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Fetal Electrophysiology

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In this issue of *Pacing And Clinical Electrophysiology*, Peter Van Leeuwen and colleagues from the Department of Biomagnetism, Gronemeyer Institute, Bochum, Germany, describe the serial heart rate variability assessment and electrophysiologic assessment of a twin pregnancy, during flecainide treatment of fetal supraventricular tachycardia in one twin.¹ This analysis of medication effects in the mother and both twins demonstrates a degree of thoroughness uncommon to many accounts of supraventricular tachycardia (SVT) management in the fetal literature.

While on the surface this manuscript represents a well-written case report, its importance is much greater to cardiac electrophysiology as a whole. Van Leeuwen demonstrates the ability, using fetal magnetocardiography (fMCG)—a completely passive, safe, and non-invasive tool—to detect potentially life-threatening heart rate variability abnormalities and cardiac conduction changes in a presumably healthy twin fetus. The author further analyzes the drug's effect in the pregnant patient herself. Whether the changes manifest in the “unaffected” twin occurred solely as the result of flecainide exposure or were due to natural complications of the twin gestation itself is not the point. This report shows the potential that a form of intensive cardiac electrophysiologic diagnostics and care might be possible using fMCG and might favorably impact pregnancy outcome.

This case also demonstrated what many of us who are involved in the prenatal electrophysiologic assessment of the developing human have long suspected—that fetal distress and perhaps demise itself might be the final common pathway resulting from diagnosable and potentially preventable cardiac electrophysiologic disease. Electrophysiologic alterations appear to elude commonly used obstetrical tests such as echocardiography and fetal monitoring. In a recent report by Zhao et al., nearly one-third of fetuses with congenital heart block were found to have unsuspected junctional or ventricular tachycardia by fMCG.²

Van Leeuwen points out the high incidence of fetal demise in the older mother. Indeed, Reddy and colleagues report that in just the last 3 weeks of pregnancy, primiparous mothers over 40 stand almost a 1% chance of fetal loss.³ Multiple pregnancy is known to be a significant risk factor for fetal death even in the most advanced healthcare systems. In one study⁴ of 355 twin and triplet pregnancies, at least one fetus died in the latter two trimesters of pregnancy in 13 (3.5%). For the monamniotic monochorionic twin pregnancy, this risk is substantially higher. Often premature delivery is implemented on the basis of very scant information regarding the fetal ischemic condition, such as reduced fetal heart rate

variability. Cardiotocography (CTG), as utilized for fetal nonstress testing, provides less precise measures than in this report using fMCG, yet when CTG or biophysical profiles are nonreassuring, many twins are delivered prematurely. As many as 26% of the twins are premature. If diagnostics such as were used in this case report were more readily available, early delivery might be reduced.

To bring fMCG to clinical use around the world will require an outcry of support. The very sensitive SQUID recording devices and accompanying magnetically shielded environments are expensive. Hospitals have not been assured of investment recovery. Study of high-risk pregnancies such as this can define the clinical role of fetal magnetocardiography in serial analysis of high-risk maternal/fetal conditions.

It is important to point out to the electrophysiologic community that it is not only those fetuses with brady or tachy arrhythmias that suffer from electrophysiologic disease warranting fMCG study, but a much greater number of unsuspected fetuses (like the accompanying twin in this case) where heart rates are considered normal by all common obstetrical monitoring techniques. Fetuses with subclinical life-threatening electrophysiologic diseases can show intermittent ischemia, T-wave alternans, QT prolongation, QRS conduction delay, and other subtle but potentially lethal manifestations of congenital or acquired cardiac ion channel dysfunction.^{5–10} Such changes can accompany medication exposure as seen here, or maternal-fetal illnesses, placental insufficiency, hydrops fetalis, or genetic or acquired ion channelopathies. If the number of electrophysiologic deaths due to these diseases in the fetus, infant, child, or young adult could be accurately assessed and compared to the number of sudden deaths in the older adult with coronary artery disease (6–12/1,000 affected individuals/year), there is little doubt that the emphasis for future cardiac research would shift to a much younger age. Given the potential for the very young to live a long and healthy life if properly diagnosed, a major effort to bring forward new exciting technologies for improved fetal electrophysiologic diagnosis is in order. Hidden electrophysiologic disease takes a great toll. It will be through these new methods of testing that medical successes will be shown to come in very small and very soft packages.

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