Uterine and umbilical artery Doppler combined with placental growth factor assay for placental function expertise in the assessment of fetal growth restriction: a response

On behalf of all our coauthors, we thank Drs Carbillon, Benbara, and Fermault for their keen interest in our work. Currently, clinicians working in high-risk pregnancy care have very limited tools with which to infer a specific placental diagnosis for severe preeclampsia or fetal growth restriction (FGR) in the antenatal period. It is well established that both clinical disorders are strongly associated with abnormal uteroplacental perfusion; our observations showed bilateral abnormal uterine artery Doppler waveforms in 58% of a cohort of 196 patients with FGR and abnormal umbilical artery Doppler studies. Convincing evidence that these circumstances result in hypoxia-mediated injury to the placental villous trees was elegantly demonstrated in a magnetic resonance oximetry publication from London, United Kingdom. Case-control studies, such as that cited by Herraiz et al, illustrate the distribution of abnormal secretion patterns of placental growth factor (PlGF) and soluble fms-like tyrosine kinase (sFLT1) in maternal blood when either preeclampsia or FGR is established, in this publication at a mean gestational age of 27 weeks. However, readers should note that pregnancies ending with 2 other placental diseases causing normotensive FGR that are also not characterized by very suppressed PlGF levels at <20 weeks of gestation, developed disease recurrence—with normal (ie, non-predictive) uterine artery Doppler waveforms. Furthermore, note that pregnancies ending with 2 other placental diseases causing normotensive FGR that are also not characterized by placental bed vascular pathology, namely, fetal vascular malperfusion and T-cell villitis of unknown etiology, likewise showed normal uterine artery Doppler waveforms.

In everyday discussions in tertiary care maternal-fetal medicine settings, most high-risk clinicians are not aware of these key concepts in placental disease-specific progression. I encourage the group led by Dr Carbillon and others to study high-risk pregnancies similarly and to always capture placental pathology, to plan clinical care for future research in a pathology-specific manner. Overall, we can therefore begin to advance the concept of placenta disease—specific clinical care to benefit our patients.
Swati Agrawal, MBBS, MSc  
Division of Maternal-Fetal Medicine (Placenta Program)  
Department of Obstetrics and Gynaecology  
Mount Sinai Hospital  
University of Toronto  
Ontario, Canada  

W. Tony Parks, MD  
Department of Pathology and Laboratory Medicine  
Mount Sinai Hospital  
University of Toronto  
Ontario, Canada  

John C. Kingdom, MD  
Division of Maternal-Fetal Medicine (Placenta Program)  
Department of Obstetrics and Gynaecology  
Mount Sinai Hospital  
University of Toronto  
Ontario, Canada

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REFERENCES

Human embryo at 10 weeks’ gestation: a letter

TO THE EDITORS: We read the manuscript “Human embryo at 10 weeks’ gestation” with some unease.1 This article provides a hysteroscopic view of an early pregnancy at the time of surgical termination—a procedure that was reported as having been performed because the pregnancy was “angular”. “Angular” pregnancy, although not defined by the authors in this article, is a term for a laterally positioned but correctly sited pregnancy. This is a variation of normal and is not associated with any adverse pregnancy outcomes.2 We believe that this term is outdated, and a recent international consensus on terminology in early pregnancy endorsed this opinion and advised that the term should not be used in clinical practice.3

In addition, we question the clinical justification to perform a “diagnostic” hysteroscopy on a wanted pregnancy that is potentially viable. Accurate assessment of pregnancy location can easily and safely be made on transvaginal ultrasound. Therefore, the rationale to perform a hysteroscopy in this case is unclear.

Joel Naftalin, PhD  
Cecilia Bottomley, PhD  
Davor Jurkovic, PhD  
Early Pregnancy Unit  
University College London Hospitals NHS Foundation Trust  
Euston Road, London, United Kingdom  
joel.naftalin@nhs.net  

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