

Gynecology and Women's Health Care

Maternal-Fetal Circulation and the Unsuspected Ability of Human Eukaryotic Cells to Dissociate the Water Molecule

Arturo Solís Herrera*, María Del Carmen Arias Esparza and Ruth Isabel Solís Arias

Department of Ophthalmology, Human Photosynthesis Study Center, Mexico.

*Correspondence author

Arturo Solís Herrera,

Department of Ophthalmology, Human Photosynthesis Study Center, Mexico.

Submitted: 7 Sept 2026; Accepted: 2 Oct 2026; Published: 23 Oct 2026

Citation: Herrera, A. S., Esparza, M. D. C. A., & Arias, R. I. S. (2026). Maternal-Fetal Circulation and the Unsuspected Ability of Human Eukaryotic Cells to Dissociate the Water Molecule. *G Women's Health Car* 7(2):1-5.

Abstract

The fetal circulation is an entirely transient event, not replicated at any point in later life, and functionally distinct from the pediatric and adult circulations. Understanding the physiology of the fetal circulation is vital for accurate interpretation of hemodynamic assessments in utero, but also for management of circulatory compromise in premature infants, who begin extrauterine life before the fetal circulation has finished its maturation. The cardiovascular system develops early in the embryonic stage of development. It starts towards the end of the third week or at the beginning of the fourth week of fetal development. Once the cardiovascular system is fully established, blood circulation commences and the embryo can directly derive nutrients from its own blood supply. In this paper we analyze some data about the characteristics of the maternal-fetal circulation, which have not been elucidated if we start from the fact that our body takes oxygen from the air that surrounds it. We also comment on the differences when we take into account the hitherto unknown capacity of human eukaryotic cells to oxygenate themselves, from the dissociation of intracellular water molecules, as in plants.

Keywords : Circulation, Fetal, Hydrogen, Energy, Oxygen, Water

Introduction

As the fetus develops within the womb, fetal circulation is established during the early stages of development, allowing the growing fetus to receive the required oxygen and nutrients as well as dispose of waste products. This type of circulation refers to the circulatory system of a fetus which differs from postnatal circulation. Fetal circulation, unlike postnatal circulation, involves the umbilical cord and placental blood vessels which carry fetal blood between the fetus and the placenta. It is usually established in the fetal period of development and is designed to serve prenatal nutritional needs, as well as permit the switch to a neonatal circulatory pattern at birth. Good respiration in the neonate depends on normal circulatory changes occurring at birth (transitional circulation), which results in oxygenation of the blood in the lungs when fetal blood flow through the placenta ceases. Prenatally, the lungs do not provide gas exchange, and the pulmonary vessels are vasoconstricted. Instead, the placenta acts as the gas exchange unit to oxygenate fetal blood. The three most important vascular structures in the transitional circulation are the ductus venosus, foramen ovale, and ductus arteriosus.

Fetal Circulation

The fetal circulation is characterized by low systemic vascular resistance (SVR) with high systemic blood flow and high

pulmonary vascular resistance with low pulmonary blood flow. Given the low oxygen tension of the fetus, the fetal circulation allows for preferential flow of the most oxygenated blood to the heart and brain, two of the three “vital organs.” With the placenta rather than the lungs being the organ of gas exchange, most of the right ventricular output is diverted through the patent ductus arteriosus (PDA) to the systemic circulation. In fact, the pulmonary blood flow constitutes only approximately 7% to 8% of the combined cardiac output in fetal lambs [1]. Once the main arteries and veins as well as the heart are developed, usually after the 8th week of fetal development, deoxygenated blood is returned from the fetal systemic circulation to the placenta via two umbilical arteries, which branch off the fetal internal iliac arteries. Highly oxygenated, nutrient-rich blood flows from the placenta to the fetus via the umbilical vein. Approximately half of the blood in the umbilical vein bypasses the liver to flow into the ductus venosus, a fetal vessel connecting the umbilical vein to the inferior vena cava. The other half flows into the sinusoids of the liver and enters the inferior vena cava via the hepatic veins.

Blood flow through the ductus venosus is regulated by a sphincter mechanism close to the umbilical vein. When the sphincter contracts, more blood is diverted to the portal vein and the hepatic sinusoids, and less to the ductus venosus. Although an anatomic sphincter in the ductus venosus has been

described, its presence is not universally accepted. However, it is generally agreed that there is a physiologic sphincter that prevents overloading of the heart when venous flow in the umbilical vein is high, as seen during uterine contractions for example. The spiral morphology of some of the blood vessels present, including in the uterus myometrium; could be the explanation for the regulation of blood flow in the absence of appropriate muscular structures, given its similarity to the Archimedean spiral (Figure 1).

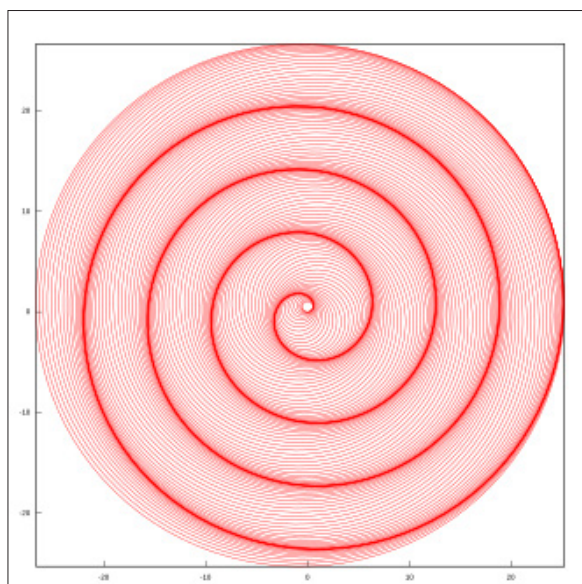


Figure 1: Osculating circles of the Archimedean spiral, tangent to the spiral and having the same curvature at the tangent point. The spiral itself is not drawn but can be seen as the points where the circles are especially close to each other [2].

The Archimedean spiral has a variety of real-world applications. Scroll compressors, used for compressing gases,

and liquids, have rotors that can be made from two interleaved Archimedean spirals, involutes of a circle of the same size that almost resemble Archimedean spirals, or hybrid curves [3].

Fetal Hemoglobin (HbF) is a principal oxygen carrier in the blood of preterm and term neonates. Compared to adult hemoglobin, it has a significantly higher affinity for oxygen, and its theoretical oxyhemoglobin dissociation curve (ODC) is left-shifted accordingly. Tissue oxygenation measured with near-infrared spectroscopy (NIRS) during neonatal intensive care is directly affected by hemoglobin concentration. We performed a systematic qualitative review regarding the impact of HbF on tissue oxygenation monitoring by NIRS [4]. But both fetal hemoglobins, as well as adult hemoglobin, and chlorophyll, are compounds derived from protoporphyrin IX, a molecule present in all living beings, which have the intrinsic property of transforming the power of sunlight into energy that can be used by living beings, through the dissociation of the water molecule. What was previously attributed only to chlorophyll in plants [5]. Therefore, the supposed greater oxygen affinity of fetal hemoglobin should be interpreted as a greater ability to dissociate the molecule from water.

Maternal-Fetal Circulation and Atmospheric Oxygen

The anatomical elements that constitute maternal-fetal circulation, as well as blood flow and its direction, have been interpreted for decades based on the (erroneous) concept that our body takes oxygen from the air around it (figure 2) But if we consider the unsuspected ability of human (and fetal) eukaryotic cells to dissociate the molecule from water, then the interpretation of the morphological characteristics of the maternal-fetal circulation begins to make sense, since we assume that fetal cells oxygenate themselves and do not depend at all on atmospheric oxygen.



Figure 2: Image generated with AI for illustrative purposes: The interpretation of the maternal-fetal circulation today is since, theoretically, our body (and that of the fetus) takes oxygen from the air that surrounds it. Note the spiral formed by the elements contained in the umbilical cord.

An example contradicts the concept that the oxygen present in the fetus comes from maternal blood and therefore from the lungs and at the end of the atmosphere.

Pregnancy provokes enormous changes in the maternal cardiovascular system. Systemic vascular resistance and blood pressures fall, prompting a ~30% rise in total blood volume and resting cardiac output. The reduction in uteroplacental vascular resistance is particularly marked, causing the major portion of the increased cardiac output to be directed to that circulation by term [6]. In different published studies on uterine blood flow, sized noninvasively using transabdominal Doppler ultrasound

near term in healthy low-altitude women, an average value of 214 mL/min/kg is estimated (minimum 138 to maximum 292). The bilateral uterine artery blood flow averaged 719 mL/min or 214 mL/min/kg fetal weight. What accounts for such high levels of uteroplacental blood flow in human pregnancy? One factor is that not all the blood reaches the placenta as ~25% is directed to the myometrium and cervix (Figure 3) [7].

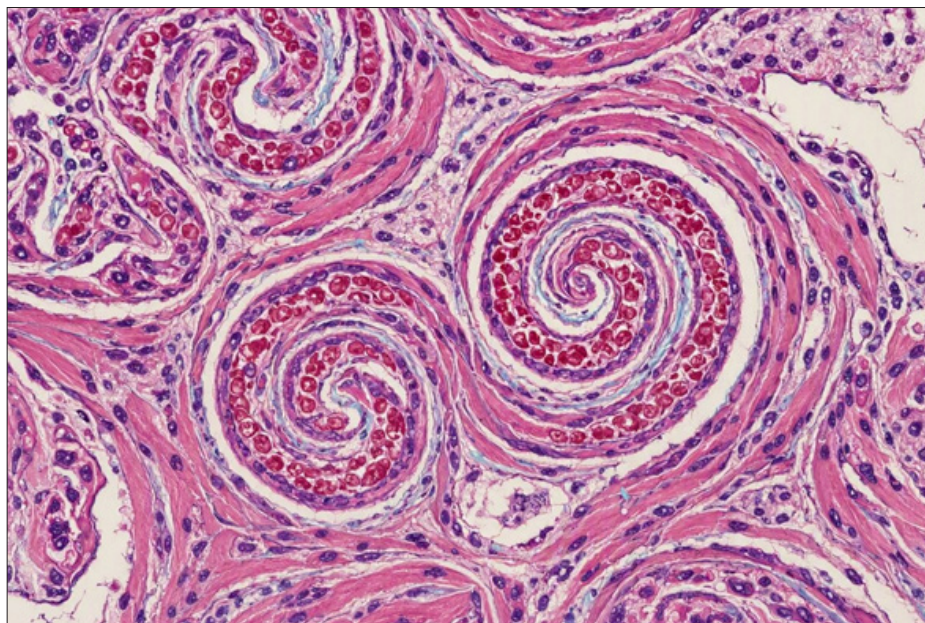


Figure 3: Spiral blood vessels, characteristic of the myometrium of the human uterus, are an important part of blood flow control.

Supposedly, a key factor stems from the venous equilibrator nature of human placental exchange and its consequences for fetal O_2 supply (from atmosphere). But with the discovery of molecules located inside eukaryotic cells, capable of transforming the power of sunlight into chemical energy that can be used by these cells, as in plants, the interpretation of the mechanisms that regulate blood flow must be radically modified, since the oxygen present in fetal tissues or even in maternal tissues comes from atmospheric oxygen, but each cell, both of the mother and the fetus, produces its own oxygen through the dissociation of the water molecule, also located

inside all cells. So, we must think that the only gas that the blood carries are CO_2 , but not oxygen; because the oxygen that is detected in the bloodstream is located inside the erythrocytes and is generated by the dissociation of the water molecule by hemoglobin, and both elements are intracellular. The opposing directions of maternal and fetal blood flow in a countercurrent system enable twice as an efficient transplacental exchange compared with a concurrent system in which maternal and fetal blood travels in the same direction and three times as efficient as a venous equilibrator system Figure 4 [8,9].

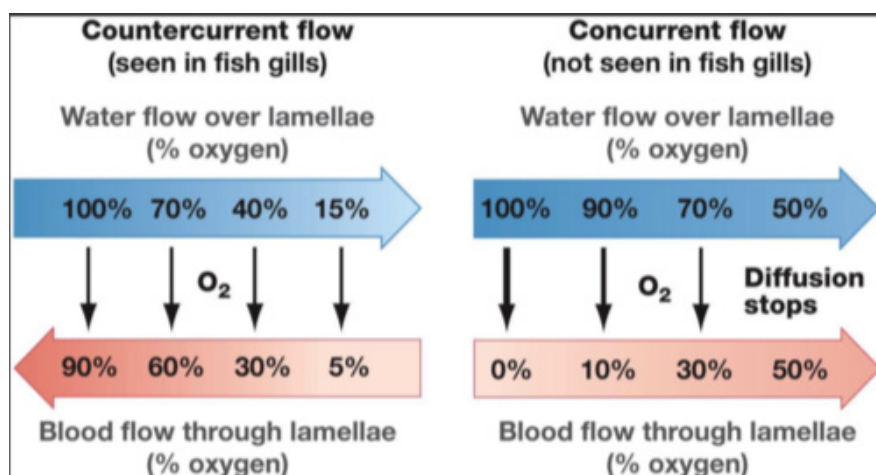


Figure 4: The current explanation of the known countercurrent mechanisms is based, erroneously, on the supposed diffusion of O_2 in water, which is minimal or absent, because water tends to repel O_2 . Therefore, the interpretation of this type of scheme must be based on the diffusion of CO_2 [10].

In a venous equilibrator system, the terminal uterine (spiral) arteries inject blood at high velocity into the intervillous space, which becomes a mixed pool stirred by fetal villi and has an intervillous P_{O_2} approximating that of the uterine vein. Given the need for an hypothetical O_2 gradient of at least 10 mmHg for diffusion to the umbilical vein (i.e., the vessel carrying oxygenated blood to the fetus), the uterine vein P_{O_2} sets the upper limit for umbilical venous P_{O_2} [11]. But let's point out a transcendental detail in the previous lines: blood does not transport O_2 , so the explanation cited above is biased, it is theoretical. Few studies have evaluated transplacental nutrient exchange in vivo in human pregnancy [12]. Only two have measured maternal arterial and uterine venous blood gases at low altitude, finding uterine venous P_{O_2} to range from 46 to 49 mmHg with an O_2 saturation of 72%–79% [13]. But we must consider that it is locally produced oxygen, which comes neither from the lungs nor from the atmosphere, before interpreting the figures and drawing conclusions.

So, the so-called gaseous exchange between the placenta and the fetus is limited to CO_2 , oppositely, O_2 is not exchanged between the mother and the fetus, in fact not even between contiguous cells, since the cells produce oxygen for themselves, and the cell that produces it consumes it, does not exchange it even with the interstitium or with nearby cells. So, when analyzing the dynamics of the bloodstream, we must think only about how CO_2 moves to reach the lungs quickly. Each cell produces oxygen by itself through the dissociation of water, as in plants.

Does The Fall in Uterine Artery Blood Flow Curtail Fetal Growth?

The altitude-associated reduction in fetal growth is not simply due to reduced uterine artery blood flow since even the lower blood flow observed at high altitude is sufficient to maintain uteroplacental O_2 delivery at near sea-level values [14]. This is a fact that confirms or at least maintains that the oxygen present inside all the cells of our body and that of the fetus, does not come from the atmosphere that surrounds us. Furthermore, the decrease in fetal growth began by pregnancy week 28 when, given its still relatively small size, O_2 supply would be expected to be sufficient for meeting fetal O_2 demands. Across many organs of the body, metabolic activity is closely linked with blood flow, with vasodilation supported by metabolic by-products such as K^+ , adenosine, lactate, H^+ , and CO_2 , thereby integrating O_2 demand with supply [15]. While it has long been known that fetal growth restriction develops when fetal O_2 supply drops below a “margin of safety”, and since the concepts expressed in the immediately preceding paragraph are totally theoretical, what follows is controversial because it is unclear which and how the pathways linking perfusion and metabolism [16].

Ovine models of heat-induced placental insufficiency result in hypoxemic and growth-restricted fetuses with altered nutrient utilization and insulin sensitivity, however, the contribution of hypoxia, that is: low levels of oxygen from the atmosphere to these fetal metabolic responses remains a critical knowledge gap [17]. While a focus within the field has been on how tissue

metabolism affects blood flow and nutrient delivery, we suggest that hypoxia itself, hitherto considered to be the result of insufficient transport of atmospheric oxygen to tissues through the bloodstream; whether resulting from placental insufficiency or lower uterine artery blood flow, can serve (theoretically) to widen the uterine arterial-uterine venous O_2 content gradient, lower umbilical venous P_{O_2} , and prompt metabolic changes to slow fetal growth to match O_2 supply with demand. But the circumstantial observation that human eukaryotic cells possess molecules derived from protoporphyrin IX, capable of dissociating irreversible, water molecules of intracellular localization also, demolishes almost the entire theoretical framework about fetoplacental circulation, which makes it necessary to elaborate a new theoretical framework, attached to the so far, unsuspected capacity of human cells to oxygenate themselves [18].

Fetal Circulation

The mechanisms governing umbilical venous return in early pregnancy remain incompletely understood, particularly the roles and interactions of arterial pulsatility, umbilical cord mechanics, and placental compliance [19]. The velocity pattern of venous return is related to cardiac performance and hepato-ductal resistance as follows: The E wave in early diastole describes the filling (driven by the central-venous pressure) of the relaxed ventricle. The time constant equals the ratio of elasticity over resistance in the ventricular wall. The A wave mirrors the contractility of atrial myocardium which is related to central venous pressure. The ratio of the forward A wave in the tricuspid valve to the backward A wave in the caval veins depends on ventricular capacitance. The S wave is related to systolic valvular shift. The velocity pulsations in the ductus venosus are transmitted backwards from the heart. The continuous velocity component in the ductus venosus and the non-pulsatile velocity in the umbilical vein are due to hepato-ductal and placental vascular resistance [20].

Numerical results of theoretical 3 D models show that venous flow is dynamically modulated by arterial pulsations transmitted through both the cord and placenta, producing phase-shifted venous deformation and low-amplitude oscillations. Like how the flow of liquids and gases works in the Archimedes spiral. Despite strong placental damping, the model predicts measurable venous flow oscillations with increasing amplitude toward the fetus. This behavior depends on cord elasticity and placental compliance. The numerical model also points out that mechanical interactions between arteries, vein, and Wharton's jelly contribute positively to the mean flow rate, demonstrating a pulsometer-like effect just as Archimedes' spiral happens. Doppler measurements confirm that venous flow is not strictly steady and that oscillation amplitude increases toward the fetus, which is consistent with simulations. Overall, our combined computational and in vivo approach reveals that the interplay between arterial pulsations and placental compliance is a key driver of umbilical venous return.

Conclusion

Therefore, the fundamental role of the ductus arteriosus, foramen ovale, ductus venosus, etc., will be better understood

if we correlate them with the biodynamics of CO₂, rather than with oxygen saturation, since each cell in our body has the necessary elements to oxygenate itself.

Acknowledgements

This work was supported by an unrestricted grant from Human-Photosynthesis® Research Centre. Aguascalientes 20000, México.

Conflict of Interest

None

References

1. Finnemore, A., & Groves, A. (2015). Physiology of the fetal and transitional circulation. *Seminars in Fetal and Neonatal Medicine*, 20(4), 210–216. <https://doi.org/10.1016/j.siny.2015.04.003>
2. Wikimedia Commons. (n.d.). Two moving spirals scroll pump [Animated image]. https://upload.wikimedia.org/wikipedia/commons/3/31/Two_moving_spirals_scroll_pump.gif
3. Sakata, H., & Okuda, M. (1997). Fluid compressing device having coaxial spiral members (U.S. Patent No. 5,603,614). U.S. Patent and Trademark Office. <https://www.freepatentsonline.com/5603614.html>
4. Pritišanac, E., Urlesberger, B., Schwaberg, B., & Pichler, G. (2021). Fetal hemoglobin and tissue oxygenation measured with near-infrared spectroscopy—A systematic qualitative review. *Frontiers in Pediatrics*, 9, 710465. <https://doi.org/10.3389/fped.2021.710465>
5. Herrera, A. (2015). The biological pigments in plants physiology. *Agricultural Sciences*, 6, 1262–1271. <https://doi.org/10.4236/as.2015.610121>
6. Osol, G., & Moore, L. G. (2014). Maternal uterine vascular remodeling during pregnancy. *Microcirculation*, 21, 38–47.
7. Blackburn, S. (2007). Maternal, fetal, and neonatal physiology: A clinical perspective (3rd ed.). *Elsevier Health Sciences*.
8. Faber, J. J., Thornburg, K. L., & Binder, N. D. (1992). Physiology of placental transfer in mammals. *American Zoologist*, 32, 343–354.
9. Leiser, R., & Kaufmann, P. (1994). Placental structure: In a comparative aspect. *Experimental and Clinical Endocrinology & Diabetes*, 102, 122–134.
10. Quizlet. (n.d.). [Image]. <https://o.quizlet.com/Smmph6iF4jnMsBa9nQn9BQ.png>
11. Battaglia, F. C., & Meschia, G. (2013). Review of studies in human pregnancy of uterine and umbilical blood flows. *Developmental Period Medicine*, 17, 287–292.
12. Michelsen, T. M., Holme, A. M., & Henriksen, T. (2017). Transplacental nutrient transfer in the human in vivo determined by 4 vessel sampling. *Placenta*, 59, S26–S31.
13. Pardi, G., Cetin, I., Marconi, A. M., Bozzetti, P., Buscaglia, M., Makowski, E. L., & Battaglia, F. C. (1992). Venous drainage of the human uterus: Respiratory gas studies in normal and fetal growth retarded pregnancies. *American Journal of Obstetrics and Gynecology*, 166, 699–706.
14. Moore, L. G., Jahnigen, D., Rounds, S. S., Reeves, J. T., & Grover, R. F. (1982). Maternal hyperventilation helps preserve arterial oxygenation during high-altitude pregnancy. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology*, 52, 690–694.
15. Clifford, P. S. (2011). Local control of blood flow. *Advances in Physiology Education*, 35, 5–15.
16. Meschia, G. (2014). Placental respiratory gas exchange and fetal oxygenation. In *Handbook of physiology* (pp. 181–191). *American Physiological Society*.
17. Wesolowski, S. R., & Hay, W. W., Jr. (2016). Role of placental insufficiency and intrauterine growth restriction on the activation of fetal hepatic glucose production. *Molecular and Cellular Endocrinology*, 435, 61–68.
18. Herrera, A. S., & Arias Esparza, M. D. (2025). The unsuspected ability of the human cell to oxygenate itself: Applications in cell biology. *Medical Research Archives*, 13(3). <https://doi.org/10.18103/mra.v13i3.6344>
19. Machrafi, H., Capelle, X., Grandfils, S., Vanlinthout, C., Kridelka, F., & Dauby, P. C. (2026). Influence of arteries, vein and placenta interactions on the blood flow in the human umbilical cord. *PLoS ONE*, 21(9), e0358954. <https://doi.org/10.1371/journal.pone.0358954>
20. Moll, W. (1999). Venous return in the fetal-placental cardiovascular system. *European Journal of Obstetrics & Gynecology and Reproductive Biology*, 84(2), 133–137. [https://doi.org/10.1016/S0301-2115\(98\)00322-4](https://doi.org/10.1016/S0301-2115(98)00322-4)

Copyright: ©2026 Arturo Solís Herrera. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.