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Comments on Greggory DeVore paper:

“Measurements of cardiac function in a prospective cross-sectional study of pregnant women between 10 and 39 weeks of gestation using a computerized brachial blood pressure cuff”

Tom Archer comments:

1) This is an outstanding and important paper which gives us (in a very thorough and easy-to-understand way) key maternal data which bring together a) gestational age, and b) maternal body position. These two factors have been difficult to separate out in many other studies and Dr. DeVore does an outstanding and thorough job. His article is a pleasure to read and was a real “eye-opener” for me.

2) He also had the wisdom of excluding women with a) hypertension, b) fetal growth restriction, c) fetal macrosomia, d) chronic hypertension, e) diabetes and f) “other systemic maternal conditions known to affect cardiovascular physiology.” The sample of women had a mean weight of 164 lbs. with a SD of 34 lbs. Hence, there were also few highly obese women in this study sample. It should be noted that these exclusions are wise, since any of the disorders listed above could confound the conclusions which were (presumably) meant to apply to “healthy” pregnancies.

3) Therefore, this was a study of “healthy women.” The result of this is that we are able to see what changes and challenges pregnancy imposes upon “healthy women.”

4) He also uses a technology (the Vicorder device) which uses a special BP cuff and a transfer function to acquire “central aortic” blood pressure tracings, which are derived from the brachial pulse waveform, in addition to giving us the “regular” brachial blood pressure readings. The ability to get “central aortic blood pressures” is a largely ignored technology which has been around for decades and which deserves more consideration by clinicians, since it looks at what pressure the left ventricle itself is exposed to during systole, which gives us insight into the degree of wall tension “stress” that the LV is exposed to during systole. This LV wall “stress” is correlated with the “central aortic blood pressure” by Laplace’s Law and is probably a major determinant of cardiac remodeling and activation of both the sympathetic nervous system (SNS) and the renin angiotensin aldosterone system (RAAS). Activation of these two systems may be responsible for long term CV damage in women who have been pregnant-- as with the long term sequelae of preeclampsia and with chronic hypertension. In short, Dr. DeVore is showing us in this article a better approach to blood pressure measurement than the usual brachial blood pressure cuff!

5) The article contains a wealth of information, but for me, here are some of the most relevant conclusions I draw from the paper:

A) **In “healthy” non-morbidly obese women cardiac output is maintained in all three positions: supine, left lateral and sitting.** This is an important finding, since other studies in other women have reported frequent decreases in CO in the supine position (as with the supine hypotensive syndrome of pregnancy and in many other less extreme examples).

B) Despite the maintained CO in all three positions, the total peripheral vascular resistance and the brachial and “central” aortic pressures are all lower in the LLD position than in the supine and sitting positions—at all gestational ages! Another way of saying this is that the blood pressures

and the total peripheral vascular resistance are higher in the supine and sitting positions than in the LLD position, and to me this suggests activation of the SNS and the RAAS in the supine and sitting positions, which is what I have proposed in my book as the “counterproductive cardiovascular compensation” which protects the mother from the supine hypotensive syndrome of pregnancy, but which also allows the persistence of the obstruction of uterine venous return associated with the supine position! In short, I believe that this paper offers evidence which is consistent with my theory of “counterproductive cardiovascular compensation” which allows the persistence of a hemodynamic situation in which the fetus, placenta and myometrium can be injured without the mother being aware that anything bad is happening to her baby.

This observation is also consistent with my theory that it is not so much the decrease in CO which may cause fetal, placental and myometrial injury, but rather the increase in uterine venous pressure.

C) Dr. DeVore seems to interpret this hemodynamic situation in the LLD position—of maintained cardiac output at a lower total peripheral vascular resistance and blood pressure-- as evidence of better organ perfusion, and I would agree, but perhaps not for the exact reason he intends. I would argue that—at least in the case of the uterus— its perfusion is better in the LLD position because of the lack of obstruction of uterine venous return and not so much due to a lower total peripheral vascular resistance, which is largely due to arteriolar tone. It is true, however, that uterine arteries and arterioles—but not spiral arteries—do constrict with increased sympathetic tone. **Therefore, perhaps our working hypothesis should be that improved uteroplacental perfusion in the LLD position is due to decreased resistance on both the arterial and venous sides of the uterine circulation!**

6) Again, I want to congratulate Dr. DeVore on this paper, as well as on the other two. I learned a great deal from reading them, and offer my constructive suggestions and amendments only with great humility in the face of such outstanding work!