Cardiac Responses to Submaximal Isometric Contraction and Aerobic Exercise in Healthy Pregnancy

VICTORIA L. MEAH[1], KARIANNE BACKX[1], JOHN R. COCKCROFT[2], ROB E. SHAVE[1,3], and ERIC J. STÖHR[1,2]

[1]*Cardiff School of Sport and Health Sciences, Cardiff Metropolitan University, Cardiff, UNITED KINGDOM;* [2]*Division of Cardiology, Department of Medicine, Columbia University Irving Medical Center, New York, NY; and* [3]*School of Health and Exercise Sciences, University of British Columbia, Kelowna, BC, CANADA*

ABSTRACT

MEAH, V. L., K. BACKX, J. R. COCKCROFT, R. E. SHAVE, and E. J. STÖHR. Cardiac Responses to Submaximal Isometric Contraction and Aerobic Exercise in Healthy Pregnancy. *Med. Sci. Sports Exerc.*, Vol. 53, No. 5, pp. 1010–1020, 2021. **Purpose:** The increased physiological demand of pregnancy results in the profound adaptation of the maternal cardiovascular system, reflected by greater resting cardiac output and left ventricular (LV) deformation. Whether the increased resting demand alters acute cardiac responses to exercise in healthy pregnant women is not well understood. **Methods:** Healthy nonpregnant (n = 18), pregnant (n = 14, 22–26 wk gestation), and postpartum women (n = 13, 12–16 wk postdelivery) underwent assessments of cardiac function and LV mechanics at rest, during a sustained isometric forearm contraction (30% maximum), and during low-intensity (LOW) and moderate-intensity (MOD) dynamic cycling exercise (25% and 50% peak power output). Significant differences (α = 0.05) were determined using ANCOVA and general linear model (resting value included as covariate). **Results:** When accounting for higher resting cardiac output in pregnant women, pregnant women had greater cardiac output during isometric contraction (2.0 ± 0.3 L·min^{-1}·m$^{-1.83}$; nonpregnant, 1.3 ± 0.2 L·min^{-1}·m$^{-1.83}$; postpartum, 1.5 ± 0.5 L·min^{-1}·m$^{-1.83}$; $P = 0.02$) but similar values during dynamic cycling exercise (pregnant, LOW = 2.8 ± 0.4 L·min^{-1}·m$^{-1.83}$, MOD = 3.4 ± 0.7 L·min^{-1}·m$^{-1.83}$; nonpregnant, LOW = 2.4 ± 0.3 L·min^{-1}·m$^{-1.83}$, MOD = 3.0 ± 0.3 L·min^{-1}·m$^{-1.83}$; postpartum, LOW = 2.3 ± 0.4 L·min^{-1}·m$^{-1.83}$, MOD = 3.0 ± 0.5 L·min^{-1}·m$^{-1.83}$; $P = 0.96$). Basal circumferential strain was higher in pregnant women at rest, during the sustained isometric forearm contraction ($-23.5\% \pm 1.2\%$; nonpregnant, $-14.6\% \pm 1.4\%$; $P = 0.001$), and during dynamic cycling exercise (LOW = $-27.0\% \pm 4.9\%$, MOD = $-27.4\% \pm 4.6\%$; nonpregnant, LOW = $-15.8\% \pm 4.5\%$, MOD = $-15.2\% \pm 6.7\%$; $P = 0.012$); however, other parameters of LV mechanics were not different between groups. **Conclusion:** The results support that the maternal heart can appropriately respond to additional cardiac demand and altered loading experienced during acute isometric and dynamic exercise, although subtle differences in responses to these challenges were observed. In addition, the LV mechanics that underpin global cardiac function are greater in pregnant women during exercise, leading to the speculation that the hormonal milieu of pregnancy influences regional deformation. **Key Words:** PRENATAL, AFTERLOAD, CYCLING, HEMODYNAMICS

APPLIED SCIENCES

Pregnancy results in profound adaptation to the maternal cardiovascular system as reflected by increased left ventricular (LV) size and cardiac output at rest (1). The significantly greater metabolic requirements along with the hemodynamic alterations to pregnancy increase cardiac demand at rest; thus, the capacity of the maternal cardiovascular system to adapt to this biological stress is an important determinant of prenatal health (2). Historically, exercise in pregnancy was discouraged because of a belief that it would overload the maternal heart, reduce uteroplacental blood flow, and impair nutrient and oxygen delivery to the fetus (3). This concern remains unsubstantiated (3), and prenatal physical activity is now actively encouraged in all healthy women (4,5); however, there is a paucity of empirical data examining cardiac responses to different types of exercise in pregnancy.

In contrast to assessing the heart at rest, understanding the dynamic response to physiological challenges (e.g., acute elevations in afterload or aerobic exercise) provides greater insight into cardiovascular function and, possibly, dysfunction (6,7). Isometric muscular contraction causes acute increases in blood pressure, heart rate, and systemic vascular resistance (SVR) in healthy adults (8,9), whereas submaximal aerobic exercise results in increased stroke volume, heart rate, blood pressure, and reductions in SVR (10,11). In nonpregnant populations, abnormal responses to these physiologic stimuli can unmask latent cardiac dysfunction and/or increased cardiovascular disease risk (6,7,12,13). At present, however, the prognostic value of such tests for cardiovascular-related pregnancy complications is limited as the normal responses to

Address for correspondence: Victoria L. Meah, Ph.D., Faculty of Kinesiology, Sport, and Recreation, University of Alberta, 8602 112 Street, Edmonton, Alberta, Canada T6G 2E1; E-mail: vmeah@ualberta.ca.

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physiological challenges in healthy pregnancy have not been well characterized (14).

Previous research in healthy pregnant women has shown that cardiac output and traditional indicators of LV function (e.g., LV fractional shortening) are maintained during dynamic cycling exercise (3). However, these measures, and others such as ejection fraction, do not fully explain how the maternal heart adjusts to acute alterations in cardiac load (preload and afterload) such as those experienced during exercise (15). Because cardiac output depends on preload, afterload, and contractility, direct measurements of heart muscle function can assist in determining whether pregnant women have an altered response to exercise. Such deformation parameters include LV strain and twist, previously termed "LV mechanics." Therefore, assessing LV deformation in conjunction with traditional echocardiographic measures in healthy pregnant women potentially provides a better understanding of the normal cardiac responses to acute changes in load and submaximal cycling exercise during gestation.

The aim of this study was to examine the dynamic cardiac responses to submaximal isometric forearm contraction and low- to moderate-intensity dynamic cycling exercise in healthy nonpregnant, pregnant, and postpartum women 1) to provide empirical data to support the safety of acute prenatal exercise in healthy pregnant women and 2) to characterize the "normal" responses to physiological challenge so that future work may investigate the prognostic value of such tests in complicated pregnancies. We hypothesized that, when accounting for baseline differences between groups, pregnant women would have similar hemodynamic (cardiac output, heart rate, stroke volume, and blood pressure) responses but greater LV mechanics (represented by greater LV strain) during isometric contraction and submaximal dynamic cycling exercise compared with nonpregnant and postpartum women.

METHODS

The study took place in the Cardiff School of Sport and Health Sciences, Cardiff Metropolitan University, UK, between January 2015 and April 2017. The experimental procedures were reviewed and approved by the Cardiff Metropolitan University Research Ethics Committee (16/3/01R). The study complied with the guidelines set out in the Declaration of Helsinki, apart from registration in a publicly accessible database.

Forty-five women volunteered to participate (nonpregnant, n = 18; pregnant [between 22 and 26 wk gestation], n = 14; postpartum [12–16 wk after delivery], n = 13) and provided written, voluntary informed consent. Based on self-report, volunteers were healthy nonsmokers, free from cardiovascular and/or metabolic diseases and were not taking any medication at the time of inclusion. All women completed relevant physical activity readiness questionnaires (16,17), and no contraindications to exercise were identified. All pregnant and postpartum women were primiparous, although women who had suffered one previous miscarriage before 12 wk gestation were included (pregnant, n = 2; postpartum, n = 2). Nonpregnant women were nulliparous and had not tried to conceive. No women in the pregnant or postpartum groups experienced cardiovascular complications during their gestation, as determined through completion of a follow-up questionnaire administered after delivery. Pregnant and postpartum women were asked to provide descriptive information regarding pregnancy outcomes; this included gestational age at delivery, mode of delivery, birthweight and sex of infant, development of complications (if any), breastfeeding status, and self-reported physical activity during pregnancy and after delivery.

Experimental protocol. Volunteers visited the laboratory on two separate occasions (at least 24 h apart) at the same time of day. Volunteers were asked to abstain from heavy exercise for 24 h and caffeine for 12 h before visiting the laboratory. On the first visit to the laboratory, anthropometric characteristics (height, body mass, and body mass index [BMI]) were determined. First, maximal voluntary contraction of the left forearm was determined through two 1-s maximal voluntary contractions, separated by 60-s rest, on a commercially available digital handgrip dynamometer (Grip-A, 5001; Takei Scientific Instruments Co. Ltd., Shinagawa-ku, Tokyo, Japan). Second, peak power output on an upright cycle ergometer was estimated using a submaximal incremental exercise test to 70% heart rate reserve (14). Heart rate reserve was calculated before the start of the test using the Karvonen method (18), in which resting heart rate was measured during 5 min of quiet rest and maximal heart rate was estimated using the equation 208 − 0.7 (age) (19). The test included a 2-min warm up at a set cadence of 50 rpm at 0 W, followed by a ramped cycle protocol with a set cadence of 70 rpm and increments of 20 $W \cdot min^{-1}$ (Lode Ergometry Manager 9.2; Lode B.V., Gronigen, The Netherlands). The test was terminated when participants reached 70% heart rate reserve and the achieved power output at 70% heart rate reserve was extrapolated to heart rate maximum. Peak oxygen consumption and peak power were then estimated. This value was then scaled for reduced cycling performance associated with the supine and left-lateral position (multiplied by 0.7) (20).

In the second visit, cardiovascular assessments were completed at rest, during an afterload challenge mediated by a standardized sustained isometric forearm contraction (30% maximal voluntary contraction) and during low- and moderate-intensity dynamic cycling exercise (25% and 50% estimated supine peak power output; Corival, Lode). Each challenge lasted between 5 and 7 min, during which blood pressure was monitored continuously and after the first 2 min of each challenge, and after the stabilization of heart rate and blood pressure, echocardiographic images were acquired. After the sustained isometric forearm contraction, volunteers had 5 min of recovery, followed by the collection of a second set of echocardiographic data to confirm cardiac function had returned to baseline. Participants then began the low-intensity dynamic cycling exercise, followed by 1–2 min rest, after which, they completed the moderate-intensity exercise bout (order not randomized).

All cardiovascular assessments were completed using a specifically designed stress table that was tilted 30° to the left

(Angio 2003, Lode). This supine, left-lateral position facilitated the collection of echocardiographic images during exercise but also has been shown to prevent inferior vena cava compression by the gravid uterus during gestation (21). All volunteers were instructed to breathe freely throughout each challenge.

Blood pressure. Resting systolic blood pressure (SBP) and diastolic blood pressure (DBP) were assessed via the auscultatory method using a stethoscope (Classic III, Littman; 3M Health Care, St. Paul, MN) and a sphygmomanometer (DuraShock DS54 Sphygmomanometer; Welch Allyn, Skaneateles Falls, NY) placed on the bare upper left arm after 5 min of seated rest. Two measurements were taken in line with the recommendations of the AHA (22) and averaged. Brachial SBP and DBP were then measured continuously using finger photoplethysmography (FinometerPRO, FMS; Finapres Measurement Systems, Arnhem, Netherlands; calibrated according to manufacturer's instructions) and recorded for later offline analysis (PowerLab; ADInstruments, Chalgrove, UK). The measured auscultatory value was used to confirm or calibrate the baseline signal, following which average values were calculated from 20 continuous waveforms (cardiac cycles) within the final 2 min of each procedure (LabChart v7, ADInstruments). Mean arterial pressure (MAP) was calculated as 1/3 SBP + 2/3 DBP.

Echocardiography. A trained research sonographer (VLM) acquired echocardiographic images using a commercially available ultrasound system and a 1.5- to 4.6-MHz phased array transducer (Vivid E9 and MS5; GE Medical Systems, Horten, Norway). Echocardiographic images were collected to allow the assessment of LV structure and function in accordance with current guidelines (23). Detailed methods have been described fully elsewhere (24). Briefly, and specific to the outcomes of this study, two-dimensional parasternal short-axis views at the base and apex as well as apical four- and two-chamber views were collected. Septal tissue velocities at the mitral annulus were also measured using Tissue Doppler Imaging. For all images, five consecutive cardiac cycles were recorded at end expiration. Data were stored for later offline analysis (EchoPAC PC Version 112.1.0, GE Medical). Measurements were made in triplicate from different cardiac cycles and the average values used for statistical analyses. A three-lead electrocardiograph was used to monitor heart rate.

Stroke volume, end-diastolic volume (EDV), and end-systolic volume (ESV) were derived via the Simpson's biplane method. Cardiac output was calculated as the product of heart rate and stroke volume. To allow for comparisons between groups, data were scaled allometrically to height using previously published or predicted exponents: cardiac output (1.83), stroke volume (2.04) (25), EDV, and ESV (2.00 as determined by the theory of dimensionality) (26). SVR was calculated as MAP/cardiac output. The coefficients of variation for measurement of cardiac output, stroke volume, and EDV during low-intensity supine cycling exercise were 3.9%, 3.2%, and 2.7%, respectively. Ejection fraction was calculated as (stroke volume / EDV) × 100. Peak systolic (S'), early (E'), and late (A') diastolic septal velocities were measured and scaled to LV length (27).

Speckle-tracking echocardiography was used to measure LV mechanics, specifically global longitudinal strain, basal and apical circumferential strain, and basal and apical rotation. Longitudinal strain was measured in the apical four-chamber view. Apical and basal circumferential strain and rotation were derived from parasternal short-axis apical and basal views. Twist was calculated as the instantaneous difference between basal and apical rotation and was divided by LV length to calculate torsion. Strain and rotation curves were transformed using software that applied a cubic spline interpolation (2D Strain Analysis Tool, Stuttgart, Germany) to enable time alignment of data, allowing for inter- and intraindividual variability in heart rate and frame rate at image acquisition. Peak and time to peak (%) strain, rotation, and twist were calculated, defined as the maximal value across the cardiac cycle. Systolic strain/twist velocity and diastolic strain/untwisting velocity (the recoil of the left ventricle during myocardial relaxation indicated by the most pronounced velocity during early diastole) were also measured. The coefficients of variation for measurement of global longitudinal strain, basal, and apical circumferential strain and twist during low-intensity supine cycling exercise were 8.5%, 12.9%, 9.2%, and 20.0%, respectively.

Statistical analyses. All data are presented as mean ± SD. All statistical analyses were conducted using SPSS Statistics (Version 20.0; IBM Corporation, Chicago, IL). Graphical representations of data were produced using GraphPad Prism (Version 7 for Mac; GraphPad software, San Diego, CA). Statistical significance was accepted at $P < 0.05$. Differences between continuous demographic characteristics of participants were determined using ANOVA after confirmation that all assumptions of the method were met (28). Independent t-tests were used to determine differences between pregnant and postpartum women for data only relevant to pregnancy (e.g., gestational age at delivery) (28). Significant differences in the incidence of pregnancy outcomes (dichotomous variables) ($\alpha = 0.05$) were determined between pregnant and postpartum groups using the test of two proportions. The effect of each challenge on hemodynamic variables in all groups was determined (within-group comparison) using paired sample t-tests (baseline vs during sustained isometric forearm contraction) and ANOVA (baseline vs low-intensity vs moderate-intensity cycling exercise) Effect size, specifically partial eta squared (η_p^2), was determined where possible within analyses.

To determine differences between groups, while accounting for differences at baseline, ANCOVA was used for comparisons of responses to the sustained isometric forearm contraction. The resting value of each parameter was included as a covariate in all analyses. Furthermore, nonpregnant women were significantly younger than pregnant and postpartum women ($P < 0.001$; Table 1). Consequently, age was also included as a covariate in all analyses. Assumptions of ANCOVA were checked (28): linear relationships and homogeneity of regression slopes between parameters were assessed by visual inspection of scatterplots. Standardized residuals for groups and the overall model were assessed by Shapiro–Wilk's test

TABLE 1. Participant demographics.

	Nonpregnant	Pregnant	Postpartum	P	η_p^2
N	18	14	13		
Age (yr)	28 ± 4**	32 ± 3	33 ± 2	**<0.001**	0.360
Body mass (kg)	64 ± 13	73 ± 8	67 ± 11	0.07	0.122
Height (cm)	166 ± 7	167 ± 4	166 ± 4	0.78	0.012
BMI ($kg \cdot m^{-2}$)	23 ± 4	26 ± 4	23 ± 4	0.07	0.118
SBP (mm Hg)	113 ± 7	109 ± 8	105 ± 6	0.12	0.098
DBP (mm Hg)	68 ± 6	63 ± 5	61 ± 4*	0.036	0.149
MAP (mm Hg)	83 ± 6	79 ± 6	75 ± 4*	0.034	0.152
Birthweight (kg)[a]	NA	3.46 ± 0.47	3.14 ± 0.50	0.10	0.107
Gestational age at delivery (wk)[a]	NA	40.3 ± 2.2	39.7 ± 1.5	0.39	0.031
Maximal voluntary contraction of the forearm (kg)	29.5 ± 4.7	26.9 ± 4.5	26.8 ± 3.0	0.13	0.093
Resistance during isometric forearm contraction (kg)	8.9 ± 1.4	8.0 ± 1.4	7.9 ± 1.0	0.08	0.113
Estimated $\dot{V}O_{2peak}$ ($mL \cdot kg^{-1} \cdot min^{-1}$)	33 ± 8	29 ± 6	28 ± 7	0.23	0.068
Estimated supine peak PO (W)	157 ± 28	150 ± 24	136 ± 22	0.08	0.111
Low-intensity PO (W)	39 ± 7	38 ± 6	34 ± 7	0.08	0.113
Moderate-intensity PO (W)	79 ± 14	75 ± 12	68 ± 11	0.08	0.113

Data are presented as mean ± SD. Differences between groups are identified using ANOVA for all parameters.
Bold value indicates significant *P* value.
*$P < 0.05$ vs nonpregnant.
**$P < 0.05$ vs pregnant and postpartum.
[a]Statistical analysis completed using independent *t*-tests.
NA, not applicable; $\dot{V}O_{2peak}$, peak oxygen consumption; PO, power output.

($P > 0.05$) for normal distribution. Homoscedasticity and homogeneity of variance were assessed by visual inspection of a scatterplot and Levene's test of homogeneity of variance, respectively. If statistical significance between groups was identified, *post hoc* analyses were performed with a Bonferroni adjustment.

Similarly, statistical differences between groups to low- and moderate-intensity dynamic cycling exercise were identified using a repeated-measures general linear model, in which the resting value was used as a covariate. *P* values were determined for between-subjects (group) and within-subjects (exercise intensity) differences and the group–exercise (Grp–Ex) interaction. Assumptions of GLM were checked (29). Mauchly's test of sphericity was used to check the sphericity on the variance–covariance matrix of the dependent variables. No epsilon adjustments were required. Homogeneity of variance–covariance matrices was tested with the Box's M ($P > 0.05$). *Post hoc* analyses of the general linear model were performed using a Bonferroni adjustment.

RESULTS

Volunteer characteristics. Pregnant and postpartum women attended the laboratory within the predefined periods (25.4 ± 0.6 wk gestation and 15.1 ± 1.3 wk after delivery, respectively). Pregnancy outcomes were not significantly different between pregnant and postpartum women (Table 1), and the distribution of infant sex (7 male infants in each group; $P = 0.842$) and the methods of delivery (9 and 10 vaginal deliveries, respectively; $P = 0.658$) were similar between groups. The majority of women in both groups breastfed for at least 6 wk (pregnant, $n = 11$ [78%]; postpartum, $n = 13$ [100%]; $P = 0.077$). In the nonpregnant group, nine women were using combined oral contraceptives and nine women had regular cycles. In the postpartum group, 12 women were not using contraception but were not cycling because of breastfeeding, and one woman had an implant placed after delivery. We do not believe that hormonal birth control/menstrual cycle phase would have affected the comparisons made in this study. Previous work (30,31) has not shown differences in cardiovascular or LV measurements across the menstrual cycle or as a result of hormonal contraception.

During gestation, 9 women (64%) in the pregnant group and 8 women (62%) in the postpartum group reported meeting physical activity guidelines on a weekly basis; 4 women (29%) in the pregnant group and 5 women (38%) in the postpartum group reported "sometimes" meeting physical activity guidelines; and 1 woman (7%) in the pregnant group reported not meeting physical activity guidelines ($P = 0.567$). Self-reported prenatal exercise included walking, running, swimming, cycling, prenatal classes, resistance training, Pilates, yoga, tennis, boxing, ballet, aerobics, and dancing. All women reported being physically active during the postpartum period.

Anthropometric characteristics were not significantly different between groups (Table 1). Resting DBP and MAP were significantly lower in postpartum versus nonpregnant women ($P = 0.036$ and 0.030, respectively), but SBP was not significantly different between groups (Table 1). Resting cardiac structure and function data from the three groups have been published previously (24). Briefly, pregnant women had significantly higher resting cardiac output compared with nonpregnant and postpartum women (pregnant, 1.8 ± 0.3 $L \cdot min^{-1} \cdot m^{-1.83}$; nonpregnant, 1.3 ± 0.2 $L \cdot min^{-1} \cdot m^{-1.83}$; postpartum, 1.3 ± 0.3 $L \cdot min^{-1} \cdot m^{-1.83}$; $P = <0.001$). This was a consequence of significantly greater heart rate (pregnant, 69 ± 7 bpm; nonpregnant, 57 ± 8 bpm; postpartum, 57 ± 10 bpm; $P = <0.001$) and stroke volume (pregnant, 24.5 ± 3.1 $mL \cdot m^{-2.04}$; nonpregnant, 20.2 ± 2.1 $mL \cdot m^{-2.04}$; postpartum, 22.1 ± 3.8 $mL \cdot m^{-2.04}$; $P = <0.001$) compared with nonpregnant and postpartum females. With regard to LV mechanics, pregnant women had significantly greater resting longitudinal and basal circumferential strain compared with nonpregnant females (−22% ± 2% vs −17% ± 3%, $P = 0.002$, and −23% ± 4% vs −16% ± 2%, $P = 0.001$, respectively), but no differences were observed for apical circumferential strain or LV twist/torsion.

There were no significant differences between groups in maximal voluntary contraction or estimated supine peak power output; therefore, the absolute intensity of the sustained isometric forearm contraction and the exercise challenges were similar between groups (Table 1). All volunteers successfully completed the sustained isometric forearm contraction; however, one nonpregnant female (discomfort from unrelated injury) and two postpartum women (required to breastfeed) did not complete the moderate-intensity dynamic cycling exercise challenge. It was not possible to monitor blood pressure in one nonpregnant female during the test protocol because of poor signal. Some echocardiographic images collected during exercise were not analyzed because of inadequate image quality

for speckle-tracking echocardiography. The *n* for each group is presented in each table in the interest of clarity.

Hemodynamic responses to sustained isometric forearm contraction. In the total cohort, the sustained isometric forearm contraction significantly increased SBP (124 ± 13 vs rest, 115 ± 11 mm Hg; $P < 0.001$), DBP (71 ± 11 vs rest, 64 ± 8 mm Hg; $P < 0.001$), MAP (89 ± 11 vs rest, 81 ± 8 mm Hg; $P < 0.001$), heart rate (62 ± 10 vs rest, 60 ± 10 bpm; $P = 0.013$), SVR (1933 ± 489 vs rest, 1862 ± 61 dyn·s^{-1}·cm^{-6}; $P < 0.001$), and cardiac output (4.0 ± 1.1 vs rest, 3.70 L·min^{-1}; $P = 0.005$) above baseline values; however, stroke volume was unchanged (rest, 62 ± 9, vs isometric forearm contraction, 64 ± 12 mL; $P = 0.072$).

There were no significant differences between groups in blood pressure or stroke volume in response to this challenge (Table 2, and see Table, Supplemental Digital Content 1, Absolute left ventricular volumes during an isolated afterload challenge, http://links.lww.com/MSS/C195). However, when accounting for differences at rest, pregnant women had significantly greater cardiac output ($P = 0.021$; Fig. 1) and significantly lower SVR ($P = 0.028$) during the sustained isometric forearm contraction compared with nonpregnant women. Despite a significant difference observed in the overall effect of heart rate responses during sustained isometric forearm contraction, there was no statistical interaction between groups *post hoc*. There were also no significant differences in traditional echocardiographic measures of systolic (ejection fraction and indexed *S*′) or diastolic (indexed *E*′ and *A*′) parameters between groups in response to sustained isometric forearm contraction (Table 2).

TABLE 2. Cardiovascular function during an afterload challenge, mediated by a sustained isometric forearm contraction at 30% maximal voluntary contraction, in nonpregnant, pregnant (22–26 wk gestation), and postpartum (12–14 wk after delivery) women.

	Nonpregnant	Pregnant	Postpartum	*P*	η_p^2
N	18	14	13		
Heart rate (bpm)	58 ± 9	71 ± 7	57 ± 9	**<0.001**	0.841
SBP (mm Hg)[a]	125 ± 10	124 ± 15	124 ± 14	0.284	0.118
DBP (mm Hg)[a]	75 ± 11	71 ± 9	67 ± 12	0.275	0.120
MAP (mm Hg)[a]	91 ± 10	88 ± 10	86 ± 12	0.511	0.079
Cardiac output (L·min^{-1}·m$^{-1.83}$)	1.3 ± 0.2	2.0 ± 0.3 *	1.5 ± 0.4	**0.022**	0.202
Stroke volume (mL·m$^{-2.04}$)	20 ± 3	25 ± 3	23 ± 6	0.175	0.098
EDV (mL·m^{-2})	37 ± 5	42 ± 5	36 ± 4	0.097	0.113
ESV (mL·m^{-2})	16 ± 3	16 ± 3	13 ± 3	0.252	0.068
SVR (dyn·s^{-1}·cm^{-6})[a]	2252 ± 505	1404 ± 262 *	1993 ± 370	**<0.001**	0.507
Ejection fraction (%)	57 ± 6	61 ± 5	62 ± 7	0.097	0.119
Indexed *S*′ (m·s^{-1}·cm)[b]	0.010 ± 0.001	0.010 ± 0.002	0.010 ± 0.002	0.966	0.015
Indexed *E*′ (m·s^{-1}·cm)[b]	0.017 ± 0.003	0.016 ± 0.003	0.016 ± 0.003	0.795	0.013
Indexed *A*′ (m·s^{-1}·cm)[b]	0.008 ± 0.002	0.009 ± 0.002	0.008 ± 0.001	0.430	0.047
Global longitudinal strain (*n*)	15	11	10		
Peak strain (%)	−16.0 ± 3.3	−21.4 ± 3.4	−18.7 ± 1.6	**<0.001**	0.468
Basal circumferential strain (*n*)	17	14	13		
Peak strain (%)	−14.6 ± 1.4	−23.5 ± 1.2 *	−20.7 ± 1.5	**<0.001**	0.518
Apical circumferential strain (*n*)	15	14	13		
Peak strain (%)	−20 ± 6	−24 ± 5	−22 ± 4	**0.032**	0.025
Rotational mechanics (*n*)	15	14	13		
Twist (°)	14 ± 4	16 ± 6	17 ± 6	0.172	0.155
Torsion (°)	1.7 ± 0.5	1.8 ± 0.8	2.0 ± 0.8	0.219	0.140
Untwisting velocity (°·s^{-1})	−104 ± 9	−104 ± 9	−103 ± 10	**0.045**	0.188

Data are presented as mean ± SD.
Statistical values (*P* and partial eta squared [η_p^2]) were determined using ANCOCA (resting value and age as covariate).
Tissue velocities indexed to LV length.
Resting data have been presented elsewhere previously (18).
Bold values indicate significant *P* value.
* $P < 0.05$ vs nonpregnant.
[a]Analyses completed on $n = 17$ nonpregnant women.
[b]Analyses completed on $n = 16$ nonpregnant, $n = 12$ pregnant, and $n = 12$ postpartum women.
S′, systolic tissue velocity; *E*′, early systolic tissue velocity; *A*′, late-diastolic tissue velocity.

LV mechanics in response to sustained isometric forearm contraction. Within-group comparisons revealed that the sustained isometric forearm contraction did not significantly change longitudinal strain (−18.2% ± 3.8% vs rest, −19.1% ± 3.4%; $P = 0.193$), basal circumferential strain (−19.2% ± 5.9% vs rest, −20.6% ± 5.4%; $P = 0.092$), apical circumferential strain (−21.6% ± 5.4% vs rest, −22.0% ± 4.3%; $P = 0.794$), or LV torsion (1.9°·cm^{-1} ± 0.7°·cm^{-1} vs rest, 1.8°·cm^{-1} ± 0.5°·cm^{-1}; $P = 0.092$) from resting values.

Similar to resting data (24), between-group comparisons showed that pregnant women had significantly greater basal circumferential strain ($P < 0.001$, Table 2 and Fig. 2) compared with nonpregnant women during the sustained isometric forearm contraction. There were no significant differences between groups in other strain parameters, LV twist, or torsion; however, there was a significant between-group effect in untwisting velocity, but no *post hoc* statistical interactions were identified (Table 2, and see Table, Supplemental Digital Content 1, Systolic and diastolic strain rates in longitudinal and circumferential strain during an isolated afterload challenge, http://links.lww.com/MSS/C195).

Hemodynamic responses to low- and moderate-intensity dynamic cycling exercise. Within groups, submaximal dynamic cycling exercise increased SBP (rest, 115 ± 11 mm Hg; low, 139 ± 16 mm Hg; moderate, 155 ± 18 mm Hg; $P < 0.001$), DBP (rest, 65 ± 8 mm Hg; low, 76 ± 10 mm Hg; moderate, 81 ± 12 mm Hg; $P < 0.001$), and MAP (rest, 81 ± 8 mm Hg; low, 101 ± 11 mm Hg; moderate, 111 ± 14 mm Hg; $P < 0.001$). These changes in blood pressure are similar to those observed in young, healthy females completing experimental trials using the same exercise modality at similar intensities (32). In addition, heart rate (rest, 60 ± 10 bpm; low, 91 ± 9 bpm; moderate, 111 ± 9 bpm; $P < 0.001$), stroke volume (rest, 61 ± 9 mL; low, 69 ± 10 mL; moderate, 73 ± 12 mL; $P < 0.001$), and cardiac output (rest, 3.7 ± 0.9 L·min^{-1}; low, 6.3 ± 1.1 L·min^{-1}; moderate, 8.1 ± 1.3 L·min^{-1}; $P < 0.001$) were increased, whereas SVR was decreased (rest, 1877 ± 410 dyn·s^{-1}·cm^{-6}; low, 1283 ± 262 dyn·s^{-1}·cm^{-6}; moderate, 1123 ± 202 dyn·s^{-1}·cm^{-6}; $P < 0.001$) from baseline in all groups.

Between-group comparisons showed no differences in heart rate, blood pressure, scaled stroke volume, scaled cardiac output (Fig. 1 and see Table, Supplemental Digital Content 2, Absolute left ventricular volumes during low- and moderate-intensity aerobic cycling, http://links.lww.com/MSS/C196), ejection

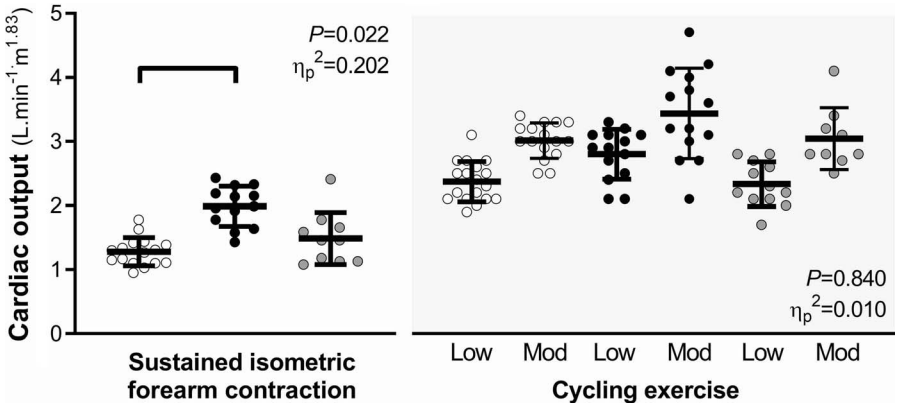

FIGURE 1—When accounting for differences between groups at rest, healthy pregnant women (*black circles*) in the late second trimester had greater cardiac output during a sustained isometric forearm contraction at 30% maximum compared with nonpregnant women (*white circles*), but similar responses to both nonpregnant and postpartum women (gray circles) during low- and moderate-intensity dynamic cycling exercise (25% and 50% estimated peak power output). Cardiac output was allometrically scaled to height using a previously published exponent (25). Statistical values (P and partial eta squared [η_p^2]) were identified using an ANCOVA for sustained isometric forearm contraction data and a general linear model for cycling exercise data (with resting value and age as covariate). Main effect for between-group comparisons reported only. Capped line indicates significant difference ($P < 0.05$) between pregnant and nonpregnant women identified in *post hoc* comparisons. Resting data have been presented elsewhere previously (24).

fraction, or SVR in response to low- and moderate-intensity dynamic cycling exercise, when accounting for resting function (Table 3). However, pregnant women had significantly greater scaled EDV during dynamic cycling exercise compared with nonpregnant women ($P = 0.020$) and significantly greater indexed S' compared with postpartum women ($P = 0.002$; Table 3). There was a significant main effect of group in both indexed E' and indexed A' in response to low- and moderate-intensity dynamic cycling exercise; however, *post hoc* differences were not identified between groups (Table 3).

LV mechanics in response to low- and moderate-intensity dynamic cycling exercise. Within groups, peak global longitudinal strain (rest, −19.1% ± 3.4%; low, −20.3% ± 2.3%; moderate, −20.9% ± 4.8%; $P = 0.331$) and peak basal circumferential strain (rest, −20.6% ± 5.4%; low, −21.0% ± 7.7%; moderate, −22.1% ± 7.9%; $P = 0.251$) were not significantly different in response to low- to moderate-intensity cycling; however, peak apical circumferential strain (rest, −22.0% ± 4.3%; low, −24.4% ± 5.4%; moderate, −27.2% ± 6.9%; $P = 0.042$) and LV torsion (rest, 1.8°·cm^{-1} ± 0.5°·cm^{-1}; low, 2.4°·cm^{-1} ± 0.8°·cm^{-1}; moderate, 2.9°·cm^{-1} ± 0.9°·cm^{-1}; $P = 0.003$) were significantly increased above resting values.

In support of findings at rest (24) and during the sustained isometric forearm contraction, pregnant women had significantly greater basal circumferential strain ($P = 0.012$, Fig. 2) compared with nonpregnant women during low- and moderate-intensity cycling; however, there were no other statistical differences in peak mechanics parameters between groups (Table 3 and see Table, Supplemental Digital Content 2, Systolic and diastolic strain rates in longitudinal and circumferential strain during low- and moderate-intensity aerobic cycling, http://links.lww.com/MSS/C196).

DISCUSSION

The aim of this study was to determine the cardiac responses to acute increases in afterload and to dynamic, submaximal exercise in healthy nonpregnant, pregnant, and postpartum women. In support of our hypothesis, pregnant women had a similar cardiovascular response to these acute challenges, supporting that the maternal heart is able to accommodate for additional metabolic and myocardial oxygen demands and the altered hemodynamics associated with moderate-intensity exercise. This study provides empirical

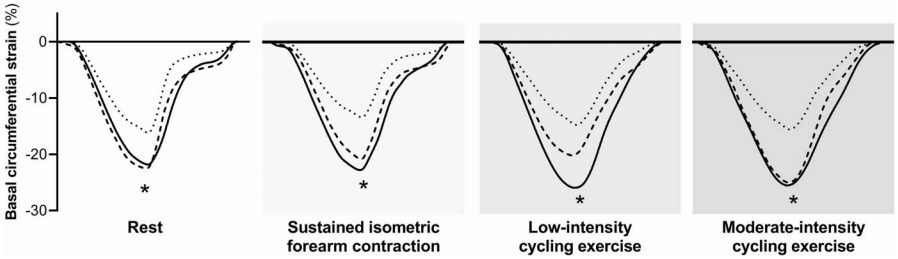

FIGURE 2—Graphical representation of basal circumferential strain at rest, during a sustained isometric forearm contraction at 30% maximum, and during low- and moderate-intensity dynamic cycling exercise (25% and 50% estimated peak power output) in nonpregnant (*dotted lines*), pregnant (*solid lines*), and postpartum women (*dashed lines*). Strain curves were transformed using a cubic spline interpolation to enable time alignment of data across the cardiac cycle and averaged for each group. Resting data have been presented elsewhere previously (24). *Significant difference ($P < 0.05$) between pregnant and nonpregnant women identified in *post hoc* comparisons.

TABLE 3. Cardiovascular responses during low- and moderate-intensity dynamic cycling exercise (25% and 50% estimated peak power output) in nonpregnant, pregnant (22–26 wk gestation), and postpartum (12–14 wk after delivery) women.

	Intensity	Nonpregnant	Pregnant	Postpartum	*P*	η_p^2
	n	17	14	13		
Heart rate (bpm)	Low	90 ± 10	97 ± 6	87 ± 10	0.486	0.043
	Mod	110 ± 10	114 ± 5	106 ± 10		
SBP (mm Hg)[a]	Low	138 ± 10	140 ± 22	138 ± 15	0.526	0.036
	Mod	151 ± 16	157 ± 19	155 ± 19		
DBP (mm Hg)[a]	Low	79 ± 9	75 ± 11	73 ± 10	0.627	0.026
	Mod	81 ± 13	81 ± 13	79 ± 10		
MAP (mm Hg)[a]	Low	102 ± 10	101 ± 12	99 ± 13	0.895	0.006
	Mod	111 ± 16	113 ± 13	109 ± 12		
Cardiac output ($L \cdot min^{-1} \cdot m^{-1.83}$)	Low	2.3 ± 0.3	2.8 ± 0.4	2.4 ± 0.4	0.840	0.010
	Mod	3.0 ± 0.4	3.4 ± 0.7	3.0 ± 0.3		
Stroke volume ($mL \cdot m^{-2.04}$)	Low	23 ± 2	26 ± 4	25 ± 4	0.830	0.011
	Mod	25 ± 3	27 ± 5	26 ± 4		
EDV ($mL \cdot m^{-2}$)	Low	36 ± 4	42 ± 5*	37 ± 3	**0.019**	0.207
	Mod	36 ± 4	42 ± 4	37 ± 4		
ESV ($mL \cdot m^{-2}$)	Low	12 ± 3	15 ± 4	12 ± 2	0.118	0.118
	Mod	11 ± 3	14 ± 4	10 ± 3		
SVR ($dyn \cdot s^{-1} \cdot cm^{-6}$)[a]	Low	1400 ± 248	1155 ± 220	1323 ± 292	0.921	0.005
	Mod	1177 ± 189	1069 ± 237	1147 ± 227		
Ejection fraction (%)	Low	67 ± 6	64 ± 7	68 ± 6	0.330	0.063
	Mod	70 ± 5	68 ± 8	72 ± 8		
Indexed *S*′ ($m \cdot s^{-1} \cdot cm^{-1}$)	Low	0.011 ± 0.001	0.012 ± 0.002	0.010 ± 0.002		
	Mod	0.014 ± 0.002	0.015 ± 0.002**	0.012 ± 0.001	**<0.001**	0.584
Indexed *E*′ ($m \cdot s^{-1} \cdot cm^{-1}$)	Low	0.016 ± 0.006	0.017 ± 0.003	0.017 ± 0.004	**0.002**	0.283
	Mod	0.019 ± 0.005	0.019 ± 0.002	0.019 ± 0.004		
Indexed *A*′ ($m \cdot s^{-1} \cdot cm^{-1}$)	Low	0.012 ± 0.002	0.011 ± 0.002	0.010 ± 0.002	**<0.001**	0.519
	Mod	0.014 ± 0.002	0.010 ± 0.005	0.012 ± 0.002		
Global longitudinal strain	*n*	13	11	7		
Peak strain (%)	Low	−21.0 ± 2.2	−21.3 ± 2.3	−17.9 ± 0.9	**0.038**	0.216
	Mod	−19.8 ± 5.7	−23.9 ± 3.1	−19.4 ± 3.8		
Basal circumferential strain	*n*	15	13	11		
Peak strain (%)	Low	−15.8 ± 4.5	−27.0 ± 4.9*	−22.5 ± 9.1	**0.007**	0.247
	Mod	−15.2 ± 6.7	−27.4 ± 4.6	−25.1 ± 6.1		
Apical circumferential strain	*n*	14	13	11		
Peak strain (%)	Low	−25.1 ± 5.4	−26.4 ± 5.8	−23.5 ± 4.3	0.476	0.043
	Mod	−30.0 ± 8.6	−25.4 ± 5.9	−26.1 ± 4.9		
Rotational mechanics	*n*	13	13	11		
Twist (°)	Low	21.3 ± 5.1	21.9 ± 7.3	20.4 ± 7.1	0.788	0.014
	Mod	25.0 ± 7.4	26.3 ± 11.1	23.6 ± 8.1		
Torsion ($° \cdot cm^{-1}$)	Low	2.6 ± 0.7	2.4 ± 0.8	2.5 ± 0.8	0.948	0.003
	Mod	3.0 ± 0.8	2.9 ± 1.2	2.9 ± 0.9		
Untwisting velocity ($° \cdot s^{-1}$)	Low	−167 ± 47	−155 ± 49	−146 ± 42	0.123	0.118
	Mod	−252 ± 72	−217 ± 88	−171 ± 63		

Data are presented as mean ± SD.
Statistical values (*P* and partial eta squared [η_p^2]) were identified using a general linear model (with resting value and age as covariate) and reported for between-group comparisons only. There were no significant group–exercise interactions. Within-group comparisons are detailed within the Results section.
Resting data have been presented elsewhere previously (18).
Bold values indicate significant *P* value.
*$P < 0.05$ vs nonpregnant.
**$P < 0.005$ vs postpartum.
[a]Analyses completed on $n = 16$ nonpregnant women and 9 postpartum women.
Mod, moderate; *S*′, systolic tissue velocity; *E*′, early systolic tissue velocity; *A*′, late-diastolic tissue velocity.

data that reinforces the safety of acute prenatal exercise within recommended guidelines in healthy pregnant women.

Hemodynamic responses to physiological challenges. Cardiac output and hemodynamics, such as heart rate, stroke volume, and blood pressure, are useful in understanding the fundamental changes to the cardiovascular system in response to prenatal exercise. Although unsubstantiated in the literature, previous concerns of exercise in pregnancy arose from the potential of an already overloaded heart not being able to augment cardiac output, consequently resulting in reduced uteroplacental blood flow (3). By contrast, this study shows that cardiac output does rise adequately in pregnant women during physiological challenges as nonpregnant women, even when accounting for differences at rest. This finding is in agreement with previous research showing that healthy pregnant women have similar hemodynamic responses to exercise as nonpregnant women (33–35) and that there are no adverse impacts of activity on metrics of fetal well-being (36), although these were not directly measured in this study. Our data extend previous observations of traditional hemodynamics through the addition of comprehensive measurements of cardiac function. Our data suggest that in healthy pregnant women, despite significant changes to resting cardiac structure and function, cardiovascular responses to submaximal dynamic cycling exercise are proportionate to the level of exertion (i.e., pregnant, nonpregnant, and postpartum women have the same increase in cardiac output). As such, the findings from this study help to reinforce the safety of acute prenatal exercise in healthy pregnant women within

recommended guidelines (4,5). Furthermore, an understanding of what constitutes a "normal" response to physical challenges during pregnancy allows future examination of the hypothesis that abnormal responses could be indicative of maternal maladaptation and the potential development of later complications such as preeclampsia (14).

Interestingly, pregnant women had significantly greater cardiac output during submaximal isometric contraction when compared with nonpregnant women. This was likely as a result of lower SVR during this challenge in the pregnant group, despite similar changes in blood pressure and LV volumes. Sustained isometric contraction results in sympathetic activation as a result of the mechano- and metaboreflexes. Consequently, increases in blood pressure, heart rate, and therefore cardiac output are observed in response to isometric handgrip in nonpregnant and pregnant populations (37,38). Recently, a blunted neurovascular transduction (or dissociation between sympathetic outflow and vascular outcome) in response to vasoconstrictive stimuli, such as the cold pressor test, has been shown in pregnant women (39). Speculatively, when compared with the nonpregnant participants, a blunted neurovascular transduction in pregnant individuals may have contributed to a lower cardiac afterload during sustained isometric contraction, thus allowing for greater cardiac output and LV strain. Future work is required to determine the specific contributors to the generation of cardiac output during an afterload challenge in pregnancy. In contrast to this observation during submaximal isometric contraction, cardiac output in response to dynamic exercise was similar between nonpregnant and pregnant women. We speculate that the increased cardiovascular demand, the greater oxygen consumption, and the metabolite accumulation during dynamic aerobic exercise attenuate the influence of systemic pregnancy adaptations—such as reduced neurovascular transduction. As such, further investigations are warranted during other modalities, such as isotonic exercise, to determine how differences in cardiovascular demand and metabolic requirements influence cardiac responses to physiologic stimuli during pregnancy.

LV mechanics in pregnancy. In the late second trimester of healthy pregnancy, global cardiac function is underpinned by greater LV strain at rest and during physiological challenges. As shown in the same cohort studied here (24), both global longitudinal strain and basal circumferential strain were significantly higher at rest in pregnant compared with nonpregnant women. The current study furthers our understanding by showing that in response to either a sustained isometric forearm contraction or a submaximal dynamic cycling exercise, basal circumferential strain remains significantly greater in pregnant women when compared with nonpregnant women. It should be noted that these observations were made without differences in traditional echocardiographic measures reinforcing the potential sensitivity of the parameters of LV mechanics to subtle changes in cardiac function. Furthermore, the measurement of peak global longitudinal strain in this study provides some support that global myocardial deformation may be greater in pregnant women; however, the *post hoc* comparisons were not adequately powered to confirm this.

LV mechanics are modulated by the interaction between the ventricular structure and the prevailing hemodynamic load, with increases in preload and contractility as well as reductions in afterload contributing to greater strain and twist (40). In this study, pregnant women had significantly greater basal circumferential strain during both the sustained isometric forearm contraction and during submaximal dynamic cycling exercise. This may, in some part, be related to greater cardiac preload of pregnancy. At rest, scaled EDV was significantly higher in pregnant women (24) and remained larger during submaximal cycling, thus leading to a greater LV stretch and greater contractility via the Frank–Starling mechanism. Previous research has shown that healthy nonpregnant women rely more heavily on augmenting EDV to facilitate an increase in stroke volume during exercise when compared with men (32). As such, the significantly larger scaled EDV observed in pregnant women during submaximal exercise may be partly responsible for the greater basal circumferential strain compared with nonpregnant and pregnant women. However, the greater preload of pregnancy cannot explain the differences in basal deformation alone. Previous analyses of cardiac mechanics at rest in the same cohort as studied here showed no relationship between LV mechanics parameters and EDV (24). Furthermore, the greater basal circumferential strain was observed during the sustained isometric forearm contraction in pregnant women, without a proportionate change in EDV. Finally, the differences observed in regional circumferential strain (at the LV base) were not mirrored by LV twist or longitudinal strain, which represent heart muscle deformation of the whole LV. It is therefore likely that factors outside hemodynamic load contribute to increased basal circumferential strain at rest and during prenatal exercise in pregnant women. Speculatively, the significantly greater basal circumferential strain observed in pregnant women may be the result of the greater inotropy via sympathetic activation (41,42) and higher circulating estradiol associated with pregnancy (43). In an animal model, estradiol has been shown to have a direct effect on contractility of basal, but not apical, myocytes via enhanced intracellular calcium ion release (44). In humans, basal mechanics in response to physiological challenges have been shown to be higher in premenopausal women compared with postmenopausal women (45). Our findings may therefore represent a region-specific adaptation of the maternal heart to the hormonal milieu of pregnancy, although this relationship requires further investigation.

LV twist and torsion (twist normalized to LV length) are measures that quantify the rotation of the obliquely orientated myofibers of the epicardium and endocardium during ventricular contraction. This wringing motion of the heart facilitates the ejection of stroke volume and is also increasingly recognized as an index of myocardial wall stress distribution (40,46). In this study, LV twist and torsion were significantly increased by a similar amount in all groups in response to submaximal dynamic cycling exercise, supporting previously published data in nonpregnant women using a similar methodology (32). The latter finding is also similar to the observations

made at rest in this cohort (24), where LV twist and torsion were not different between nonpregnant, pregnant, and postpartum women. These findings provide some support that the contribution of rotational mechanics to the generation of stroke volume, the coupling of systolic and diastolic twist mechanics, as well as the distribution of myocardial wall stress in response to physiological challenge may be similar between healthy nonpregnant, pregnant, and postpartum women. It should be considered that the coefficient of variation for the measurement of twist during exercise was relatively high (20.0%) despite standardized image acquisition and analysis by a trained sonographer. Future work should aim to investigate rotational mechanics in more detail and with a larger cohort of pregnant women.

Study considerations. Although our sustained isometric forearm contraction caused a modest but significant rise in blood pressure, the magnitude of response is not comparable with other methods of augmenting afterload. Increases in SBP of between 30 and 35 mm Hg, alongside significant increases in heart rate, have previously been observed during isometric handgrip in healthy populations (males, nonpregnant, and pregnant women) (8,9). By contrast, the method used within this study induced a significant rise in SBP of 9 mm Hg (95% confidence interval, 5 to 14 mm Hg) without a concurrent rise in heart rate. We are therefore confident that the challenge used here caused a meaningful increase in afterload, but our data cannot be extrapolated to greater increases in cardiac afterload.

Second, this cohort included healthy Caucasian women who were physically active, which is not reflective of a general population from either an ethnic or an activity status. In the wider general population, only 15% of pregnant females meet physical activity guidelines according to objective physical activity assessment (47). Within our study, 14 out of 15 pregnant females and all postpartum females reported meeting physical activity guidelines in a self-report questionnaire. It is therefore possible that physiological adaptations associated with regular physical activity may have influenced our findings in these groups. The effects of chronic exercise on cardiac remodeling and function in a healthy adult population are well established (48) and have also been observed in pregnant and postpartum females. Previous research has shown that exercising mothers have significantly lower resting HR and higher heart rate variability compared with inactive mothers (49), supporting the contention that exercise has a chronotropic effect irrespective of gestation. In addition, after delivery, active females have been shown to have greater total blood volume, EDV, and cardiac output in comparison with inactive females, highlighting that exercise-mediated hematological changes affect maternal cardiac function (50,51). Ante- and postnatal exercise may therefore influence cardiac adaptation during and after pregnancy, and there would be value in completing this study again in a wider population of pregnant women.

Third, previous research has demonstrated differences in cardiac mechanics across gestation (52), altered hemodynamic responses to physiological challenges in healthy women across gestation (53), and shown differential responses in women diagnosed with hypertensive complications (54). As such, these results cannot be generalized to women outside this gestational age, nor to women that experience pregnancy complications. However, the data from this study in a well-controlled group of healthy pregnant women provide the first steps to further develop the understanding of whether physiological challenges can be used to predict cardiovascular complications of gestation. As such, future studies should investigate the cardiovascular responses to physiological challenges across gestation and also in pregnant women at risk of or diagnosed with complications where prenatal exercise is not contraindicated.

Finally, this study used a cross-sectional design to enable comparison of nonpregnant, pregnant, and postpartum females. In particular, the comparison between pregnant and postpartum women could have been improved if a longitudinal design was used. Because of time constraints and feasibility, this was not possible. Future work should seek to complete such assessments at preconception, across all trimesters of pregnancy, and into the postpartum period to account for inherent biological variability between individuals.

Conclusions. The results suggest that the maternal heart can appropriately respond to the increased demand, and changes in load, associated with physical activity currently recommended for healthy pregnant women, and therefore support the safety of acute prenatal exercise. However, there are subtle differences in cardiac responses to aerobic and isometric exercise that may in part result from differences in systemic vascular function during pregnancy as well as the cardiac loading of each challenge. In addition, the LV mechanics that underpin global cardiac function, particularly basal circumferential strain, are greater in pregnant women at rest and during exercise, leading us to speculate that the hormonal milieu of pregnancy influences regional LV mechanics in this group, which may be akin to processes that have been suggested in relation to the menopause (45). Furthermore, these data provide an initial understanding of what constitutes a "normal" response to physical challenges during pregnancy. Thus, future work can seek to determine whether abnormal responses may have a prognostic value in identifying the development of later complications in gestation.

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