modification of the uterine veins modulates placental blood flow. ■

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For drainage of decidual blood, the maternal venous compartment operates as 1 functional unit with the heart and the microcirculation: a response

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I am grateful for the evaluative read and comments on the review "Origins of abnormal placentation: why maternal veins must not be forgotten." The clarification of the histologic features of peri-implantation trophoblast interference with decidual veins is very constructive. This explanation again emphasizes that this process is different from what happens at the arterial site and occurs weeks before the spiral arteries become patent.

In addition, another very important point is cited in the comment: there is always an open communication between the implantation site and the maternal veins. It should be recognized that the same is true on the other side of the venous vascular tree: in contrast to the ventricular outflow tracts, there is no valve between the right atrium and the vena cava. This makes the venous compartment an open (nevertheless dynamic) canal between the internal organs and the heart. This serves its physiological property to operate as 1 functional unit with the heart and the microcirculation. As a consequence, venous drainage from the intervillous space does not solely depend on successful or unsuccessful venous modifications at the site of implantation, as the commentators suggested. An abnormal function downstream from the site of implantation or an inadequate response of distant maternal veins or cardiac diastole to early gestational trophoblast signaling will, in a retrograde direction, influence the outflow of blood from the intervillous space. The same is true for all other internal organs. From basic physiology studies, it is well known that, in contrast to the arterial system, minor pressure changes in the venous compartment can cause major volume shifts. Abnormal venous hemodynamic function, already present before conception, has been linked with unexplained recurrent pregnancy loss[1] and is associated with low plasma volume.[2] The latter has been shown to predispose to recurrence of hypertensive complications in pregnancy.[3]

I am grateful for stressing once more the need to increase the research into the role of the currently underexplored venous compartment before and during pregnancy and to align the evidence from histologic placental bed studies with that from maternal hemodynamics research. ■

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