Letter to the Editor

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Therapeutic trials of maternal position therapy are needed to clarify the causes of abnormal maternal hemodynamic profiles and reduced umbilical vein flow in fetal growth restriction

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To the Editor,

I congratulate Dr. Daniele Farsetti and his colleagues for their recent intriguing contribution to the Journal of Perinatal Medicine, "Umbilical venous flow and maternal hemodynamics as predictors of impaired fetal growth in gestational diabetes" [1]. This new study, which builds upon their already well-developed concept of the "cardiac-fetal-placental unit" [2] shows that in patients with gestational diabetes, reduced umbilical venous flow (UV-Q) percentiles and low cardiac output (CO)/high systemic vascular resistance (SVR) maternal hemodynamic profiles are associated with reduced future birth weights. I wish to point out, however, that the explanation of these thought-provoking associations remains unclear for the following reasons:

(1) Maternal hemodynamic profiles are whole-body assessments of global maternal hemodynamic function and are therefore relatively insensitive to local and possibly severe reductions of placental perfusion and fetal oxygenation due to elevated uterine venous pressure [3].
(2) Chronic and recurrent obstruction of maternal venous return may periodically reduce maternal CO, thereby activating the sympathetic nervous system and the renin angiotensin aldosterone system, which may then create a persistent "low CO/high SVR" hemodynamic profile [3].
(3) UV-Q may decrease quickly and dramatically due to changes in maternal position which increase intervillous space pressure – which then produces limited "sluice flow" within the fetal vessels of the chorionic villi [3].
(4) A reduced UV-Q percentile at 28–32 weeks of gestation might be the *result* of previous fetal injury, as well as a *cause* of reduced future growth since an already-injured fetus may not grow as well as an uninjured one and may therefore not need as much oxygen to support its already limited growth potential.
(5) Despite possibility 4), I believe that reduced UV-Q is, in fact, an important and common cause of decreased fetal growth and that obstruction of uterine venous return may be an important cause of both reduced UV-Q and reduced uteroplacental perfusion [3].

One way to clarify the causes and significance of both abnormal hemodynamic profiles and reduced UV-Q would be to perform randomized controlled trials of cardiac output-guided maternal positioning to systematically prevent obstruction of maternal and uterine venous return during the second half of gestation. If such "position therapy" produced parallel improvement of a) hemodynamic profiles, b) UV-Q percentiles and c) fetal growth, this result would strengthen the theory that obstruction of uterine venous return at the inferior vena cava is a preventable cause of fetal growth restriction. Investigation of this possibility would also do much to refine and clarify the valuable concept of the cardiac-fetal-placental unit [2, 4].

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Conflict of interest: The author knows and has consulted and corresponded extensively with Markus Osypka, PhD, owner of Osypka Medical, Inc. which manufactures and sells the Electrical Cardiometry system of impedance cardiography, which the author used in his research at UCSD from 2008 until 2015. The author does not have and has never had any business or financial relationship with Dr. Osypka.

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