance test for all high-risk conditions—there isn’t one! The clinician should determine the nature of the underlying pathophysiology of the high-risk condition and then apply the appropriate condition-specific antepartum fetal testing.



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