The forgotten ventricle—right ventricular remodeling across pregnancy and postpartum: a report of original research

OBJECTIVE: Despite well-known cardiovascular adaptations leading to increased cardiac output, little is known about the right ventricle's (RV) response to the physiological demands of pregnancy.[1] Because complex geometry limits echocardiography, the standard for evaluation of the RV is cardiac magnetic imaging (cMRI).[2,3] Using cMRI, we report changes in the RV with associated biomarker surveillance.

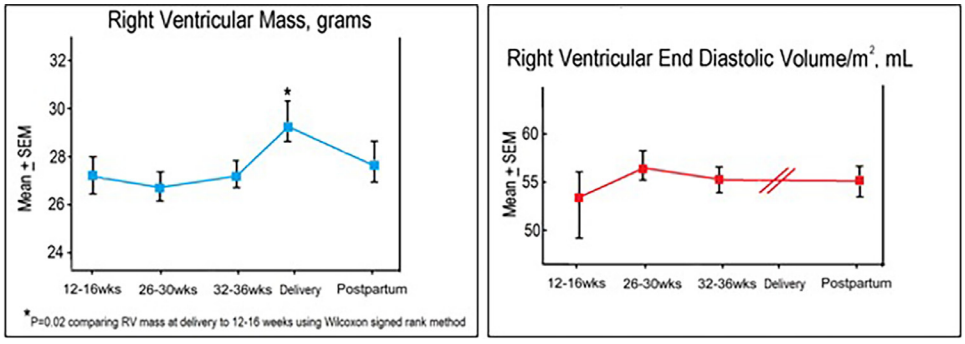
STUDY DESIGN: This was a secondary analysis of a prospective, observational study of pregnant individuals throughout pregnancy and the postpartum period using cMRI.[4] Twenty-three nulliparas aged 18−30 years with a singleton pregnancy and without hypertension, pregestational diabetes mellitus, preexisting cardiovascular disease, or substance use between June 2012 and May 2014 were included with Institutional Review Board approval. Each patient underwent cMRI at 12−16, 26−30, 32−36 weeks, delivery, and 12 weeks postpartum on a 1.5-Tesla coil system. End-diastolic and end-systolic endocardial and epicardial borders were systematically traced to measure RV dimensions. RV mass was used as a marker of cardiac remodeling, and papillary muscles and trabeculations were not included in the calculations. Right ventricular end-diastolic volume (RVEDV) was used as a measure of function and was evaluated in the left-lateral position. N-terminal pro-B-type natriuretic peptide (NT-proBNP) was measured using the STAT assay (NT-proBNP II STAT; Roche Cobas e601) at 12 weeks, delivery, and 12 weeks postpartum as a marker of ventricular volume expansion and pressure overload. Variables were compared with a function of time across pregnancy and indices stratified by habitus at the time of cMRI. RV mass was compared using Wilcoxon signed rank, and RVEDV was compared using repeated measures, mixed-random, and fixed-effects.

RESULTS: RVMass by cMRI across pregnancy and postpartum is shown in Figure 1. RVMass significantly increased from a mean±SEM of 27.4±0.6 g at 12−16 weeks to 29.3±0.9 g at delivery (P=.02), and this finding persisted when indexed to habitus (P=.03). Conversely, RVEDV did not significantly differ throughout pregnancy—53.0±3.0 mL/m^2 compared with 55.3±1.6 mL/m^2 (P>.05) (Figure). At 12 weeks postpartum, all changes of right ventricular remodeling—defined as RV mass—resolved.

Median (interquartile range [IQR]) NT-proBNP showed no significant differences at entry to the study (46 [33−75] pg/mL), at delivery (38 [18−81] pg/mL), or postpartum (17 [<15−75] pg/mL) (all P>.05). These findings persisted when analyzed according to habitus (P=.58) with distribution of median (IQR).

CONCLUSION: Using cMRI, the RV undergoes remodeling across pregnancy with subtle but statistically significant changes in RVMass from 12−16 weeks to delivery with resolution by 12 weeks postpartum. There were no significant changes in the RVEDV during any epoch of pregnancy or 12 weeks postpartum. We did not identify any significant changes in the serial measurement of NT-proBNP—a marker of ventricular volume expansion and pressure overload.

FIGURE
Right ventricular variables



A, RV mass (g). **B,** RV end-diastolic volume (mL/m^2). *Asterisk* denotes P=.02 comparing RV mass at delivery to 12−16 wks using the Wilcoxon signed rank method.

RV, right ventricle.

Ambia. Right ventricle remodeling during pregnancy. Am J Obstet Gynecol Glob Rep 2024.

Because increased RVEDV (>84 mL/m^2) is one of the most important markers for progressive right-sided heart failure, this nonsignificant finding may indicate that any increase in RVEDV in a pregnant patient could be a sign of right-heart failure. Further research into the impact of pregnancy on cardiac volume is necessary to clarify if changes exist, as these may be harbingers of future right-sided cardiac dysfunction with important implications for those with congenital heart disease or pulmonary hypertension.[5]

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The authors report no conflict of interest.
A portion of this paper was presented at the Society for Maternal-Fetal Medicine's 37th annual Pregnancy Meeting poster session IV on January 27, 2017 (Abstract number 641).

CREDIT AUTHORSHIP CONTRIBUTION STATEMENT: **Anne M. Ambia:** Writing – review & editing, Writing – original draft, Visualization, Validation, Investigation. **David B. Nelson:** Writing – review & editing, Writing – original draft, Methodology, Investigation, Data curation, Conceptualization. **Robert B. Martin:** Writing – review & editing, Investigation, Formal analysis. **Robert D. Stewart:** Writing – review & editing, Methodology, Investigation, Data curation, Conceptualization. **Susan Matulevicius:** Writing – review & editing, Resources, Methodology, Investigation, Data curation, Conceptualization. **Donald D. McIntire:** Writing – review & editing, Formal analysis, Data curation. **F. Gary Cunningham:** Writing – review & editing, Writing – original draft, Resources, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

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 https://doi.org/10.1016/j.xagr.2024.100335