

# Relation between exercise central haemodynamic response and resting cardiac structure and function in young healthy men

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## Summary

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**Background** Left ventricular (LV) structure and function are predictors of cardiovascular (CV) morbidity and mortality and are related to resting peripheral haemodynamic load in older adults. The central haemodynamic response to exercise may reveal associations with LV structure and function not detected by traditional peripheral (brachial) measures in a younger population.

**Purpose** To examine correlations between acute exercise-induced changes in central artery stiffness and wave reflections and measures of resting LV structure and function.

**Methods** Sixteen healthy men (age  $26 \pm 6$  year; BMI  $25.3 \pm 2.7 \text{ kg m}^{-2}$ ) had measures of central haemodynamic load measured before/after a 30-s Wingate anaerobic test (WAT). Common carotid artery stiffness and reflected wave intensity were assessed via wave intensity analysis as a regional pulse wave velocity (PWV) and negative area (NA), respectively. Resting LV structure (LV mass) and function [midwall fractional shortening (mFS)] were assessed using M-mode echocardiography in the parasternal short-axis view.

**Results** There was a significant association between mFS and WAT-mediated change in carotid systolic BP ( $r = -0.57$ ,  $P = 0.011$ ), logNA ( $r = -0.58$ ,  $P = 0.009$ ) and PWV ( $r = -0.44$ ,  $P = 0.045$ ). There were no significant associations between resting mFS and changes in brachial systolic BP ( $r = -0.26$ ,  $P > 0.05$ ). There were no associations between resting LV mass and changes in any haemodynamic variable ( $P > 0.05$ ).

**Conclusion** Exercise-induced increases in central haemodynamic load reveal associations with lower resting LV function in young healthy men undetected by traditional peripheral haemodynamics.

## Introduction

Reductions in function of the left ventricle (LV) and LV hypertrophy are strong predictors of cardiovascular morbidity and mortality (Levy et al., 1990; Koren et al., 1991; Gardin & Lauer, 2004). Peripheral blood pressure (BP) is a common measure of cardiovascular health, and high peripheral BP is well correlated with cardiac target organ damage and future cardiac events in various clinical populations including older adults and adults with hypertension (Sharman et al., 2015). BP is amplified as it moves away from the heart, and this is most notably affected by artery stiffness and pressure from wave reflections. Because of this amplification, BP in the periphery is not necessarily reflective of the true afterload experienced

by the LV (Chirinos et al., 2010; Zamani et al., 2015), and significant variance exists in central haemodynamic load even among individuals with the same or similar peripheral BP (Sharman et al., 2008). Central BP is the source of resistance that the LV must overcome and as such increases the workload of the LV. Over time, increased LV work contributes to reductions in LV function and hypertrophy of the cardiac muscle. Indeed, central BP is a stronger correlate of future cardiovascular morbidity such as LV hypertrophy (Roman et al., 2010), and mortality than peripheral BP (Pini et al., 2008; Chirinos et al., 2010; Sharman & LaGerche, 2015; Sharman et al., 2015).

Measuring arterial load under resting conditions with peripheral BP does not provide insight into typical day-to-day

haemodynamic stress placed on the central vasculature and LV. In older adults or hypertensive individuals, submaximal exercise as a stressor is a sufficient stimulus to reveal characteristics of the central vasculature that are not observable at rest and thus uncover associations with target organ damage that might go unnoticed if relying on resting measures (Chirinos et al., 2010; Keith et al., 2013). In young, apparently healthy normotensive populations, however, a submaximal stimulus may not be sufficient to unmask otherwise occult associations between haemodynamic load and LV structure/function (Fagard et al., 1995; Cardillo et al., 1996), and this may be related to the nature of the exercise stimulus as well as the method of BP appraisal. While lower intensity aerobic exercise imposes a volume load on the cardiovascular system, higher intensity anaerobic exercise introduces a pressure load. Therefore, when examining associations between exercise-mediated changes in haemodynamic load and cardiac structure/function in younger normotensive, apparently healthy adults, it may be more practical to use an exercise stimulus that 'overloads' rather than 'unloads' the LV.

We have recently shown that 30 s of high-intensity exercise in the form of a Wingate anaerobic test (WAT) results in significantly greater increases in central versus peripheral BP (Babcock et al., 2015). This exercise modality also increases central artery stiffness and pressure from wave reflections (Babcock et al., 2015). The purpose of the current study was to examine the associations between resting left ventricular structure and function and measures of central haemodynamic load, both at rest, and changes produced by ( $\Delta$ ) high-intensity exercise. It was hypothesized that higher exercise-induced increases in central BP would be associated with lower resting cardiac function (appraised as LV fractional shortening) and greater cardiac (LV) mass. It was further hypothesized that exercise-induced increases in central artery stiffness and pressure from wave reflections would also be associated with resting LV function and structure.

## Methods

Sixteen healthy adult men volunteered to participate in this study. According to a self-reported physical activity questionnaire, participants ranged from inactive to recreationally active individuals. Exclusion criteria included history of smoking, cardiovascular disease, renal disease and respiratory disease determined through self-reported health history. The Institutional Review Board of Syracuse University approved this study. All participants provided written informed consent.

## Study design

Participants reported to the Human Performance Laboratory on one occasion. This visit followed a fast of  $\geq 4$  h and abstention of caffeine, alcohol and vigorous exercise for  $\geq 12$  h. Following consent and basic anthropometric measures,

participants assumed a supine position on a cushioned examination bed, and a blood pressure cuff and ECG electrodes (single-lead CM<sub>5</sub> orientation) were applied. Participants laid in the supine position for a period of 10 min in a quiet, dimly lit, temperature-controlled laboratory to ensure blood pressure had stabilized at resting levels. Participants rolled to the left lateral decubitus position and measures of left ventricular structure and function were performed. Participants resumed the supine position, and baseline measures of blood pressure (brachial and carotid) and arterial stiffness were obtained.

After baseline measures, the blood pressure cuff and ECG leads were removed and participants underwent a brief familiarization of exercise testing procedures. Each participant adjusted the seat height and handles on a cycle ergometer to ensure a fit that was comfortable for the participant. Participants were allowed to cycle for a brief time to ensure comfort. They were then given a brief exposure to the resistance/load against which they would pedal and performed two light sprints to become familiar with the Wingate anaerobic test (WAT) starting procedure. They were informed that the addition of the resistance would be sudden 'dropped'. Participants dismounted the cycle ergometer and were then allowed to warm up with 5 min of brisk treadmill walking. Following this warm-up, participants remounted the cycle ergometer and completed a 30-s WAT. After completing the WAT, the participant completed 2 min of active recovery, during which time they pedalled at a self-selected pace against self-selected resistance. Following the recovery period, participants were escorted back to the examination table, and blood pressure and vascular measures were repeated in the same manner as baseline. The immediacy of these postexercise measures was made evident by Nikolic et al. (2014).

## Wingate anaerobic test

Each participant performed a 30-s Wingate anaerobic test (WAT) on an electronically braked bicycle ergometer (Excalibur; Lode B.V., Groningen, the Netherlands). The resistance was set at 7% of body mass. The participant pedalled against zero watts (W) of resistance for 30 s. During the last 10 s of this period, participants were instructed to begin pedalling as rapidly as possible. The resistance was applied electronically and participants pedalled with maximal effort for 30 s while receiving very loud verbal encouragement. Participants remained seated throughout the WAT. Upon completion of the 30-s test, participants were instructed to continue pedalling against light resistance (approximately 80 W) for 2 min prior to being escorted to the examination table for recovery measures.

## Anthropometrics

Height and weight were assessed via a wall-mounted ruler and electronic scale, respectively.

## Brachial blood pressure

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured on the left arm using a validated, automated oscillometric cuff (EW3109; Panasonic Electric Works, Secaucus, NJ, USA). Mean arterial pressure (MAP) was calculated as  $(1/3) \times \text{SBP} + (2/3) \times \text{DBP}$ . Pulse pressure (PP) was calculated as SBP – DBP.

## Carotid blood pressure

Pressure waveforms were obtained in the right carotid artery using applanation tonometry from a 10-s epoch (SphygmoCor; AtCor Medical, Sydney, NSW, Australia). Carotid pressure waveforms were ensemble-averaged to a single waveform and calibrated to brachial MAP and DBP. Pulse pressure amplification was calculated as Brachial PP/Carotid PP.

## Cardiac structure and function

Images of the left ventricle were obtained using echocardiography (ProSound α7; Aloka, Tokyo, Japan) and 2.5- to 5.0-MHz phased-array probe. The participant was placed in left lateral decubitus position, and the heart was imaged in the parasternal short-axis view. Semi-automated callipers were used in M-mode to assess intraventricular septal thickness, posterior wall thickness and internal chamber diameter in both systole and diastole. Left ventricular mass was calculated using the Teichholz formula as follows:

$$\text{LVM} = 1.04 [(IVS_d + LVID_d + LVPWT_d)^3 - LVID_d^3] - 13.6,$$

where IVS<sub>d</sub> is the intraventricular septal wall thickness at end diastole, LVID<sub>d</sub> is the left ventricular internal diameter at end diastole and LVPWT<sub>d</sub> is the left ventricular posterior wall thickness at end diastole. Midwall fractional shortening (mFS) was calculated using the following equation:

$$\text{mFS} = \frac{(LVID_d + H_d/2) - (LVID_s + H_s/2)}{LVID_d + H_d/2}$$

where H<sub>d</sub> and H<sub>s</sub> are calculated as  $(\text{PWT}_d + \text{IVS}_d)/2$  and  $(\text{PWT}_s + \text{IVS}_s)/2$ , respectively.

## Carotid doppler ultrasonography

Longitudinal images of the left common carotid artery (CCA) were obtained using Doppler ultrasound (ProSound α7; Aloka) and 7.5- to 10.0-MHz linear-array probe. Images were acquired 5–10 mm below the carotid bulb. Forward and reflected pressure wave intensity measures were derived from wave intensity analysis (WIA) combined with eTracking software. The distance from the near- to far-wall CCA lumen–intima interface was continuously traced through at least eight cardiac cycles, creating a distension waveform similar to a

pressure waveform (Van Bortel et al., 2001; Niki et al., 2002). These WIA distension waveforms were calibrated using simultaneously measured CCA systolic and diastolic pressures obtained via applanation tonometry, described above. Range-gated colour Doppler signals, averaged along the Doppler beam, were used to measure flow waveforms. Sample volume was manually adjusted to encompass the entire vessel, and an insonation angle  $\leq 60^\circ$  was maintained for all measures.

Wave intensity was calculated using the time derivative of simultaneously obtained blood velocity (U) and pressure (P), where wave intensity =  $(\frac{dp}{dt} \times \frac{dU}{dt})$ . The area under the curve created by this equation represents the energy transfer of the wave (Sugawara et al., 2009). Forward wave intensity ( $W_1$ ) represents a compression wave produced by the contracting LV during early systole as it interacts with the regional vessel. The negative area (NA) occurring immediately after  $W_1$  is a compression wave travelling backwards from the periphery due to the reflected waves. Carotid artery stiffness was also determined from wave intensity analysis using a regional pulse wave velocity (PWV) derived from the water-hammer equation as  $c = (\frac{dp}{dU})/\rho$ , where c represents PWV and ρ represents blood density (assumed to be a constant  $1.055 \text{ kg cm}^{-3}$ ) (Harada et al., 2002). Augmentation index (AIx) was derived from the distension wave as the difference in peak pressure – pressure at the inflection point and expressed relative to total pulse pressure and taken as a measure of global wave reflections.

## Statistical analysis

All data are reported as mean  $\pm$  SD, and statistical significance was established *a priori* as  $P < 0.05$ . All baseline haemodynamic variables were measured in duplicate, and their arithmetic means were used for subsequent analysis. Normality of distribution for variables was assessed qualitatively using histograms and Q-Q plots as well as quantitatively using the Shapiro–Wilk test.  $W_1$  and NA derived from wave intensity analysis were not normally distributed and thus log-transformed to create a normal distribution prior to parametric statistical analysis. Left ventricular mass was expressed relative to body surface area using the equation  $\text{BSA} = \sqrt{w \times h}/3600$ , where w is weight in kilograms and h is height in centimetres (Verbraecken et al., 2006). Values from pre-WAT measures were subtracted from post-WAT values to calculate absolute change scores ( $\Delta$ ), and associations of interest were examined using Pearson's correlation coefficients. Partial correlations were performed to adjust for potential confounders. t-Tests were used to compare baseline values to post-WAT values. All statistical analyses were executed using Statistical Package for the Social Sciences (SPSS, version 21; IBM, Chicago, IL, USA).

## Results

Sixteen men participated in this study. Participant descriptive characteristics and WAT performance variables are presented

**Table 1** Participant Demographics ( $n = 16$ ).

| Variable                                   | Mean $\pm$ SD          |
|--|------------------------|
| Age (years)                                | 26 $\pm$ 6             |
| Height (cm)                                | 180.63 $\pm$ 4.13      |
| Body mass (kg)                             | 82.30 $\pm$ 8.23       |
| Body Mass Index ( $\text{kg m}^{-2}$ )     | 25.25 $\pm$ 2.71       |
| Mean power (W)                             | 581.19 $\pm$ 114.58    |
| Peak power (W)                             | 776.94 $\pm$ 156.17    |
| Time to peak power (s)                     | 4.05 $\pm$ 2.62        |
| Relative mean power ( $\text{W kg}^{-1}$ ) | 7.14 $\pm$ 1.53        |
| Relative peak power ( $\text{W kg}^{-1}$ ) | 10.51 $\pm$ 3.34       |
| Rate to fatigue ( $\text{W s}^{-1}$ )      | 14.01 $\pm$ 5.90       |
| Total work (J)                             | 16,695.63 $\pm$ 953.93 |
| LV mass ( $\text{g m}^{-2}$ )              | 98.6 $\pm$ 28.8        |
| Fractional shortening (%)                  | 18.7 $\pm$ 4.0         |
| Ejection fraction (%)                      | 57.6 $\pm$ 8.9         |

in Table 1. With the exception of AIx ( $P > 0.05$ ), WAT resulted in significant increases in all other haemodynamic indices assessed ( $P < 0.05$ , Table 2). The increase in carotid PP was significantly greater than the increase in brachial PP ( $+33 \pm 13$  versus  $+21 \pm 12$  mmHg,  $P < 0.05$ ). The correlations between resting/exercise change ( $\Delta$ ) haemodynamic variables with resting mFS are displayed in Table 3. As can be seen, there were significant associations between mFS and changes ( $\Delta$ ) in central haemodynamic load (carotid SBP, carotid PWV and carotid logNA,  $P < 0.05$  for all). There were no associations between exercise-induced change ( $\Delta$ ) in brachial SBP and mFS ( $P > 0.05$ ). Associations between mFS and exercise-induced change ( $\Delta$ ) in central haemodynamics remained after adjusting for age, body mass index and exercise-induced change ( $\Delta$ ) in brachial SBP (Table 3,  $P < 0.05$ ). The association between mFS and exercise-induced change ( $\Delta$ ) in carotid SBP remained after additional adjustment for change ( $\Delta$ ) in PWV ( $r = -0.542$ ,  $P < 0.05$ ) but not change ( $\Delta$ ) in logNA ( $r = -0.380$ ,  $P > 0.05$ ). There were no associations between any haemodynamic index measured and LV mass ( $P > 0.05$ ).

To assess whether exercise intensity and/or exercise test performance affected outcomes, we adjusted correlations for rate to fatigue as well as relative mean power output produced during the WAT. Associations between mFS and exercise-induced changes ( $\Delta$ ) in central haemodynamics remained significant after adjusting for the following WAT metrics: adjusted partial correlations for exercise ( $\Delta$ ) central SBP ( $r = -0.62$ ,  $P < 0.05$ ), exercise ( $\Delta$ ) in carotid logNA ( $r = -0.52$ ,  $P < 0.05$ ) and exercise ( $\Delta$ ) carotid PWV ( $r = -0.50$ ,  $P < 0.05$ ).

## Discussion

The present study examined the relationship between structural and functional properties of the left ventricle and properties of the large central arteries. The novel findings of this study were as follows. In healthy young men, resting LV function as assessed via midwall fractional shortening was inversely associated with WAT-mediated changes in carotid SBP,

pressure from wave reflections and carotid artery stiffness. There was no association between WAT-mediated change in brachial SBP and LV function nor were there associations between change in any haemodynamic parameter and LV mass. Associations between LV function and exercise-induced central haemodynamic load remained after adjusting for potential confounders and exercise-induced change in brachial SBP. These findings suggest that WAT-induced changes in central haemodynamic load are able to detect associations with resting LV function not detected by conventional resting or exercise brachial BP in young, apparently healthy men.

According to our primary findings, change in exercise central haemodynamic load (increase in carotid SBP, carotid wave reflection intensity and carotid stiffness) was associated with resting LV function in young, apparently healthy men. Associations between haemodynamic load and LV function were stronger with central versus brachial pressures and associations remaining significant even after adjusting for brachial pressures. Brachial BP may not be an adequate surrogate of central haemodynamic burden due to notable differences in pressure throughout the systemic circulation. Blood pressure is amplified as it moves away from the LV owing to transitions in vessel wall composition (elastic to muscular) and calibre (larger to smaller) as well as pressure from wave reflections. Indeed, our findings highlight that immediately after high-intensity exercise (a physical stress), there were greater increases in central versus peripheral BP, and this is consistent with our previous work with mental stress (Spartano et al., 2014). As such, studies relying on the exercise BP response measured in the brachial artery note disparate associations with LV structure and function. In older adults or adults with hypertension, central pressures more closely approximate peripheral pressures (Neisius et al., 2012) and the brachial BP response to exercise tends to more closely associate with measures of LV structure/function (Ren et al., 1985; Sung et al., 2003). Indeed, the peripheral exercise BP response predicts cardiovascular morbidity and mortality to a greater extent than resting BP in older hypertensive adults (Mundal et al., 1996; Kurl et al., 2001). However, in younger adults, the brachial BP response to exercise does not consistently associate with LV mass or LV systolic function (Lauer et al., 1992; Fagard et al., 1995; Cardillo et al., 1996; Markovitz et al., 1996; Takamura et al., 2008). Thus, the use of central exercise haemodynamic load may be insightful when examining associations with potential target organ damage in younger adults.

High-intensity exercise revealed associations between central haemodynamic load and resting LV function that were not detected with resting measures: an increase in central artery stiffness and pressure from wave reflections was associated with lower midwall fractional shortening. Increased central artery stiffness has a direct impact on myocardial energetics at rest (Kelly et al., 1992; Saeki et al., 1995) and during exercise (Kim et al., 1995); there is an increased energetic cost to maintain a given ejection/outflow due to a less compliant central vasculature. Central artery stiffening augments LV

**Table 2** Haemodynamic parameters at rest and following acute high-intensity exercise.

| Variable  | Baseline    | Post-Ex     | P-value |
|---|-------------|-------------|---------|
| Brachial Systolic Blood Pressure (mmHg)           | 120 ± 8     | 152 ± 13    | <0.001  |
| Brachial Diastolic Blood Pressure (mmHg)          | 74 ± 5      | 84 ± 5      | <0.001  |
| Brachial Pulse Pressure (mmHg)                    | 47 ± 6      | 68 ± 12     | <0.001  |
| Mean Arterial Pressure (mmHg)                     | 89 ± 5      | 107 ± 7     | <0.001  |
| Carotid Systolic Blood Pressure (mmHg)            | 114 ± 8     | 159 ± 12    | <0.001  |
| Carotid Pulse Pressure (mmHg)                     | 40 ± 9      | 74 ± 13     | <0.001  |
| Carotid Pulse Wave Velocity ( $\text{m s}^{-1}$ ) | 3.3 ± 1.4   | 4.6 ± 1.3   | 0.032   |
| Carotid $\log W_1$ ( $\text{mmHg m s}^{-3}$ )     | 0.88 ± 0.27 | 1.66 ± 0.25 | <0.001  |
| Carotid $\log NA$ ( $\text{mmHg m s}^{-2}$ )      | 1.43 ± 0.38 | 2.51 ± 0.55 | <0.001  |
| Carotid Augmentation Index (%)                    | 2.0 ± 8.1   | 9.0 ± 25.4  | 0.301   |
| Pulse Pressure Amplification                      | 1.18 ± 0.16 | 0.91 ± 0.12 | <0.001  |
| Heart Rate (bpm)                                  | 59 ± 10     | 95 ± 13     | <0.001  |

dysfunction through altered dependence of myocardial perfusion on systolic performance (Kelly et al., 1992; Saeki et al., 1995). Increases in central artery stiffness may also affect LV function by altering the timing and/or magnitude of wave reflections. In our study, the change in central artery stiffness was strongly associated with the change in wave reflection intensity with WAT ( $r = 0.60$ ,  $P < 0.05$ ). Both timing and

magnitude of wave reflections impact LV function (Zannoli et al., 1999; Borlaug et al., 2007). In young healthy adults, wave reflections arrive in diastole and this has a favourable impact on myocardial stress and coronary perfusion. However, a temporal shift of the returning wave to an earlier point in the cardiac cycle (i.e. systole) owing to reduced (faster) transit time from higher PWV alters loading sequence, negatively affecting myocardial wall stress (Chirinos et al., 2009). The cardiomyocyte appears particularly vulnerable to the ill effects of wave reflections arriving in late systole, reducing longitudinal systolic function (Borlaug et al., 2007; Chirinos et al., 2009, 2013).

We noted disparate associations between resting versus exercise wave reflection intensity and mFS. Previous work has demonstrated an inverse relationship between resting pressure from wave reflections and LV function in older adults and adults with hypertension (Paglia et al., 2014). In contrast, we discovered that young individuals with higher pressure from wave reflections at rest demonstrated increased midwall fractional shortening. This paradoxical observation may be attributed to the Anrep effect (von Anrep, 1912; Cingolani et al., 2013). To counteract higher levels of arterial pressure, myocardial contractility increases as a compensatory means of maintaining stroke volume ejection and overall adequate perfusion of target organs. Whether this increased LV work may contribute to reduced LV function and increased LV mass over time requires further study.

We noted associations between exercise central haemodynamic load and LV function but not structure in our sample of young healthy adults. Few studies have examined associations between central haemodynamic load provoked with a stressor and cardiac target organ damage. Chirinos et al. (2010) found

**Table 3** Univariate and partial correlations between resting and change from rest to postexercise ( $\Delta$ ) haemodynamics and LV function.

| Variables                           | Fractional shortening |              |                          |              |  |              |
|-------------------------------------|-----------------------|--------------|--------------------------|--------------|--|--------------|
|                                     | Zero-order            |              | Adjusted for age and BMI |              | Adjusted for age, BMI, and brachial exercise SBP |              |
|                                     | r                     | P            | r                        | P            | r  | P            |
| Resting Brachial SBP                | -0.119                | 0.331        | -0.327                   | 0.127        | —  | —            |
| Resting Carotid SBP                 | -0.006                | 0.491        | -0.181                   | 0.268        | —  | —            |
| Resting Carotid PWV                 | 0.270                 | 0.156        | 0.250                    | 0.195        | —  | —            |
| Resting Carotid $W_1$               | <b>0.515</b>          | <b>0.021</b> | <b>0.529</b>             | <b>0.026</b> | —  | —            |
| Resting Carotid NA                  | 0.419                 | 0.053        | 0.427                    | 0.064        | —  | —            |
| Resting Carotid AIx                 | -0.117                | 0.333        | -0.212                   | 0.233        | —  | —            |
| Exercise ( $\Delta$ ) Brachial SBP  | -0.256                | 0.169        | -0.350                   | 0.110        | —  | —            |
| Exercise ( $\Delta$ ) Carotid SBP   | <b>-0.565</b>         | <b>0.011</b> | <b>-0.562</b>            | <b>0.018</b> | <b>-0.509</b>                                    | <b>0.038</b> |
| Exercise ( $\Delta$ ) Carotid PWV   | <b>-0.437</b>         | <b>0.045</b> | <b>-0.468</b>            | <b>0.046</b> | <b>-0.421</b>                                    | 0.058        |
| Exercise ( $\Delta$ ) Carotid $W_1$ | -0.262                | 0.164        | -0.366                   | 0.099        | -0.374   | 0.094        |
| Exercise ( $\Delta$ ) Carotid NA    | <b>-0.583</b>         | <b>0.009</b> | <b>-0.606</b>            | <b>0.011</b> | <b>-0.580</b>                                    | <b>0.019</b> |
| Exercise ( $\Delta$ ) Carotid AIx   | -0.085                | 0.377        | -0.019                   | 0.474        | 0.044  | 0.443        |

SBP, systolic blood pressure; PWV, pulse wave velocity; NA, negative area; AIx, augmentation index; BMI, body mass index.

Significant correlations ( $P < 0.05$ ) are highlighted in bold. Exercise ( $\Delta$ ) metrics calculated as absolute change scores (postexercise value - resting value).

that isometric handgrip-induced increases in central artery impedance were predictive of LV mass in older hypertensive adults. Similarly, Keith et al. (2013) found that exercise-induced increases in central artery stiffness were associated with measures of renal target organ damage in older adults, but not younger adults. It is possible that changes in LV function may precede changes in LV structure (Hacker et al., 2006). Midwall fractional shortening has been shown to be predictive of LV diastolic dysfunction (Schussheim et al., 1998), LV exercise dysfunction and LV mass (Schussheim et al., 1997) and heart failure (Aurigemma et al., 2001). Thus, understanding haemodynamic factors that may correlate with reduced mFS is clinically relevant.

### Limitations and delimitations

The present study is cross-sectional and only provides insight into associations between haemodynamic indices and LV structure and function; this study is unable to elucidate the causal relationship of the variables. All cardiac values obtained were within clinical normal ranges; thus, exercise central haemodynamic load was not predictive of LV dysfunction *per se*, but rather lower-normal function. This study was conducted in young healthy men only. Previous studies have noted no sex differences in the haemodynamic response to high-intensity exercise (Rossow et al., 2010; Babcock et al., 2015). However, there may still be differences in how these indices associate with LV structure and function in women. As women age, there are greater increases in central artery stiffness and wave reflection intensity contributing to greater decrements in LV function, greater increases in LV mass and

ultimately higher prevalence of heart failure (Shim et al., 2011; Russo et al., 2012; Coutinho et al., 2013). Moreover, associations between central haemodynamic load and LV structure/function are greater in women, suggesting that women may be more susceptible to the deleterious effects of central haemodynamic load (Higashi et al., 2013; Coutinho et al., 2015). Thus, examination of the exercise central haemodynamic response as it relates to LV structure/function in young women as a means of unmasking early signs of cardiac target organ damage will be an important future study.

This study used very high-intensity exercise as a stressor, which may not be a practical stressor to use in a clinical/screening setting and may not be appropriate for older adults or adults at risk for CVD. Future research is needed to explore associations between LV structure/function and central haemodynamic load derived from lower intensity exercise in young adults.

### Conclusion

The use of high-intensity exercise to provoke changes in central haemodynamic load akin to what occurs with ageing and in the presence of hypertension, namely increases in central artery stiffness and wave reflection intensity, reveals associations with resting LV function in young, apparently healthy men not detected with resting or exercise brachial BP.

### Conflict of interest

The authors have no conflict of interests to disclose.

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