Boundary Conditions for Three Placental Flow Fields That May Predict and Cause Intra-Uterine Growth Retardation

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Introduction

Altered end-diastolic flow in the umbilical artery is a reliable predictor of intrauterine growth retardation (IUGR). It appears that an earlier and more sensitive prediction can be made when chorionic vessel flow patterns are characterised; we hypothesize that this results from remodelling of the villous circulatory bed. However, neither the true extent nor the causative mechanism of these altered flow patterns is known. In addition to the villous tree, the lacunar blood flows and the spiral arteries may also be prime suspects. Unfortunately, direct observation of these physical flows is restricted.

Preliminary enquiry indicates that each system may be compared to a unique physical flow problem. The villous tree as a complicated flow-in-pipes system; the lacunar pools as a porous medium flow; the spiral arteries as a wall shear stress interactions problem.

The Chorionic Vascular Tree

The chorionic vascular tree may be approximated to a system of flow-in-pipes. In engineering systems, these problems are normally solved empirically. These solutions approximate losses and gains in a flow system by accounting the effects of standard piping elements (i.e. elbows, tees, pumps, contractions/expansions, etc.). However, the implied assumptions of such an engineering system may not be justifiable in the variable flow setting of the chorionic vascular tree. Among the most difficult challenges are:

•determination of an idealised geometry for the vascular tree,
•understanding of the fluid dynamic effects of the pulsatile flow
•determination of an acceptable approximation for blood viscosity
•understanding the wall effect of the elastic blood vessels

Utero-Placental Flow

Numerous approximations of uteroplacental blood flow and solute transport have been proposed as a means to uncover underlying pathophysiologic processes (of IUGR and other entities). In these efforts, analytical solutions or mathematical models are applied to approximate physical processes in the uteroplacental flow bed. These models, while intricate, do not account for the presumed variations in intervillous flow mechanics that may be seen.

The utero-placental flow in a single cotyledon (or placentone) is generated by the 'sources' and 'sinks' of spiral arteries and decidual veins. In light of the symmetric homogeneity of the villous tree, it may be reasonable to construct an idealised model of the placentone and determine utero-placental flow experimentally.

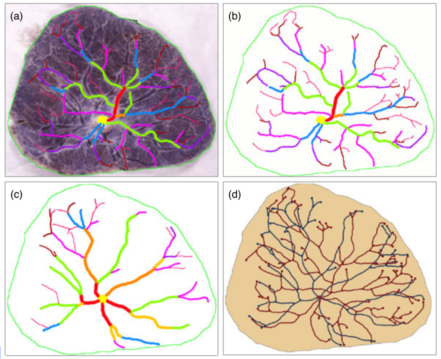

Fig 1. Highlighted vasculature of the chorionic plate: a) Photograph of placenta with arterial tree, b) and c) arterial and venous vasculature, d) computational approximation of both vascular trees (from 2).

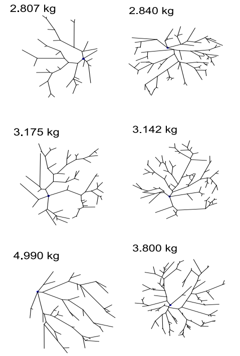

Fig 2. Variation of the chorionic vasculature, six computational arterial tree networks from human placenta, with fetal birth weights (from 2.)

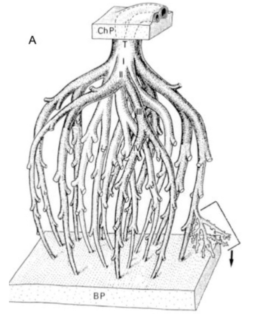

Fig 3. A: Large stem villi of the villous tree, B) Higher magnification of peripheral branches of the villous tree (from 1).

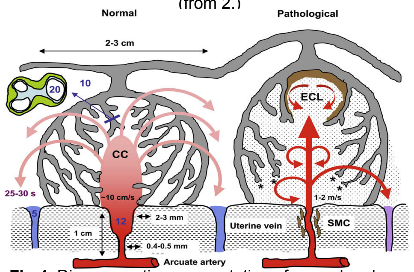

Fig 4. Diagrammatic representation of normal and pathologic alteration of the villous tree in response to altered spiral arterial in-flow (as predicted by 3.)

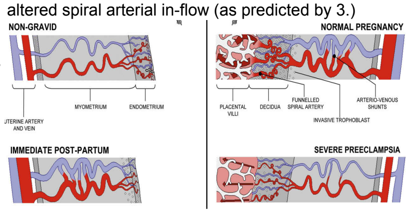

Fig 5. Diagrammatic representation of spiral arteries in the non-gravid uterus, in pregnancy, in pre-eclampsia, in post-partum (from 3).

The Spiral Arteries

Variation in the size and shape of the spiral arteries is associated with the physiological state of the uterus (non-gravid, post-partum, gravid, in pre-eclampsia, etc.). In the diseased state, the causality of these morphological changes in the spiral arteries is not entirely known (altered trophoblastic invasion has been implicated). Nonetheless, it is postulated variations in spiral arterial flow bring about other changes in the health of the uterus, fetus, and mother (such as villous tree changes, IUGR, pre-eclampsia, and so on). Furthermore, the shear interactions between the blood flow and arterial walls may result in additional alteration of the geometry of the spiral arteries.

Accordingly, it would be beneficial to precisely know the flow in the spiral arteries in order to accurately determine uteroplacental flow, and to assess whether incipient changes in spiral arterial flow incite further local and regional pathologic changes.

Our Goals

1. Determine the extent of feasible simplification of the chorionic vascular tree.
2. Determine an idealised geometry for the fetal villous tree within the uteroplacental blood pool.
3. Determine the uteroplacental blood flow velocity field, using a scaled experimental model.
4. Determine idealised geometries for the spiral arteries in normal gravid and non-gravid uterus, with a goal of computing shear-wall interactions and their possible effect on spiral arterial development.

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