Origin of maternal hypervolemia

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Maternal hypervolemia is currently believed to be caused by the mineralocorticoid activity of maternal steroids. This is incorrect for a number of reasons. Progesterone actually acts more like a diuretic by competing with aldosterone for the same receptor sites. The second reason is that while estrogen and progesterone are increased by 10 times during pregnancy, they are increased by 20 times at puberty with no hypervolemia. Misunderstanding the origin of maternal hypervolemia has led to misunderstanding its only related pathology preeclampsia/eclampsia. After all preeclampsia is the only disease in which maternal circulatory volume is adversely affected. To understand maternal hypervolemia we have to go back to our basic physiology that any physiologic hypervolemia is a response to hypovolemic stimuli or a hypovolemic state. In a normal pregnancy the hypovolemic state is initiated by the growing and demanding fetus. Maternal bleeding into the fetal circulation triggers maternal circulatory regulatory mechanisms, the renin-angiotensin-aldosterone system. The activation of this cascade by the hypovolemic effect of the concepts results in salt and water retention simply because angiotensin is not only the most potent known vasoconstrictor but also a very potent promoter of salt and water retention as well. The level of maternal hypervolemia corresponds to the hypovolemic state imposed by the fetus and its growing needs and maintained throughout the pregnancy. How? The equation blood pressure=cardiac output x peripheral resistance explains that. We know that blood pressure is almost constant in a normal pregnancy, while cardiac output (circulatory volume) is increased. This means that peripheral resistance must have been decreased, manifested by massive venodilatation. Decreased peripheral resistance provides; Room for the expanding circulatory volume and dampens the vasoconstrictive effects of angiotensin. Like other hypovolemic states, renal regulatory prostaglandins are secreted in pregnancy also, which dampens the vasoconstrictive effect of angiotensin furthermore. These are responsible for the normal pregnancy refractoriness to the pressure effect of angiotensin. The equilibrium of these factors is crucial in maintaining a normotensive hypervolemic pregnancy. While there are many publications on how the growing fetus is affected by changes in maternal circulatory volume there has not been any article arguing the hypovolemic effect of the fetus on maternal circulation.

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