Systolic and Diastolic LV Mechanics During and Following Resistance Exercise

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Purpose To improve the current understanding of the impact of resistance exercise on the heart, by examining the acute responses of left ventricular (LV) strain, twist and untwisting rate ('LV mechanics').

Methods LV echocardiographic images were recorded in systole and diastole before, during and immediately after (7-12 s) double leg press exercise at two intensities (30% and 60% of maximum strength, 1-repetition-maximum, 1RM). Speckle tracking analysis generated LV strain, twist and untwisting rate data. Additionally, beat-by-beat blood pressure was recorded and systemic vascular resistance (SVR) and LV wall stress were calculated.

Results Responses in both exercise trials were statistically similar ($P > 0.05$). During effort, stroke volume decreased while SVR and LV wall stress increased ($P < 0.05$). Immediately following effort, stroke volume returned to baseline while SVR and wall stress decreased ($P < 0.05$). Similarly, acute exercise was accompanied by a significant decrease in systolic parameters of LV muscle mechanics ($P < 0.05$). However, diastolic parameters, including LV untwisting rate, were statistically unaltered ($P > 0.05$). Immediately following exercise, systolic LV mechanics returned to baseline levels ($P < 0.05$) but LV untwisting rate increased significantly ($P < 0.05$).

Conclusions A single, acute bout of double leg-press resistance exercise transiently reduces systolic LV mechanics, but increases diastolic mechanics following exercise, suggesting that resistance exercise has a differential impact on systolic and diastolic heart muscle function. The findings may explain why acute resistance exercise has been associated with reduced stroke volume but chronic exercise training may result in increased LV volumes.

Key words: resistance exercise; LV twist; diastolic function; athlete's heart

Introduction

Millions of people engage in physical activity and the cardiovascular health benefits of regular exercise are well described (14). Whilst both endurance ('aerobic') and resistance exercise have been associated with positive cardiovascular effects (14, 45), controversy remains over the specific impact these two types of exercise have on the heart. Previous studies have consistently reported that acute endurance exercise is associated with an increase in stroke volume and that chronic exercise training also results in an increase in cardiac volumes as well as altered cardiac function (27, 32). Conversely, the data on resistance exercise training are inconclusive, but some studies suggest that acute resistive effort reduces left ventricular (LV) stroke volume (5, 18), while strength trained athletes may have unaltered or enhanced systolic and diastolic LV size and/or function (1, 22, 25, 31, 32). The assessment of novel indicators of cardiac function before, during and immediately following resistance exercise will elucidate the acute effects of resistance exercise on LV function, which may assist the interpretation of chronic adaptation to strength training.

During ventricular contraction, the LV muscle shortens in the circumferential and longitudinal plane which is characterized by a twisting motion of the LV around its long-axis. Throughout the subsequent diastolic relaxation, rapid LV untwisting occurs, which has been associated with passive restoring forces and LV suction (23, 24). Technologies now exist that enable the quantification of parameters like LV circumferential strain and twist ('LV mechanics') across the cardiac cycle (8, 30, 33, 35, 37, 39). These measurements focus on the heart muscle function, as opposed to the hemodynamic focus of previously used indicators including stroke volume and trans-mitral blood velocities. Accordingly, assessing LV mechanics will likely assist in understanding the acute impact of resistance exercise upon cardiac function. Furthermore, the inclusion of post-exercise measurements will help in identifying whether any changes in systolic and diastolic LV function observed during resistance exercise are transient

or last into the recovery period. As LV mechanics are afterload-dependent (7, 21) and the major impact of resistance exercise is thought to be related to afterload(12, 20), LV mechanics need to be interpreted in the context of alterations in parameters reflecting afterload. Without assessing intrinsic cardiac contractility, what constitutes the best parameter to represent 'afterload' is somewhat controversial (17). However, both systemic vascular resistance (SVR) and LV wall stress have been studied during resistance exercise and may be influencing LV mechanics (12, 18). Therefore, the aim of this study was to examine systolic and diastolic LV muscle mechanics as well as SVR and LV wall stress before, during and immediately after an acute resistive effort at two different exercise intensities. We hypothesized that 1) acute resistance exercise would significantly increase systemic vascular resistance (18) and LV internal (as opposed to transmural) wall stress and concomitantly reduce LV systolic mechanics and 2) that these acute alterations would be restored to baseline levels immediately after acute exercise effort. Because of the known interdependence of LV twist and untwisting rate (36), we anticipated the responses in LV untwisting rate to mirror those of LV twist.

Methods

As this experiment formed part of a larger investigation, some of the experimental methods for this study have been reported previously (39). However, the present article addresses a different question and we repeat only the methods and data essential to the novel findings presented here. We do so in the interest of conciseness and hope that this does not cause the reader any inconvenience.

Study Population

Following ethical approval from the Cardiff Metropolitan University School of Sport Ethics Committee, 15 healthy males (age: 21 ± 3 years; height: 176.5 ± 6.2 cm; mass; 80.6 ± 15.3 kg;

double leg-press one-repetition maximum, 1RM: 317 ± 72 kg) volunteered and provided written informed consent to take part in the study.

Preparatory pilot work

For this experiment, extensive pilot work was undertaken to generate the final protocol for data collection. First, the mode of exercise was determined in relation to the method chosen to obtain valid echocardiographic data. Thus, similar to Haykowsky *et al*. (12), we chose double-leg press as the chest is stable and the echocardiographic windows enable for acquisition of data with appropriate quality, which would not be possible during other modes of resistance exercise such as bench press. Second, we wanted to obtain data at two different exercise intensities to a) increase the robustness of our observations due to repeated measurements and b) determine whether the responses would be intensity-dependent. The aim of the pilot work was to ensure that a protocol was generated that would enable the successive data collection of ten echocardiographic images (i.e. ten separate exercise efforts per exercise intensity), without causing a hemodynamic drift from the first to the last double leg press. This was successfully achieved by including a 2-min rest period in between each single double-leg press. A schematic diagram of the final protocol including the time points of data collection is shown in (39). Please note that the time to obtain echocardiographic images immediately following exercise took 10-15 seconds.

Echocardiography

A total of ten echocardiographic images were recorded from the following echocardiographic windows: parasternal long-axis, parasternal short-axis (mitral valve, papillary muscle and apex), apical 4 chamber, apical 2 chamber, trans-mitral Doppler, tissue Doppler (septum and RV) and M-mode of the LV color Doppler map. For this study, the following parameters were

calculated, all in accordance with the latest guidelines on chamber quantification (16). Parasternal long-axis images were analyzed for end-diastolic and end-systolic dimensions and LV mass (using the formula by the American Society of Echocardiography), obtained from measurements performed directly on still images of the 2-D video loop. LV mass was allometrically scaled to body surface area according to DuBois & DuBois (9). Relative wall thickness (RWT) was calculated as 2 x posterior wall thickness (cm) divided by end-diastolic diameter (cm). LV volumes were analyzed using the Simpson's biplane method and cardiac output was calculated as the product of heart rate and stroke volume. From the trans-mitral Doppler signal, peak early (E) and late (A) diastolic blood velocity as well as the ratio (E/A) were determined. Systolic (S') and diastolic (E' and A') tissue Doppler velocities were obtained by placing a sample volume in the basal region of the septum and RV free wall, and peak velocities were measured using a software-integrated caliper.

Speckle tracking analysis of circumferential strain, strain rate, twist and untwisting rate and twist-to-shortening ratio. The speckle tracking echocardiography procedures have been described previously in separate publications (36) as well as the study associated with this investigation (39). From the raw data output, beat-by-beat interpolated basal and apical data were time aligned and subtracted from each other, producing beat-by-beat twist, twist rate, strain and strain rate curves. From the twist rate curve, the first most negative deflection in early diastole was defined as peak untwisting rate. Twist-to-shortening ratio, an indicator of the amount of twist per LV shortening that has been proposed to represent the balance between subendocardial and subepicardial forces (2, 19), was calculated as previously described (41). Since untwisting rate is at least in part associated by the preceding contraction, and normalization may be essential for the interpretation of differences in untwisting rate (40), we also calculated relative untwisting rate by dividing untwisting rate by peak twist.

Systemic vascular resistance (SVR) and end-systolic LV meridional wall stress

SVR was calculated as mean arterial blood pressure divided by cardiac output. End-systolic LV meridional wall stress was calculated using a modified combination of validated equations (11, 26). The final equation used was

$$0.334 * SBP * 0.9 * LVIDs / (SWTs+PWTs)/2) * (1+(SWTs+PWTs)/2) / LVIDs$$

where SBP was systolic blood pressure in mmHg which was multiplied by 0.9 to reflect the lower pressure at end-systole,(13) LVIDs was the LV end-systolic internal diameter in mm and SWTs and PWTs were the end-systolic septal and posterior wall thicknesses in mm, respectively. This equation generated data in mmHg, which were converted into $(dyne/cm^2)*10^{-3}$ by applying a conversion factor of 1.333.

Statistical Analyses

Two-way repeated measures ANOVA was used to identify main effects over time (pre, during, post) and between exercise intensities (30% and 60% of 1 RM) as well as interaction effects. If significant main effects were detected, *post hoc* one-way repeated measures ANOVA and Tukey's multiple comparison tests were applied. Significance was accepted at $P < 0.05$. For all statistical analyses, GraphPad Prism (GraphPad Prism for Windows, version 5.0.1; GraphPad Inc., San Diego, CA, USA) was used. Data are presented as means ± SD.

Results

Resistance and LV systolic function

Baseline cardiac dimensions of the study population are presented in Table 1. Resistance exercise significantly increased SVR (Figure 1) and end-systolic LV meridional wall stress, which was accompanied by the previously reported increase in heart rate and a reduction in stroke volume (39). Similarly, there was a significant reduction in all systolic LV muscle

parameters (twist, TSR, circumferential strain and circumferential strain rate) during double-leg press exercise, while systolic tissue Doppler velocities were statistically unaltered. Immediately following exercise, all systolic parameters that had been altered during effort were restored to baseline levels or even surpassed baseline values (see Table 2).

LV diastolic function

In contrast to systolic parameters, there was no significant change in any diastolic parameters during exercise (Table 3), except for a significant increase when untwisting rate was normalized to the preceding LV twist. Immediately following the double-leg press effort, a statistically significant increase in some diastolic parameters was observed, most notably in LV untwisting rate, normalized LV untwisting rate and diastolic circumferential strain rate at the apex, both of which increased significantly above exercise and baseline levels, respectively (Figure 2).

Discussion

The main aim of this study was to examine the acute effects of a single bout of resistance exercise upon systolic and diastolic LV muscle function. There were two main findings: 1) Any reduction in systolic LV parameters observed during acute resistance exercise effort was transient and immediately restored to normal baseline levels after exercise and 2) Diastolic LV function was not altered during a single acute resistive effort, but was significantly enhanced above baseline levels immediately after exercise. Together, the present data suggest that an acute resistive effort, albeit of low volume of work, may acutely have a differential impact on systolic and diastolic LV mechanics. As such, our results provide a potential explanation for the differential systolic and diastolic adaptation previously reported with regular resistance training. The next paragraphs will first discuss the transient nature of the reduction in LV

function during resistance exercise before evaluating the uncoupling of systolic and diastolic LV function in relation to this type of exercise.

Transient reduction in systolic LV function during resistive exercise effort

Resistance exercise-induced reductions in systolic LV function, for example in stroke volume, have been reported previously (28). Although some studies have found opposing results (1), the methodological approaches in those studies (for example linear echocardiographic measurements that do not take into account the possible shape changes of the LV during marked alterations in loading state) likely influenced the results. The reduction in stroke volume observed in the present study agrees with the responses observed in other experimental conditions that increased afterload (15, 44). Accordingly, reduced afterload following resistance exercise likely restored the normal stroke volume in the present study, as supported by the near significant change in end-systolic volume, without significant alterations in end-diastolic volume.

The most novel aspect of this experiment was the inclusion of measurements of LV muscle mechanics, with the intention to advance the current understanding of the impact of resistance exercise on cardiac function. In general, the response in LV twist mechanics could not be attributed to the prevailing hemodynamics and heart rate, since EDV, ESV, heart rate and cardiac output (see the previously published results in 39) did not match the pattern of response of LV twist and untwisting rate. Still, a clear decline in systolic LV twist, strain and strain rate was observed during the resistive effort. This is in accordance with previous studies investigating general cardiac function during resistance exercise (28). Furthermore, the reduction in systolic LV mechanics agrees with studies examining the isolated effects of changes in afterload upon LV mechanics, showing that increased LV afterload acutely reduces LV muscle mechanics (4, 7, 10, 39, 41, 44). The present data show that the increased SVR and

wall stress during resistance exercise were also accompanied by similar responses in systolic LV mechanics, including TSR. This suggests that resistance exercise alters the balance between endocardial and epicardial forces in the left ventricle, towards a more dominant contribution of the endocardial fibers (19), which may be associated with increased wall stress. Similar to the known influence of increased afterload and wall stress on a reduction in myofiber shortening velocity (6, 29), all systolic LV mechanics parameters in this study and the previously published report (39) declined during resistance exercise. However, it must be noted that these results contradict the findings of Haykowsky *et al.* (12) who found that LV end-systolic transmural wall stress did not change during acute resistance exercise. The present data cannot be interpreted to represent the same physiology as that shown by Haykowsky and colleagues, as intrathoracic pressures were not ascertained in the present investigation. From this experiment, it would appear that internal LV wall stress that does not include the contribution of intrathoracic pressures is more closely associated to LV systolic mechanics than transmural LV wall stress. Whether increased wall stress is directly responsible for the reduction in systolic LV mechanics remains to be determined. Since resistance exercise also increases diastolic blood pressure, thus facilitating enhanced coronary perfusion, it is possible that this kind of physical effort improved endocardial perfusion and consequently enhanced the contribution of endocardial fibers, resulting in a significant decrease in TSR during double leg press.

Importantly, in the present study all systolic parameters returned to baseline levels immediately after exercise, and circumferential strain rate at the apex even surpassed the baseline values following resistance exercise at 60% of 1RM. These data provide strong indication that an acute bout of resistance exercise at low to medium intensities does not reduce systolic LV function beyond the duration of the exercise effort. Of course, further work is required to ascertain whether similar responses will be seen in a more ecologically-valid setting, where people perform multiple repetitions and sets of resistance exercise also at higher

workloads, as is common practice (3). The biggest difference between the current study and more real-life based scenarios could lie in the time course of recovery of systolic LV function, whereby a more repetitive and more intense effort may require a longer time for LV systolic mechanics to return to normal baseline values. Still, it is likely that systolic function will recover even with greater resistance exercise stimuli, as supported by reports of similar or even enhanced stroke volume and LV contractility in strength trained athletes compared with untrained individuals (1, 32, 42). These data suggest that the overall impact of resistance exercise training may be very similar to that observed during the acute effort performed in the present study. Taken together, the present data suggest that acute lower-limb resistance exercise does not have negative effects on systolic LV function in healthy individuals.

Uncoupling of systolic and diastolic LV mechanics

A novel observation in the present study was the uncoupling of systolic and diastolic LV mechanics, as reflected by the significant reductions in systolic LV mechanics during resistive effort, whilst no significant change in diastolic LV mechanics was observed at the same time point. In fact, a significant increase in untwisting rate was seen when normalized to twist. These results suggest a preservation and maybe even a necessary increase of diastolic LV muscle function during acute resistive effort. The mechanisms for a maintained diastolic LV muscle function during resistance exercise are unknown at this point in time. It is possible that the increased sympathetic state during acute exercise effort compensated for some of the decline in LV twist as recently suggested (4, 34). However, an increased storage of potential energy appears unlikely since ESV did not decrease beyond its normal resting state (43), even though this cannot be fully excluded. A more likely explanation may therefore be that the combination of hemodynamic changes and altered sympathetic state may not have altered the passive stiffness of the myocardium, which has been shown to be associated with untwisting rate (24).

Following resistance exercise, diastolic circumferential strain rate at the LV base returned to pre-exercise levels. However, LV untwisting rate and diastolic circumferential strain rate at the apex were significantly enhanced during the 60% 1RM trial. The mechanisms for this response also require further examination, but the current data suggest altered intrinsic relaxation or a reduced diastolic load of the LV (24). Together with previous reports of enhanced diastolic function in strength trained athletes (1), the present results advance these recommendations by indicating that individuals with reduced diastolic function may benefit most from this exercise modality.

Methodological strengths and weaknesses

Despite an extensive pilot period and a strong attempt to work as precisely as possible, some limitations related to this experiment need to be acknowledged. Echocardiographic imaging is challenging during physical effort when heart rates and lung volumes are increased. However, images can be obtained during intense physical effort (8, 30, 37, 38) and in this study, young healthy individuals were enrolled that had appropriate echocardiographic windows in the semi-supine position. Furthermore, any small deviations from optimal echocardiographic windows may have resulted in small under- or overestimation of some values, however the within-subject comparison was not impacted due to a standardized positioning of the cardiac ultrasound transducer in all six measurement time points. The similar pattern observed between the two independent exercise trials (30% 1RM and 60% 1 RM) seems reflective of the high reliability of the data. The low *P*-values in both trials further support this. The authors acknowledge that the mode of exercise can only be considered ‘experimental’ as the real-world performance of resistance exercise typically involves multiple repetitions and multiple sets.

Additionally, the present results are currently only true for young healthy men and the authors apologize for not having included data on females at this point in time. In the future, it is highly recommended that both men and women across the entire age spectrum be studied during resistance exercise.

Conclusions

A single, acute bout of double leg-press resistance exercise transiently reduces systolic LV muscle mechanics, but increases diastolic function following exercise, suggesting that resistance exercise has a differential impact on systolic and diastolic heart muscle function. The findings may explain the previously described differential effects of resistance exercise training on systolic and diastolic function.

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Figure legends

Figure 1. Systemic vascular resistance (SVR) and LV wall stress. During the resistance exercise effort, SVR and wall stress increased significantly. Immediately following resistance exercise, SVR and wall stress decreased and SVR even dropped significantly below levels measured prior to exercise. *: 30% and 60% significantly different from 'Pre'; $: 30% and 60% significantly different from 'During'. PRE: Baseline immediately before resistance exercise; DURING: Data obtained during a single bout of double leg press resistance exercise; POST: data obtained 7-12 seconds following resistance exercise.

Figure 2. Left ventricular (LV) twist and untwisting rate. A) Acute resistance exercise was associated with a significant reduction in systolic LV twist, whilst diastolic LV untwisting rate was statistically unaltered. In contrast, immediately following resistance exercise LV twist returned to baseline (and even exceeded it) but LV untwisting rate was significantly enhanced

above baseline values. **B)** When LV twist was normalized to LV shortening as reflected by the twist-to-shortening ratio (TSR), a significant reduction was observed DURING, followed by a significant increase above baseline immediately POST resistance exercise. Conversely, when LV untwisting rate was normalized to the preceding peak LV twist, a significant increase DURING and POST was observed. These data suggest that altered diastolic function DURING and POST was altered by different mechanisms. *: significantly different from PRE; †: significantly different from DURING. PRE: Baseline immediately before resistance exercise; DURING: Data obtained during a single bout of double leg press resistance exercise; POST: data obtained 7-12 seconds following resistance exercise.