A systematic review of resting left ventricular systolic and diastolic function and adaptation in elite weightlifters

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Background: This review aims to establish what effect weightlifting has on the systolic and diastolic function of the left ventricular (LV).

Sources of data: PubMed; ISI Web of Knowledge; Cochrane Library and Ovid Medline were searched in February 2012 to find literature on the effect of weightlifting on the LV cardiac function.

Areas of agreement: Stroke volume, posterior wall thickness and ventricular filling time and rate were seen to increase. A decrease in the resting heart rate was seen.

Areas of controversy: Blood pressure and LV morphological changes were equivocal.

Growing points: Weightlifting causes recognizable functional change to the LV, some of these changes may confer benefits such as improvements in the systolic function.

Areas timely for developing research: Discrepancies exist with regard to regional LV morphological change, as the evidence suggests the LV does not adapt in a homogenous manner. Attempts should be made to separate performance-enhancing drug users from those who compete drug free.

Keywords: weight lifting/cardiovascular adaptation/left ventricle/weight training

Introduction

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Heavy resistance training (RT) and its consequent cardiac effects have been comprehensively investigated. Resistance trained athletes range from those exclusively participating in weightlifting, bodybuilding and powerlifting to those using strength training as an extension to their sporting practice to develop and enhance beneficial physical attributes.1–3
Among RT athletes, however, weightlifters alone are seldom considered independently, and studies describe divergent findings in consideration to left ventricular (LV) adaptation. Studies report weightlifting bearing significant correlations with LV hypertrophy (LVH) and increasing LV mass (LVM) when indexed to body size\textsuperscript{4,5} and when considered as an absolute value.\textsuperscript{6} However, contrasting studies show no relationship among these athletes against suitable controls once standardized to lean body mass (LBM) or body surface area (BSA),\textsuperscript{8–10} even though training exposure lasted >10 years.\textsuperscript{7} In light of this, Washam \textit{et al.}\textsuperscript{11} illustrated a correlation between training experience and absolute LV posterior wall thickness (PWT) and septal wall thickness (ST) when normalized to LBM in female weightlifters. These relationships, however, were not significant when normalized to BSA.

MacDougall \textit{et al.}\textsuperscript{12} showed that some RT lifts induce extremely high blood pressure (BP) levels, in excess of 480/350 mmHg during commonly used exercises. The described pressure overload induced in such circumstances is the hypothesized stimulus to this cardiac hypertrophy, although this does not explain why in some subjects this adaptation is absent.

Diastole and systole are seldom considered in unison in weightlifters. Diastole is responsible for the adequate filling and dilation of the ventricles to provide the necessary stroke volume (SV) to maintain a sufficient cardiac output for systole. Systole is involved with active myocardial contraction and consequent propelling of blood held in the ventricles after diastole to the lungs and body through the pulmonary and systemic circulations, respectively. Measurements taken which help determine the cardiac function are attributed to these stages, and, by analysing these measures concurrently, an explanation could be given for the aforementioned discrepancies. This review aims to establish the effects of weightlifting on the systolic and diastolic function of the left ventricle.

**Search strategy**

A literature search was performed in February 2012 in Pubmed; ISI Web of Knowledge; The Cochrane Database and Ovid Medline. In each of these:

(Left ventricle OR left ventricular AND diastolic function AND athlete OR athletes OR weightlifters OR weightlifting NOT disease OR failure).

The results obtained, along with the selection criteria to narrow these results, are illustrated in Figure 1.
Inclusion criteria consisted of athletes whose training included exclusively weightlifting and no endurance based or cross training. Cardiac-related measurements were made using either 2-D, M-mode or Doppler echocardiography (EC) at rest. Of the remaining seven studies, all but one involved subjects competing at the national level, with the remainder featuring athletes from club-level competition all sufficiently adapted to their sport. All but one study featured athletes whose training experience surpassed 6 years and who trained >10–15 h weekly. Many were free of any known cardiovascular disease or risk factors. Two studies mention the use of pharmacological agents—Vinereanu et al. specifically asked those who took known metabolic agents not be volunteer for participation in the study, and MacFarlane et al. recognized that three of their athletes admitted to using anabolic/androgenic drugs preceding the study. Despite these confounding effects, these athletes were included in the study, thus threatening the reliability of findings. All results from physical examinations and laboratory tests were normal for all subjects. Each study consisted of age-matched sedentary individuals as controls—with no significant age differences between groups.

Fig. 1 Search strategy used to source relevant articles.
Table 1 illustrates some of the weightlifters’ characteristics used in the group-matching process. All data are mean ± SD.

Table 1 highlights some clear differences in the studied populations—the mean age ranges from 23.0 to 47.8 years and weight from 78.7 to 94.0 kg. The body surface area (BSA) correlates with LVH, especially in those exposed to chronic pressure overload such as weightlifters, and hence cardiac-related measurements are often taken in reference to this. All participants involved were male, and therefore the findings from this review cannot be directly extended to the female population. However, this eliminated a possible confounding variable.

### Discussion

Blood pressure measurements across various studies report no differences in either diastolic or systolic BP or systolic BP alone between weightlifters and sedentary controls. In contrast, Adler et al. show significant differences between systolic \((P < 0.0001)\) and diastolic \((P < 0.018)\) BP between weightlifters and controls. However, the age of athletes in other studies differs considerably, with a mean age of 24.3 ± 1.2 years or up to an age of 47.8 ± 3.2 years, as opposed to Adler et al. that features weightlifters at a mean age of 31.2 ± 3.8 years. This difference reflects physiological changes that are attributed to training exposure, which is then lost as an athlete approaches middle age. Although Fisman et al.’s study features athletes of a similar age range (29.0 ± 4.0 years) to Adler et al.’s, and, although both studies include a large numbers of weightlifters, all from national

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**Table 1** Weightlifters’ anthropometric characteristics used in the group-matching process.

<table>
<thead>
<tr>
<th>Study</th>
<th>Study design</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Body mass index ((kg/m^2))</th>
<th>Body surface area ((m^2))</th>
</tr>
</thead>
<tbody>
<tr>
<td>D’Andrea et al.</td>
<td>Prospective controlled</td>
<td>47.8 ± 3.2</td>
<td>N/A</td>
<td>N/A</td>
<td>1.93 ± 0.6</td>
</tr>
<tr>
<td>Vinereanu et al.</td>
<td>Prospective controlled</td>
<td>36.0 ± 6.0</td>
<td>N/A</td>
<td>N/A</td>
<td>2.1 ± 0.2</td>
</tr>
<tr>
<td>Adler et al.</td>
<td>Prospective controlled</td>
<td>31.2 ± 3.8</td>
<td>94.0 ± 5.0</td>
<td>29.5 ± 2.8</td>
<td>N/A</td>
</tr>
<tr>
<td>MacFarlane et al.</td>
<td>Cross sectional</td>
<td>24.3 ± 1.2</td>
<td>78.7 ± 3.6</td>
<td>N/A</td>
<td>1.9 ± 0.1</td>
</tr>
<tr>
<td>Fisman et al.</td>
<td>Prospective controlled</td>
<td>23.0 ± 4.0</td>
<td>80.0 ± 7.0</td>
<td>N/A</td>
<td>1.9 ± 0.1</td>
</tr>
<tr>
<td>Fisman et al.</td>
<td>Prospective controlled</td>
<td>29.0 ± 4.0</td>
<td>80.0 ± 6.0</td>
<td>25.6 ± 2.3</td>
<td>N/A</td>
</tr>
</tbody>
</table>

All data are mean ± SD, N/A refers to data not available.
teams with similar training exposure, disparity remains. Methodological differences may explain this disagreement, whereby two studies\textsuperscript{15,16} mention the use of cuff sphygmomanometry. Fisman \textit{et al}.\textsuperscript{18} describe this method being used at the brachial site, in accordance with the recommendations of the American Heart Association. Also, Adler \textit{et al}.\textsuperscript{16} recognized that the body mass in weightlifters was greater than that in controls ($P < 0.0001$). Body mass correlates with cardiac morphology,\textsuperscript{19} and therefore this is a potential confounder.

A resting heart rate (RHR), an indirect indicator of SV and diastolic filling capacity, shows imbalanced trends across the studies. Master category weightlifters (mean age: $47 \pm 3.2$ years) have significantly lower RHR ($P < 0.05$) than age-matched controls ($69.9 \pm 9.94$ versus $72.9 \pm 7.9$ beats/min, respectively)\textsuperscript{7}—Fisman \textit{et al}.\textsuperscript{17,18} ($P < 0.001$), and Adler \textit{et al}.\textsuperscript{16} used electrocardiography to monitor RHR,\textsuperscript{16,17} and their results agree with those above. Neither Fisman \textit{et al}.\textsuperscript{18} D’Andrea \textit{et al}.\textsuperscript{7} nor the remaining studies\textsuperscript{4,15} mention any detail regarding measurement methods. No significant difference was mentioned in the remaining studies.\textsuperscript{4,15}

Adler \textit{et al}.\textsuperscript{16} and Fisman \textit{et al}.\textsuperscript{18} showed that SV was significantly greater ($P < 0.05$) in weightlifters than controls, thus complementing the aforementioned decreased RHR. Contrastingly, Fisman \textit{et al}.\textsuperscript{17} and D’Andrea \textit{et al}.\textsuperscript{7} were unable to identify a difference between groups. This discrepancy is mentioned in two studies\textsuperscript{17,18} despite control groups being almost identical and both similarly deriving SV from cavity measurements. Conversely, D’Andrea \textit{et al}.\textsuperscript{7} calculated SV by obtaining LV outflow by Doppler US, whereby the product of LV outflow tract area and outflow velocity were multiplied—perhaps a more direct measure than the estimation through cavity measurements. Adler \textit{et al}.\textsuperscript{16} does not mention the methodology of obtaining SV.

The morphology of the LV can be evaluated by assessing measures that quantify regional and global hypertrophy (Table 2). Five studies\textsuperscript{4,7,16–18} report consistent findings regarding the LVM, concluding that measures in weightlifters are significantly greater than that in controls ($P < 0.01$, $P < 0.001$, $P < 0.001$). Fisman \textit{et al}.\textsuperscript{17,18} used end-diastolic wall thickness and cavity dimensions to estimate LVM, whereas Adler \textit{et al}.\textsuperscript{16} used the leading-edge method from the American Society of EC guidelines (AmSocECG). In one study,\textsuperscript{4} LVM was indexed to BSA to correlate this difference, and the result remained consistent ($P < 0.01$). This is further supported by D’Andrea \textit{et al}. and Vinereanu \textit{et al}. using the Penn convention indexed for height\textsuperscript{7} and the method of Devereux with the application of the Penn convention,\textsuperscript{15} which showed a significantly increased LVM index of weightlifters ($P < 0.001$, $P < 0.05$). The reliability of EC estimations of LVM in comparison to each other and to three-dimensional magnetic resonance
Table 2  LV characteristics of weightlifters (WL) versus sedentary controls (C).

<table>
<thead>
<tr>
<th>Study</th>
<th>Group demographics</th>
<th>LVM</th>
<th>LVMI</th>
<th>LVESID</th>
<th>LVEDID</th>
<th>LVESV</th>
<th>LVEDV</th>
<th>PWT</th>
<th>ST</th>
</tr>
</thead>
<tbody>
<tr>
<td>D’Andrea et al.</td>
<td>45 (20 WL/25C)</td>
<td>N/A</td>
<td>$P &lt; 0.001$</td>
<td>—</td>
<td>—</td>
<td>N/A</td>
<td>N/A</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
</tr>
<tr>
<td>Vinereanu et al.</td>
<td>25 (11 WL/14C)</td>
<td>N/A</td>
<td>$P &lt; 0.05$</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>$P &lt; 0.05$</td>
<td>$P &lt; 0.05$</td>
</tr>
<tr>
<td>Adler et al.</td>
<td>96 (48 WL/48C)</td>
<td>$P &lt; 0.0001$</td>
<td>N/A</td>
<td>N/A</td>
<td>—</td>
<td>—</td>
<td>$P &lt; 0.05$</td>
<td>$P &lt; 0.05$</td>
<td></td>
</tr>
<tr>
<td>MacFarlane et al.</td>
<td>20 (10 WL/10C)</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>N/A</td>
<td>—</td>
<td>N/A</td>
<td>N/A</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Fisman et al.</td>
<td>36 (16 WL/20C)</td>
<td>$P &lt; 0.0001$</td>
<td>N/A</td>
<td>N/A</td>
<td>—</td>
<td>—</td>
<td>$P &lt; 0.001$</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Fisman et al.</td>
<td>74 (40 WL/34C)</td>
<td>$P &lt; 0.01$</td>
<td>N/A</td>
<td>N/A</td>
<td>—</td>
<td>$P &lt; 0.01$</td>
<td>$P &lt; 0.01$</td>
<td>—</td>
<td></td>
</tr>
</tbody>
</table>

LVM, left ventricular mass; LVMI, left ventricular mass index; LVESID, left ventricular end-systolic internal dimension; LVEDID, left ventricular end-diastolic internal dimension; LVESV, left ventricular end-systolic volume; LVEDV, left ventricular end-diastolic volume; PWT, posterior wall thickness; ST, septal wall thickness. All levels of significance stated except where N/A, data not available and —, no significant difference.
imagery (MRI) show that EC estimations produce similar results to one another, both quantitatively and prognostically. However, when compared with MRI, the confidence limits of estimates extend towards $\pm 55 \, \text{g}$, which may easily prevent or portray a difference being seen.

LV dimensions and volume are both reflections of LV filling and emptying capabilities, taking measures at end diastole and end systole, respectively. Many studies did not take readings related to these variables, and from those that did, only Fisman et al. reported a significantly greater LV end-diastolic volume (EDV) compared with controls ($P < 0.01$). The methods of these studies were in accordance with the American Society of Cardiology, which used the leading-edge method. Fisman et al. used cavity measurements to calculate LV volumes. Vinereanu et al. used a modified Simpson’s method, which does not under- or over-estimate calculations with regard to the LV function compared with contrast ventriculography.

PWT and ST were measured in all studies, with results showing consistent trends. PWT was significantly greater in weightlifters than controls. ST, although greater in all studies, was only noted as being significantly greater in weightlifters than controls by Vinereanu et al. and Adler et al. ($P < 0.05$), and by D’Andrea ($P < 0.01$). All studies used two-dimensional M-mode EC to assess these variables, whilst some implicitly stating a parasternal long-axis orientation of the EC transducer. The AmSocECG recommends this orientation for the visualization of the above variables, and the studies using this method can thus be compared more confidently.

The studies used either ejection fraction (EF) or fractional shortening (FS) to assess the systolic LV function. Three studies found no significant difference to controls when measuring EF. Similarly, MacFarlane et al. was unable to identify a notable difference between weightlifters and controls with regard to FS—however, D’Andrea recognized a significantly increased FS in weightlifters ($P < 0.01$). Four studies estimated the systolic function with cavity measurements obtained from two-dimensional M-mode EC, whereas MacFarlane et al. did not report any detail concerning this measurement.

Doppler EC was used to assess the rate of blood flow at particular periods during ventricular filling (Fig. 2).

Figure 2 shows the peak flow velocity readings from two studies, both confirming significantly ($P < 0.05$ and $P < 0.02$) higher peak flow velocity in athletes ($112 \pm 9$ and $114 \pm 12 \, \text{cm/s}$) in comparison with controls ($103 \pm 10$ and $102 \pm 9 \, \text{cm/s}$), respectively. This contradicts previous findings from Fisman et al., whereby EF was not significantly different, and leads us to question the validity of two-dimensional M-mode EC in measuring EF, and perhaps systolic function overall. The validity of EC, however, has been criticized in several
studies, although it is considered a reliable indicator of cardiac functional assessment. As a result of this, many studies have used EC to assess the cardiac function in healthy individuals, in pathology, and also athletes. On both occasions, measurements were taken in an apical four-chamber view after adjustments to obtain ‘optimal flow’ patterns. One can infer from this that all readings reflect the peak function, although positioning among the subjects may have varied. This affects the reliability of measurements, but may also reflect anatomical differences among the subjects.

Figure 3 displays acceleration and deceleration rates across two studies. Adler et al. reported that both acceleration and deceleration rate measurements in weightlifters were significantly greater than that in controls (1242 ± 176 versus 890 ± 154 and 414 ± 44 cm/s/s versus 344 ± 45 cm/s/s, respectively, P < 0.0001). Fisman et al. also noted that the acceleration rates of weightlifters were significantly greater than that of controls (1370 ± 120 versus 1220 ± 110 cm/s/s, P < 0.01).

Data concerning the mean acceleration, deceleration and isovolumetric relaxation times are shown in Figure 4. Only Adler et al. reported significantly shorter times for weightlifters in all three of the
variables (55 ± 4 versus 69 ± 7 ms, 164 ± 4 versus 177 ± 9 ms and 63 ± 3 versus 74 ± 6 ms, respectively; \( P < 0.0001 \)). Vinereanu et al.\textsuperscript{15} measured two of the variables, and found that the mean deceleration time and isovolumetric relaxation times in weightlifters were not significantly different from controls (174 ± 33 and 78 ± 26 ms versus 176 ± 38 and 84 ± 15 ms, respectively). Both studies describe the sample volume placement at the mitral valve leaflets, whilst measurements were taken over two\textsuperscript{15} and five\textsuperscript{16} complete cardiac cycles during passive end-expiration. The methodology between the studies is very similar—perhaps Vinereanu et al.\textsuperscript{15} may have required more subjects to recognize a difference.

Figure 5 displays information regarding the peak early and atrial (late) velocities—indicating the time for which the LV fills with blood, during diastole or early systole, respectively. Adler et al.\textsuperscript{16} report significantly greater (\( P < 0.0001 \)) velocities in weightlifters compared with controls.

Figure 6 illustrates the mean \( E/A \) ratio, a measurement particularly relevant in the cardiac dysfunction, whereby the \( E/A \) ratios are reduced, as LV filling becomes more dependant on atrial contraction (atrial) than on passive diastolic filling (early). \( E/A \) ratios were

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**Figure 3** Acceleration and deceleration rates of blood flow in athletes (A) and controls (C).

Data are expressed as \( \bar{x} ± s \). Athletes exhibited significantly greater rates of acceleration and deceleration of blood flow compared with sedentary controls. **\( P < 0.01 \); ***\( P < 0.001 \).
Fig. 4 Mean acceleration, deceleration and isovolumetric relaxation times in athletes (A) and controls (C). Data are expressed as $\bar{x} \pm \sigma$. Athletes were shown by Adler et al. to exhibit quicker relaxation times than controls; however, this was not shown by Vinereanu et al. ***$P < 0.001$.

Fig. 5 Peak early and atrial (late) velocities in athletes (A) versus controls (C). Data are expressed as $\bar{x} \pm \sigma$. Adler et al. reported significantly greater blood flow velocities in athletes than controls. ***$P < 0.0001$.
significantly greater in weightlifters (1.80 ± 0.2 for weightlifters versus 1.40 ± 0.20; *P* < 0.001 and 2.38 ± 0.16 versus 1.99 ± 0.10; *P* < 0.001), indicating a more efficient mechanism of LV filling. The ECG equipment used throughout the studies was similar, with 2.5,15,17,18 and 2.5–4.0 MHz7 transducers. Methods employed often lacked specific details, yet some explicitly stated working in accordance with American Society of Echocardiography with regard to BP,17,18 LVM4,7,15, EF,7,15 LV volumes/diameters,4,7,15,16 and ST and PWT.4,7,16

Only three studies7,16,18 mention the use of blinding during data collection, using two independent observers to measure variables. No other studies mentioned such practices, thus potentially introducing observer bias within results.

The methods of group comparison were appropriate, with all studies stating details of tests employed to establish correlations within and between groups—some variation, however, exists in reporting specificities. Given the nature of study designs (Table 2), randomization did not take place.

**Conclusion**

BP does not vary greatly within the populations, a difference recognized only by one study.16 An improved cardiac efficiency has been demonstrated through widespread reports of decreased RHR7 and increased SV amongst weightlifters.16–18 The majority of studies
investigating the systolic function through EF and FS have portrayed no significant differences, although one study demonstrated an increased FS to a significant degree.\(^7\) \((P < 0.01)\).

Studies reported significantly increased LVM\(^4,7,16\) and ST\(^7,15\) and PWT demonstrated widespread evidence to a significantly increased degree in weightlifters.\(^7,15,17,18\) No differences were observed in LV volumes and dimensions in either phase of the cardiac cycle. This proposes that hypertrophy does not occur in a consistent, homogenous way, and studies must focus into the causative factors for regional morphological change. There may be several factors causing this discrepancy, which include the use of anabolic/androgenic substances\(^31\) to impair both the systolic and diastolic function; the requirement of long-term assessment of training volume and genetic variance within weightlifters. Additionally, the error margin of equipment used may have influenced attaining reliable readings. Ideally, MRI provides a valid means of measuring many variables.\(^20\)

LV filling abilities within one study\(^16\) portray a quickened filling ability, with a higher proportion of filling occurring in diastole, rather than in early systole. Vinereanu et al. reported no differences between two related variables.

Further investigations are necessary to fully understand the mechanisms involved in LV adaptation. Nevertheless, weightlifting seems to confer beneficial attributes to the individual in terms of the cardiac function, perhaps dispelling previous myths associated with the topic.

References


