Hemodynamic Analysis of Hyrtl Anastomosis in the Human Placenta

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Abstract

The Hyrtl anastomosis is a common connection between the umbilical arteries near the cord insertion in most human placentas. It was speculated that it equalizes the blood pressure between the territories supplied by the umbilical arteries. However, its functional role in the regulation and distribution of fetal blood flow to the placenta has not yet been explored. A computational model has been developed for quantitative analysis of hemodynamic characteristic of the Hyrtl anastomosis in cases of discordant blood flow in the umbilical arteries. Simulations were performed for cases of either increased placental resistance at the downstream end or reduced arterial blood flow due to some pathologies upstream of one of the arteries. The results indicated that when placental territories of one artery impose increased resistance to fetal blood flow, the Hyrtl anastomosis redistributes the blood flow into the second artery in order to reduce the large pressure gradients which are developed in the affected artery. When one of the arteries conducts a smaller blood flow into the placenta and a relatively smaller pressure gradient is developed, the Hyrtl anastomosis rebuilds the pressure gradients in the affected artery and redistributes blood flow from the unaffected artery to the affected one to improve placental perfusion. In conclusion, the Hyrtl anastomosis plays the role of either a safety valve or a pressure stabilizer between the umbilical arteries at the placental insertion.

Key words: Umbilical artery, Fetal blood circulation, Biofluid simulations, Discordant blood flow.
Introduction

The placenta is a vital organ that maintains feto-maternal exchange of oxygen, nutrients and waste products during fetal development. Fetal blood to the placenta is provided by a pair of umbilical arteries which arise from the fetus internal iliac arteries. Naturally, it is assumed that umbilical blood flow is equal in both arteries, and consequently, they are expected to perfuse similar volumes of placental territories. However, Doppler measurements of velocity waveforms and post-labor evaluations revealed cases of discordant umbilical arteries (Naki et al., 1997; Trudinger & Cook, 1988; Harper & Murnaghan, 1989; Dolkart et al., 1992), which may be attributed to significant differences in either arterial diameter or the placental volume supplied by these arteries. It was also observed that the degree of discordance is largely reduced as pregnancy advances probably due to the maturation of the Hyrtl anastomosis between the umbilical arteries (Predanic et al., 1998).

The Hyrtl anastomosis is a common connection between the umbilical arteries in most human placentas (Fig. 1a). The anatomical structure of this anastomosis is of a large variability, but it was always found in the vicinity of the cord insertion (Raio et al., 1999 and 2001). Generally, it was hypothesized that the Hyrtl anastomosis plays an active role in the regulation and distribution of fetal blood flow to the placenta (Ulberg et al., 2003b). It was speculated that it equalizes the blood pressure between the territories supplied by the umbilical arteries (Priman, 1959). It was also considered as a shunt (e.g., safety valve) in case of partial compression of the placenta during uterine contractions or occlusion of one umbilical artery (Priman, 1959; Benirschke & Kaufmann, 1995). Nevertheless, the hemodynamic characteristics of Hyrtl anastomosis in feto-placental circulation have not been evaluated. Accordingly, a computational study has been developed for quantitative analysis of the functional role of Hyrtl anastomosis in distribution of fetal circulation in case of discordant blood flow in the umbilical arteries.
Method

Description of the Model

Hyrtl anastomosis may be either a single connecting vessel or a fusion between the umbilical arteries (Raio et al., 2001; Ulberg et al., 2003b). The majority of the anastomoses (up to 90%) are of a single connecting tube, which may be transverse or oblique to the arteries (Ulberg, 2003a). The physical model for the present study was chosen to be an H-type anastomosis. Thus, the two umbilical arteries are represented by two straight tubes and the Hyrtl anastomosis is included by a transverse connection (Fig. 1b). The diameters, $D_1 = D_2 = 4$ mm, and arterial length of 80 mm, where $L_{up} = L_{down} = 40$ mm of the umbilical arteries, as well as the geometry of the Hyrtl anastomosis ($D_{anast} = 4$ mm, $L_{anast} = 8$ mm) were chosen from averaged morphometric data (Raio et al., 1999 and 2001; Ulberg et al., 2001; Priman, 1959).

The downstream resistance of the arterial vasculature in the placental territories which are supplied by each umbilical artery is represented by a cylindrical porous media of length $L_{porous} = 20$ mm (Fig. 1b). The resistances of the porous cylinders downstream of arteries 1 and 2 are $R_1$ and $R_2$, respectively. The length of the upstream segment of the model ($L_{up}$) was chosen to ensure a parabolic velocity distribution in the vicinity of the anastomosis. The length of the downstream segment ($L_{down}$) was chosen to ensure reduction of the parabolic velocity distribution into a uniform flow (i.e., plug flow) when fetal blood arrives to the porous media. In this study we assumed that fetal blood flow in the umbilical arteries may be simulated by a uni-directional steady flow into the placental vasculature.

Governing Equations

The physical domain of the problem is a network of rigid three-dimensional (3D) tubes. Fetal blood flow into the placenta is assumed to be steady, incompressible and laminar with no-slip conditions at the walls. Accordingly, the continuity and Navier-Stokes equations are,

$$\nabla \cdot \mathbf{U} = 0$$

(1)
\[
(U \cdot \nabla)U = -\frac{1}{\rho} \nabla P + \nu \nabla^2 U - \frac{\nu}{\alpha} U
\] (2)

where \( U \) is the velocity vector, \( P \) is the fluid pressure and \( \rho \) and \( \nu \) are the fluid density and kinematical viscosity, respectively. The last term on the right hand side of Eq. (2) represents the dissipative force of the porous media at the peripheral end of the umbilical arteries. The viscous resistance coefficient \( \frac{1}{\alpha} \) is given by (Blevin, 1984),

\[
\frac{1}{\alpha} = \frac{RA}{\mu L}
\]

where \( R \) is the resistance, \( A \) is the cross-section area, \( L \) is the length of the porous cylinders and \( \mu \) is the viscosity. For simulations of the resistance of normal placental territories downstream of each umbilical artery it is assumed to be \( R_N=4.325\times10^5 \) N-s/m\(^5\) (0.053 mmHg/ml/min), where the subscript \( N \) stands for 'normal' (Pennati et al., 1997). For fetal blood at body temperature we used \( \rho = 1060 \) kg/m\(^3\) and \( \mu = 0.004 \) kg/m-s (Guettouche et al., 1992; Thompson & Trudinger, 1990).

**Boundary Conditions**

Normally, fetal blood flow into the placenta during the third trimester is at a rate of \( Q_N=500 \) ml/min. Accordingly, if the umbilical arteries are identical, the averaged flow rate through each is 250 ml/min (Lees et al., 1999). Hence, for the normal symmetric case we assumed that the flow into each artery is \( Q_1 = Q_2 = 250 \) ml/min. The published data for arterial pressure in the umbilical arteries is ranging between 2,665 Pa and 10,665 Pa (20 to 80 mmHg) (Reynolds, 1978; Vermeulen, 1982; Weiner et al., 1989). For the present model (Fig. 1b) we assumed that the normal inlet pressure is \( P_{\text{inlet \: N}}=5,150 \) Pa (38.6 mmHg) similar to the data of Weiner et al. (1989). The outlet of the model is assumed to be at the level of fetal capillaries.
within the cotyledons, hence, \( P_{\text{outlet}} = 1,200 \) Pa (9 mmHg) (Nicolini et al., 1989; Pennati et al., 1997).

In order to explore the role of the anastomosis in cases of discordant umbilical arteries it is necessary to simulate cases with different boundary conditions due to discordant fetal blood flow in the umbilical arteries. The variability of such cases is enormous, and thus, we investigated two limiting states. In the first we assumed that the total fetal blood flow into the placenta is constant and divided between the umbilical arteries. Hence, any reduction of the input flow rate to one artery leads to a corresponding increase in the second one. This state may be considered as if the umbilical arteries are supplied by a flow generator (FG), and mathematically it is described by \( Q_1 + Q_2 = Q_N \). In the second state we assume that the pressure at the inlet to both umbilical arteries is always identical independent on downstream resistance variations. This state may be considered as if the umbilical arteries are supplied by a pressure generator (PG), and mathematically it is described by \( P_{\text{inlet} 1} = P_{\text{inlet} 2} = P_{\text{inlet} N} \) for each artery.

**Plan of Numerical Simulations**

The normal fetal blood flow in two identical umbilical arteries was simulated by \( Q_1 = Q_2 = Q_N / 2 \), \( P_{\text{inlet} 1} = P_{\text{inlet} 2} = P_{\text{inlet} N} \) and \( R_1 = R_2 = R_N \). In order to simulate cases of discordant blood flow in the umbilical arteries and the resulted contribution of the Hyrtl anastomosis we assumed two general options. In the first, the flow into one artery is smaller than into the other one due to either geometry differences between the arteries or some upstream pathology (Heifetz, 1988; Cook et al., 1995; Sanchez et al., 2005). We assume that the input flow rate may decrease by 20% or 40% of the normal flow. In the second option, the umbilical arteries supply placental territories of different resistances to fetal blood flow (Thompson & Trudinger, 1990; Ménigault et al., 1998). Here, we assumed cases of 30%, 60% and 100% increase in placental resistance to artery 1, while the resistance to artery 2 remains \( R_N \). A summary of all simulations of discordant fetal blood flow in the umbilical arteries is provided.
in Table 1. Evaluation of blood flow distribution due to the Hyrtl anastomosis was done for both states of FG and PG boundary conditions. The results for two independent umbilical arteries without the anastomosis (i.e., a single uniform tube) were computed for the given inlet flow rate and the outlet pressure ($P_{\text{outlet}}$).

**Computation Method**

The governing equations were simultaneously solved by implementing the finite volume computational fluid dynamics package of FLUENT (Fluent Inc., Lebanon, NH). The Euler implicit algorithm was used for solving of the partial differential equations in a segregated manner. A spatially second order upwind discretization scheme was used to minimize numerical dissipation. The 3D geometry of the umbilical arteries and their connection via the Hyrtl anastomosis was converted into a discrete mesh with GAMBIT (Fluent Inc., Lebanon, NH). The mesh was composed of 880,193 cells in order to ensure a parabolic velocity profile in the midst of the anastomosis. For the normal symmetric case a single umbilical artery consisted of only 249,644 cells. The mesh volume density was much larger in the vicinity of the anastomosis.
Results

Computational simulations of discordant flow in the umbilical arteries were performed by imposing on artery 1 either an increased placental resistance at the downstream end or a reduced inlet blood flow due to some upstream pathology, while artery 2 is subjected to normal blood flow. In order to simplify the presentation of the results we investigated the pressure distributions along the central axis of each umbilical artery as a representative of the three-dimensional distribution of arterial pressure. The results of blood flow in the umbilical arteries when artery 1 feeds placental territories with increased resistance are depicted in Fig. 2 for numerical simulations with and without the Hyrtl anastomosis in comparison with the normal case of identical arteries feeding a symmetric placenta. The results for the model with the anastomosis are presented for boundary conditions of either FG or PG.

In the absence of the anastomosis, increased placental resistance by 30%, 60% and 100% at the peripheral end of artery 1 (while \( Q_1 = 250 \text{ ml/min} \)) resulted in significant increases in the pressures up to 20%, 41% and 70%, respectively (Figs. 2a and c). Note that the static pressure at the junction that simulates the insertion of the umbilical artery in the placenta is elevated, while the slope of the pressure along the artery is practically identical. This pattern is due to the increased placental resistance that increases the pressure drop along the porous media that represents the placenta, but does not change the pressure drop along the artery segment. On the other hand, when the anastomosis was included in the model using the FG boundary conditions the increase in pressures is only 10%, 19% and 28%, respectively, while using the PG boundary conditions yielded pressure distributions along the vessel which are almost identical to the normal symmetric case (Fig. 2c). As blood flows through the anastomosis from artery 1 into artery 2, which is feeding placental territories with normal resistance (\( R_2 = R_N \)) the pressure drop along the central axis of artery 2 is also increasing due to increased blood flow through the porous cylinder (Fig. 2b). For the simulation with FG boundary conditions the pressures in artery 2 demonstrates an increase of 9%, 16% and 24% compared...
with normal symmetric placenta, as the resistance of the placental territories of artery 1 increases by 1.3, 1.6 and 2.0, respectively.

The corresponding redistribution of blood flow via the Hyrtl anastomosis is demonstrated in Fig. 3. The results are shown for the different increased resistance of $R_1$ normalized by the normal placental resistance. The differences between inlet and outlet flow rates of the umbilical arteries due to the flow through the anastomosis are given in Figs. 3a and b for the simulations with FG and PG boundary conditions, respectively. It clearly demonstrated that the anastomosis is distributing the inlet blood flow by transferring some of the blood flow of artery 1 into artery 2. The flow rate through the anastomosis itself is depicted in Fig. 3c., which demonstrates the increased blood flow rate through the anastomosis ($Q_{HA}$) as the resistance of the placental territories supplied by artery 1 is increased. The diverting of blood flow from artery 1 into artery 2 decreases the pressure drop between the anastomoses and the placental territories of artery 1, which would otherwise be larger when $R_1$ is increased.

The pressure distributions along the central axis of the umbilical arteries for 20% and 40% reduced flow rate at the inlet to artery 1 due to an upstream pathology is shown in Fig. 4 for cases with and without Hyrtl anastomosis in comparison with the normal case. In the absence of the anastomosis, reduction in the flow rate into artery 1 significantly reduces the pressures along the tube with a maximal decrease of 16% and 32% for 20% and 40% reduction of inlet flow, respectively (Figs. 4a,c). However, when the anastomosis was present in the model using the FG boundary conditions blood flow was redistributed from artery 2 into artery 1 and the axial pressure drop demonstrated a maximal decrease of only 2% and 4%, respectively (Fig. 4a). Using the PG boundary conditions similarly yielded a maximal decrease in pressure drop of 3% and 7%, respectively (Fig. 4c). The variations of the corresponding pressure distributions along the central axis of artery 2 compared to the normal identical arteries feeding a symmetric placenta are very small (Fig. 4b). The corresponding largest increase in
pressure drop in artery 2 due to redistribution of some blood into artery 1 is only 2% and 5%, respectively, for simulation with the FG boundary conditions.

The corresponding variation of inlet and outlet flow rates as a function of the reduced flow rates into artery 1 are given in Fig. 5a,b. For upstream discordant arterial flow the anastomosis compensates the missing flow rate in artery 1 by transferring some of the blood from artery 2 into artery 1 (Fig. 5a). In case of upstream pathologies the efficacy of the anastomosis is demonstrated by the significant increase of blood flow through the anastomosis as the inlet flow rate into artery 1 decreases (Fig. 5c).
Discussion

Hyrtl’s anastomosis between the umbilical arteries is present in the majority of pregnant women just upstream of the insertion into the placenta. In this work we performed a quantitative analysis of the role of this anastomosis in equilibrating the pressure gradients in discordant umbilical blood flow in the arteries and the corresponding re-distribution of blood perfusion into the placenta. We have selected for the analysis two possible situations that lead to discordant umbilical blood flow; increased resistance of the placental territories supplied by one of the arteries and reduced blood flow into one of the umbilical arteries due to increased upstream resistance. The simulations presented in this work have demonstrated the different physical role of Hyrtl anastomosis in equilibration of discordant umbilical blood flow due to different causes.

The numerical prediction of pressure distribution along the axis of the umbilical arteries when the peripheral resistance of placental territories to one of them is increased (Fig. 2 a, c) demonstrates the role of the anastomosis as a safety valve for releasing highly developed pressures in one side. We compared the pressure distribution along the central axis of artery 1 for a case of $R_1=2R_N$ with that of the averaged area pressure distribution along the axis, and found that both curves were practically identical. This supports the use of the pressure at the central axis as a representative pressure. When the placenta is partially occluded to fetal blood flow (e.g., $R_1>R_N$ ; $R_2=R_N$), inlet pressure in the artery 1 may rise up to 70% (for $R_1=2R_N$) with respect to the reference normal in the absence of the Hyrtl anastomosis. For simulations with the FG boundary conditions the maximal rise is only 28%. Nevertheless, existence of the anastomosis largely reduces this pressure rise to values very close to the normal model when PG boundary conditions are incorporated (Fig. 2c). The inlet pressure in the unaffected artery (i.e., artery 2) is also increasing in the predictions with FG boundary conditions (Fig. 2b) because additional blood flow is supplied via the anastomosis just before the outlet into the placenta. The efficacy of the anastomosis increases when the resistance of placenta...
territory increases and more blood is transferred into other territories fed by the unaffected artery (Fig. 3).

In this study we solved the governing equations for two limiting cases of PG and FG boundary conditions. The FG condition corresponds to a generator with infinite internal resistance \( R_{\text{Generator}} \to \infty \), while the PG condition corresponds to a generator with negligible internal resistance \( R_{\text{Generator}} \to 0 \) (Desoer & Kuh, 1969). In reality, fetal heart is the generator that supplies arterial blood into the umbilical arteries and may have a finite resistance \( 0 < R_{\text{Generator}} < \infty \). Thus, real cases are most likely to be on the hatched area between the lines for PG and FG (Fig. 3c). Moreover, if the resistance to blood flow through the anastomosis increases (for example, a very small anastomosis diameter), the hatched area in Fig. 3c becomes narrower and moves downward towards the abscissa. In the absence of Hyrtl anastomosis \( R_{\text{Anastom}} \to \infty \) \( Q_{\text{HA}} \) becomes zero.

The placenta is an organ that develops to maturity, along with the umbilical cord, within a relatively short time. When significant discordant arterial blood flow is being developed in the umbilical cord it has been found in a single placenta with non-anastomosing umbilical arteries that the larger artery supplied a larger volume of placental territories (Hitschold, 1992). Similarly, an averaged asymmetry of about 1:2 between placental areas supplied by each umbilical artery was observed in 61 placentas with Hyrtl anastomoses (Ulberg et al., 2001). When discordant flow is being developed due to increased resistance at the placental territories of one artery, the concomitant development of a Hyrtl anastomosis induces more blood supply to the other artery and thereby more blood to its placental territories. As a result, the placental territories supplied of the more efficient artery are expected to develop larger volumes with increased exchange surfaces than those supplied by the affected artery. In fact, this pattern of development was observed in a relatively old study of 78 placentas with Hyrtl anastomoses (Szpakowski, 1974).
In a more recent study of the fetal vasculature of 210 placentas only a few were found to have symmetric placental pools supplied by identical umbilical arteries (Bekov, 1990). In 67% of the placentas, discordant umbilical arteries were observed with the larger artery supplying larger placental surface. The ratio between these surfaces was 1:2 in 39% of the casts, 1:3 in 15% and 1:4 in 8%. Similarly, it was also found that discordance in the diameters of the umbilical arteries is associated with abnormal insertion of the umbilical cord in the placenta (Raio et al., 1998). In fact, French groups (cited in Raio) observed that marginal insertion of the umbilical cord is more likely in cases of discordant umbilical arteries or a single umbilical artery. Thus, the Hyrtl anastomosis may be considered as a guard that redirect fetal blood flow in order to induce development of an asymmetric placenta that will ensure optimal performance during embryonic development.

When blood flow rate in artery 1 is much smaller than in artery 2 due to some upstream pathology or arteries with different diameters, the pressure drop in artery 1 is much smaller in the absence of the Hyrtl anastomosis (Fig. 4a,c). A drop of 16% and 32% of the maximal values of the normal artery were observed for 20% and 40% reduction of the inlet blood flow. When the Hyrtl anastomosis is present it is compensating the flow rate in artery 1 by redistributing the blood flow between arteries 1 and 2, and thereby the pressure drop in the artery 1 is re-elevated nearly to the normal values. Namely, the decrease of 16% and 32% in the pressure is reduced in the presence of an anastomosis to only 2% and 4% for the FG boundary conditions (Fig. 4a), and to 3% and 7% for the PG boundary conditions (Fig. 4c), respectively. If $R_{Anastom}$ is increased, the hatched area in Fig. 5c becomes narrower and moves towards the abscissa. In the absence of Hyrtl anastomosis ($R_{Anastom} \rightarrow \infty$) $Q_{HA}$ becomes zero. Here again, the anastomosis is acting as a pressure equilibrator and restores the outlet flow as if the umbilical arteries are normal (Fig. 5c).

In the present model we assumed for the Hyrtl anastomosis an idealized geometry of a perpendicular single tube connecting between the parallel umbilical arteries (Fig. 1b). This
assumption is based on observations in real placentas, which reported that Hyrtl anastomosis was mostly (up to 90%) a single true connection between the arteries (Ulberg, 2003a). In terms of hemodynamic characteristics the H-type model represents the geometry of a single connecting tube. In case of an inclined anastomosis there may be an additional pressure drop, but it is very small and with negligible effects on the overall flow pattern. Thus, the H-type Hyrtl anastomosis is a good representative model for all cases of a single connecting tube anastomosis. Similarly, in cases the Hyrtl anastomosis is a fenestration or a fusion between the arteries (up to 10% of the observed placentas; Ulberg, 2003a) the anastomosis resistance to blood flow is much smaller than that of the H-type and its functional performance will largely improve.

In conclusion, we conducted for the first time a quantitative analysis of the hemodynamic characteristics of fetal blood flow through the Hyrtl anastomosis in the mature placenta. The results of this study clearly demonstrated the important functional role of the Hyrtl anastomosis in regulating arterial blood flow in discordant arteries in order to equilibrate the pressure gradients in the arteries before insertion into the placenta. When placental territories of one artery impose increased resistance to fetal blood flow, the Hyrtl anastomosis redistributes the blood flow into the second artery in order to reduce the large pressure gradients which are developed in the affected artery. As a result, the placental territories with rich blood supply are likely to be developed into larger volumes than the part with increased resistance in order to maintain fetal well being. When one of the arteries conducts a smaller blood flow into the placenta and a relatively smaller pressure gradient is developed, Hyrtl anastomosis rebuilds pressure gradients in the affected artery and redistributes blood flow from the unaffected artery to the affected one to improve placental perfusion.
Reference


Figure Legends

Figure 1 (a) Anatomical scheme of the umbilical cord and placenta showing the Hyrtl anastomosis. (b) The physical model of two umbilical arteries connected via an H-type Hyrtl anastomosis.

Figure 2 Distribution of static pressure along the central axes of the umbilical arteries and porous cylinders in models with and without the Hyrtl anastomosis for different resistances of the placental territories fed by artery 1. (a) Pressure in artery and cylinder 1 for boundary conditions of FG, (b) Pressure in artery and cylinder 2 for boundary conditions of FG, (c) Pressure in artery and cylinder 1 for boundary conditions of PG, (d) Pressure in artery and cylinder 2 for boundary conditions of PG. Legend of symbols: $R_1=1.3R_N$ without HA - ●, with HA - ○; $R_1=1.6R_N$ without HA - ▲, with HA - △; $R_1=2R_N$ without HA - ■, with HA - □.

Figure 3 Blood flow rates in the umbilical arteries and the Hyrtl anastomosis for increasing resistance $R_1$ of the placental territories fed by artery 1. (a) Arterial inlet and outlet flow rates for boundary conditions of FG, (b) Arterial inlet and outlet flow rates for boundary conditions of PG, (c) Flow rate through the Hyrtl anastomosis. Legend of symbols: inlet to artery 1 - ■, outlet of artery 1 - □; inlet to artery 2 - ●, outlet of artery 2 - ○.

Figure 4 Distribution of static pressure along the central axes of the umbilical arteries and porous cylinders in models with and without the Hyrtl anastomosis for different reduced inlet flow rates into artery 1. (a) Pressure in artery and cylinder 1 for boundary conditions of FG, (b) Pressure in artery and cylinder 2 for boundary conditions of FG, (c) Pressure in artery and cylinder 1 for boundary conditions of PG, (d) Pressure in artery and cylinder 2 for boundary
conditions of PG. Legend of symbols: $Q_1=0.8RQ_N$ without HA - ●, with HA - ○; $Q_1=0.6RQ_N$ without HA - ▲, with HA - △.

**Figure 5** Blood flow rates in the umbilical arteries and the Hyrtl anastomosis for reduced inlet flow rates into artery 1. (a) Arterial inlet and outlet flow rates for boundary conditions of FG, (b) Arterial inlet and outlet flow rates for boundary conditions of PG., (c) Flow rate through the Hyrtl anastomosis. Legend of symbols: inlet to artery 1 - ■, outlet of artery 1 - □; inlet to artery 2 - ●, outlet of artery 2 - ○.

**Table Legends**

**Table 1** Different types of discordant fetal blood flow in the umbilical arteries.
Table 1. Types of fetal blood flow in the umbilical arteries.

<table>
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<tr>
<th>Type of Discordant Arteries</th>
<th>With Hyrtl Anastomosis</th>
<th>No Anastomosis</th>
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<tr>
<td></td>
<td>Flow Generator (FG)</td>
<td>Pressure Generator (PG)</td>
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<td>Increased Placenta Resistance (downstream resistance)</td>
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<td>R₂=Rₙ</td>
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<td>Increased Placenta Resistance (downstream resistance)</td>
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<tr>
<td>Increased Upstream Resistance</td>
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<td>Increased Upstream Resistance</td>
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Figure 1a.
Figure 1b.
Figure 2.
Figure 3.
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Figure 5.