Left Ventricular Pressure–Volume and Frank-Starling Relations in Endurance Athletes
Implications for Orthostatic Tolerance and Exercise Performance

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Background. Endurance athletes have a high incidence of orthostatic intolerance. We hypothesized that this is related to an abnormally large decrease in left ventricular end-diastolic volume (LVEDV) and stroke volume (SV) for any given decrease in filling pressure.

Methods and Results. We measured pulmonary capillary wedge (PCW) pressure (Swan-Ganz catheter), LVEDV (two-dimensional echocardiography), and cardiac output (C2H2 rebreathing) during lower body negative pressure (LBNP, −15 and −30 mm Hg) and rapid saline infusion (15 and 30 ml/kg) in seven athletes and six controls (VO2max, 68±7 and 41±4 ml/kg/min). Orthostatic tolerance was determined by progressive LBNP to presyncope. Athletes had steeper slopes of their SV/PCW pressure curves than nonathletes (5.5±2.7 versus 2.7±1.5 ml/mm Hg, p<0.05). The slope of the steep, linear portion of this curve correlated significantly with the duration of LBNP tolerance (r=0.58, p=0.04). The athletes also had reduced chamber stiffness (increased chamber compliance) expressed as the slope (k) of the dP/dV versus P relation (chamber stiffness, k=0.008±0.004 versus 0.031±0.004, p<0.005; chamber compliance, 1/k=449.8±283.8 versus 35.3±4.3). This resulted in larger absolute and relative changes in end-diastolic volume over an equivalent range of filling pressures.

Conclusions. Endurance athletes have greater ventricular diastolic chamber compliance and distensibility than nonathletes and thus operate on the steep portion of their Starling curve. This may be a mechanical, nonautonomic cause of orthostatic intolerance. (Circulation 1991;84:1016–1023)

Endurance training results in central and peripheral adaptations that markedly improve an individual’s ability to perform physical work. Athletes are thus commonly assumed to be able to tolerate many kinds of physiological stressors better than nonathletes. Gravitational stress, or orthostasis, however, may be one condition during which being an endurance athlete may be a disadvantage.1,2

Tolerance to orthostatic stress requires the successful integration of multiple elements of cardiovascular control. Orthostatic hypotension results when there is an abnormally large postural decrease in central blood volume, cardiac filling pressures, and stroke volume (SV), or inadequate neurohumoral reflex responses to orthostasis, primarily tachycardia and vasoconstriction.3 Orthostatic intolerance has been reported to be particularly prevalent in endurance-trained athletes1,2 and has been attributed primarily to abnormalities in baroreflex regulation of heart rate4 and peripheral resistance.5,6 However, a potentially important but previously unrecognized mechanism for orthostatic intolerance in athletes might also be related to structural or mechanical adaptations in the cardiovascular system.

We have recently demonstrated that individuals with a large resting SV have a disproportionately large decrease in SV during lower body negative pressure (LBNP) and that these characteristics may be important features of orthostatic intolerance in endurance athletes.7 Furthermore, studies of the effect of volume loading on SV and cardiac output...
indicate that individuals with the largest SV at rest have the greatest increase in SV after loading. These observations suggest that endurance training, which delivers an acute volume load to the heart during exercise, may alter the compliance characteristics of the left ventricle. Such an adaptation may be very beneficial during exercise, allowing for increased diastolic reserve and therefore increased SV and cardiac output in response to increased filling pressures. This adaptive response, however, may be detrimental during decreases as opposed to increases in central blood volume and cardiac filling. We postulated that athletes have a steeper ventricular function and flatter diastolic pressure-volume curve than sedentary subjects over the physiological range of filling pressures. During orthostasis, this characteristic results in an abnormally large decrease in ventricular volume when filling pressure is reduced. To test this hypothesis, we examined left ventricular pressure-volume relations and Starling curves relating left ventricular end-diastolic pressure (LVEDP) to SV in endurance-trained athletes compared with sedentary controls and compared differences in these relations to differences in orthostatic tolerance.

Methods

Subjects

Subjects were 13 healthy male volunteers in whom clear images could be obtained by screening two-dimensional echocardiography. Subjects were non-obese nonsmokers less than 40 years of age and were recruited and selected into two groups based on current level of endurance training and maximal aerobic power (VO2max). Endurance athletes ran at least 50 miles per week or bicycled at least 250 miles per week for at least 3 years (VO2max >60 ml/kg/min; n=7); sedentary controls performed no regular exercise (VO2max <45 ml/kg/min; n=6).

The study was approved by the University of Texas institutional research review committee, and informed consent was obtained from all subjects before the study.

Aerobic Power

VO2max was determined using a continuous incremental protocol on a treadmill. After a 5-minute warm-up period, subjects began running at an individualized speed (6–12 mph) and 0% grade. The grade was subsequently increased by 2% every minute until an increase in work load produced no further increase in VO2. Gas concentrations and ventilation were determined on a breath-by-breath basis using a mass spectrometer (Perkin-Elmer) and turbine flow meter (SensorMedics), respectively, and interfaced with a dedicated AT&T computer for calculation of VO2.

Plasma volume was measured at a separate session after 30 minutes of supine rest using Evans blue indicator dilution. A blood sample was also drawn through a peripheral vein and analyzed in triplicate for hematocrit (HCT) by the microcapillary centrifuge technique (Damon). Plasma volume was divided by (1–HCT) to give a measure of blood volume, applying customary corrections for peripheral sampling (0.92) and red cell trapping of plasma (0.98).

Ventricular Filling Pressure and Cardiac Output

The mean pulmonary capillary wedge (PCW) pressure was used as a measure of LVEDP. A 6F balloon-tipped, flow-directed pulmonary arterial catheter (Swan-Ganz, American Edwards Laboratories) was placed percutaneously from a right brachial vein and advanced under fluoroscopic guidance into the PCW position. Correct position of the catheter was confirmed fluoroscopically and by the presence of characteristic pressure waveforms. The catheter was connected to a physiological pressure transducer (Gould-Statham) with the zero reference point set at 5 cm below the sternal angle. The signal was passed through an amplifier (Hewlett-Packard 8805B) to an inkjet recorder (Mingograph) calibrated to display 1 mm Hg/2 mm paper. Mean PCW pressure was determined visually at end expiration.

To minimize perturbation of the system being evaluated by frequent injections of saline, cardiac output was measured with a standard inert gas rebreathing technique using acetylene as the soluble gas and helium as the insoluble gas. Adequate mixing of rebreathing gas in the lung was confirmed by a constant level of helium in all cases. This technique has been described previously from our laboratory and has been validated against both green dye and thermodilution methods in normal subjects and in patients with significant cardiopulmonary disease. Heart rate was measured and averaged over 20 seconds during the rebreathing, and SV was then calculated.

Ventricular filling was manipulated using a sequence of LBNP and rapid saline infusion as shown in Figure 1. LBNP was accomplished by placing the subjects in a cylindrical metal tank sealed at the level of the iliac crest. Suction was provided by a vacuum pump and controlled with a regulator calibrated against a mercury manometer. Measurements of PCW pressure and SV were made after at least 30 minutes in the supine position at rest and after 5 minutes each at −15 mm Hg and −30 mm Hg LBNP. After the LBNP and at least a 15-minute rest period, resting measurements were repeated to ensure return to baseline condition. Ventricular filling was then increased using a rapid infusion of warm isotonic saline (37°C, 10 min/l). The saline was infused through the proximal port of the Swan-Ganz catheter and through an 18-gauge peripheral i.v. placed in the opposite antecubital vein. Measurements of PCW pressure and SV were again made after 15 ml/kg and 30 ml/kg saline were infused.

Ventricular Volumes

Left ventricular volumes were determined using two-dimensional echocardiography. Images were obtained from standard windows using a phased-array
transducer with a frequency of 2.5 or 3.5 MHz (Hewlett-Packard 77020A Ultrasound Imaging System). Data were collected on videotape and played back on a videotape recorder (Panasonic AG-6200). The images were digitized on an image processing and quantitation system (MICROSONICS CAD886, Version 2.5). A continuous-loop presentation was used to capture the best cardiac cycles for analysis. Two or three beats were averaged during steady-state hemodynamic conditions, with the subject pausing during the respiratory cycle at end expiration.

Measurements were made of the left ventricular area from the parasternal short-axis window at the level of the mitral valve and papillary muscles, and major and minor axes lengths were obtained from the short-axis and apical long-axis views. Volumes were then calculated in biplane format using the MICROSONICS software by modeling the left ventricle as a prolate ellipsoid (length-area method) using standard formulas as recommended by the American Society of Echocardiography. All echocardiographic studies were analyzed by a skilled technician unaware of fitness group or the purpose of the study. In our hands, echocardiographically derived measurements of left ventricular end-diastolic volume (LVEDV) have an interobserver variability of 10%.

Furthermore, validation studies within our laboratory comparing echocardiographic volumes with biplane angiography at cardiac catheterization in 30 subjects demonstrated an $r$ value of 0.90 with a standard error of the mean of 26 ml.

**Orthostatic Tolerance**

Tolerance to LBNP, which decreases central blood volume in a graded fashion, was used as a measure of orthostatic tolerance. At a separate session, at least 48 hours after but within a week of the Starling curve determination, the subjects were placed in the LBNP tank, and the suction was increased in a stepwise fashion according to the following protocol: -15 mm Hg×15 minutes, -30 mm Hg×5 minutes, -40 mm Hg×15 minutes, and -55 mm Hg (the maximum suction generated by our device)×30 minutes. This protocol has been used in previous studies of cardiovascular regulation following head-down tilt and allows data collection at both high and low levels of LBNP as well as a test of maximal tolerance. LBNP was discontinued if the subject developed signs or symptoms of presyncope (sudden onset of nausea, sweating, lightheadedness, bradycardia, or hypotension) or a sustained decrease in systolic blood pressure below 90 mm Hg. An index of tolerance, LBNP×time, was calculated by summing the product of pressure and duration at each level of LBNP.

**Statistics**

We performed linear regression on the individual data to identify the slope of the relation between the change in PCW pressure and the change in SV during both LBNP and saline infusion. The slopes for the subjects were then grouped and the means compared separately for both a decrease and increase in left ventricular filling using the unpaired $t$ test. This strategy was determined a priori. We also compared the relation between end-diastolic volume and SV between groups using a repeated-measures analysis of covariance with a time-varying covariate (BMDP 5V, BMDP Statistical Software Inc., Los Angeles). The relation between the slope of the linear portion of the PCW pressure/stroke volume curve during decreased left ventricular filling and LBNP×time was determined by linear regression with Pearson's correlation.

Pressure-volume curves were analyzed by modeling the relation according to the exponential equation \( P = ae^{V} + b \), where \( P \) is PCW pressure, \( V \) is LVEDV, and \( a \), \( b \), and \( k \) are constants. Curve fitting was performed with the Marquardt-Levenberg algorithm using commercially available software (SIGMA-PLT 4.04, Jandel Scientific, Corte Madera, Calif.). The constant \( k \) then represents the slope of the relation describing the instantaneous change in pressure for a change in volume at any given pressure (dP/dV versus \( P \)), and was used as an estimate of chamber stiffness. We also calculated the inverse of \( k \) to provide an index of chamber compliance. Curve-fitting parameters were calculated for each individual
curve, and the means were compared between groups using the unpaired t test.

Hemodynamic variables were compared using a repeated-measures analysis of variance with Scheffe’s post hoc for multiple comparisons.

Results

Subject Characteristics

The subjects were closely matched for age, height, and weight (Table 1). LBNP tolerance was significantly lower in the athletes compared with the nonathletes: 1,345 mm Hg min ± 668 versus 2,516 ± 881 mm Hg min (p < 0.02). Six of seven athletes had presyncopal reactions during maximal LBNP testing compared with one of six nonathletes. The athletes had larger VO₂max by design (68.4 ± 6.6 versus 40.8 ± 4.4 ml/kg/min) as well as larger LVEDV, SV, and plasma/blood volumes.

Starling Curves (PCW Pressure Versus SV)

Representative pressure tracings from one subject (Figure 2) demonstrate that with our techniques of LBNP and saline infusion, we were able to vary PCW pressure over a wide physiological range of almost 20 mm Hg. Hemodynamic conditions associated with this change in cardiac filling are provided in Table 2. The relations between PCW pressure and SV for the mean group data (Figure 3) demonstrate two important points. First, for any given filling pressure, the athletes had a greater stroke volume than the nonathletes. Second, they also had a greater change in stroke volume with any change in central blood volume and cardiac filling pressure, as manifested by the slope of the steep linear portion of these curves. This slope, determined over a range of filling pressures from 1 to 13 mm Hg by linear regression (mean r value of 0.92 ± 0.03 for all subjects), was significantly steeper in the athletes at 5.5 ± 2.7 ml/mm Hg versus 2.7 ± 1.5 ml/mm Hg in the nonathletes (p = 0.03). The functional consequence of this concept (Figure 4) is that a decrease in PCW, for example, from 10 to 5 mm Hg as might occur going from supine to upright posture, would be associated with a 27-ml decrease in SV in the athletes (25% of baseline), nearly twice the 14-ml decrease in SV that would be observed with the same stress in the nonathletes (17% of baseline). The magnitude of this decrease in SV appeared to play an important role in determining LBNP tolerance because the slope of the steep linear portion of the PCW pressure versus SV curve correlated significantly with LBNP x-time (Figure 5; r = 0.58, p = 0.04).

The relation between LVEDV and SV was determined as linear for both groups (r = 0.97 for athletes, 0.65 for nonathletes), with the athletes shifted to higher values of LVEDV and SV than the nonathletes. Analysis of covariance could not distinguish between the slopes of these relations, however, suggesting that the athletes had greater filling but similar left ventricular systolic function measured as ejection fraction compared with the nonathletes.

Pressure–Volume Curves

The mean grouped data relating LVEDV to PCW pressure are shown in Figure 6. The curve for the athletes is shifted downward and to the right; therefore for any given end-diastolic volume, the athletes had a much lower PCW pressure than the nonathletes, suggesting greater chamber compliance and distensibility. This observation is confirmed statistically by fitting the data to the exponential curve P = aeᵇ + b. For the athletes, the mean curve was P = 0.023e⁰.⁰⁰３V + 0.394; for the nonathletes it was P = 0.035 ᵇ e⁻⁰.⁰³V - 2.926. The individual chamber stiffness constant k was significantly less (reflecting
greater chamber compliance) in the athletes compared with the nonathletes (chamber stiffness, $k=0.008 \pm 0.004$ versus $0.031 \pm 0.004$, $p<0.005$; chamber compliance, $1/k=449.8 \pm 283.8$ versus $35.3 \pm 4.3$, $p<0.05$). Thus, they had larger absolute (64 versus 14 ml) and relative (30% versus 9% of baseline) changes in end-diastolic volume over the range of central blood volume induced by LBNP and saline loading.

**Discussion**

This study provides new information regarding cardiovascular regulation in endurance athletes in two important ways. First, athletes have more compliant, distensible ventricles and therefore a steeper

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**TABLE 2.** Hemodynamics

<table>
<thead>
<tr>
<th></th>
<th>Heart rate (beats/min)</th>
<th>Blood pressure (mm Hg)</th>
<th>Stroke volume (ml)</th>
<th>TPR (dyne·sec·cm⁻²)</th>
<th>Cardiac output (l/min)</th>
<th>Pulmonary capillary wedge pressure (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Athletes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Rest</td>
<td>53±3</td>
<td>88±1</td>
<td>113±4</td>
<td>1,204±6</td>
<td>5.97±0.32</td>
<td>10.1±0.8</td>
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<td>−15 mm Hg LBNP</td>
<td>55±3</td>
<td>89±1</td>
<td>95±6*</td>
<td>1,399±100</td>
<td>5.31±0.48</td>
<td>6.4±0.7†</td>
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<td>−30 mm Hg LBNP</td>
<td>61±5</td>
<td>79±7‡</td>
<td>74±4‡</td>
<td>1,449±172‡</td>
<td>4.53±0.19‡</td>
<td>3.4±0.7†</td>
</tr>
<tr>
<td>Repeat baseline</td>
<td>54±3</td>
<td>90±1</td>
<td>111±4</td>
<td>1,218±61</td>
<td>5.94±0.22</td>
<td>10.3±0.6</td>
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<td>93±2</td>
<td>130±5†</td>
<td>1,015±77</td>
<td>7.59±0.59†</td>
<td>15.4±1.1†</td>
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<td>61±4</td>
<td>93±2‡</td>
<td>131±5</td>
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<td>955±67§</td>
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<tr>
<td>Before max LBNP test</td>
<td>56±4</td>
<td>84±3</td>
<td>110±6</td>
<td>1,112±55</td>
<td>6.07±0.14</td>
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<tr>
<td><strong>Nonathletes</strong></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>63±4</td>
<td>83±1</td>
<td>77±4</td>
<td>1,399±73</td>
<td>4.81±0.29</td>
<td>10.5±0.6</td>
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<tr>
<td>−15 mm Hg LBNP</td>
<td>62±4</td>
<td>85±2</td>
<td>78±7</td>
<td>1,467±85</td>
<td>4.72±0.33</td>
<td>7.5±0.5†</td>
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<td>−30 mm Hg LBNP</td>
<td>69±5</td>
<td>84±3</td>
<td>63±6§</td>
<td>1,582±71§</td>
<td>4.50±0.50§</td>
<td>5.1±0.7†</td>
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<td>84±2</td>
<td>84±6</td>
<td>1,256±101</td>
<td>5.46±0.39</td>
<td>10.3±0.5</td>
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<td>71±3</td>
<td>85±2</td>
<td>98±6†</td>
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<td>87±2</td>
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<td>65±4</td>
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<td>83±4</td>
<td>1,231±112</td>
<td>5.39±0.39</td>
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</tr>
</tbody>
</table>

TPR, total peripheral resistance; LBNP, lower body negative pressure. Values are mean±SEM.

*p<0.10; †p<0.01 vs. rest; ‡p<0.05; §p<0.01 for comparison between −30 mm Hg LBNP and 30 ml/kg saline (if no difference from rest); ||p<0.05.

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**FIGURE 3.** Mean grouped data ± SEM, demonstrating the Starling curves relating pulmonary capillary wedge pressure to stroke volume in athletes and nonathletes. The mean curves are representative of the individual data. Lines are computer fits of the best polynomial regression through the data.

**FIGURE 4.** Individual data points represent the decrease in stroke volume that would be seen in each subject if pulmonary capillary wedge (PCW) pressure decreased from 10 to 5 mm Hg, similar to what might occur with standing. The athletes had a significantly greater fall in stroke volume for the same decrease in filling pressure ($p<0.05$). Two athletes had the same fall in stroke volume, and their points appear superimposed.
slopes of the Frank-Starling curve relating left ventricular filling pressure to stroke volume than nonathletes. Second, the slope of the steep linear portion of this relation is an independent predictor of orthostatic intolerance, and thus may play a role in explaining the increased prevalence of orthostatic hypotension in well-trained endurance athletes with high aerobic power.

The relation between LVEDP and SV describes one form of Starling’s law of the heart. In normal sedentary supine men, the left ventricle appears to operate near the peak or plateau of this hyperbolic curve at an LVEDP of approximately 10 mm Hg. Thus, with exercise in the supine position, diastolic reserve is limited and virtually completely utilized at lower levels of exercise. Studies examining exercise responses in untrained subjects using radionuclide techniques suggest that in such individuals, the Frank-Starling mechanism is important in increasing SV and cardiac output primarily at submaximal levels of exercise. As maximal exercise loads are approached, increasing heart rate and contractility become the predominant mechanisms for meeting further increases in metabolic demand. The upright position, by precipitating venous pooling and decreasing central blood volume, appears to shift the heart to the flatter portion of the diastolic pressure-volume curve and the steeper portion of the LVEDP/SV curve, allowing a greater increase in LVEDV and SV during submaximal exercise. However, ventricular distension is still limited at peak exercise.

On the other hand, elite athletes appear to have an increased capacity to utilize the Frank-Starling mechanism to increase SV during exercise. This increase in SV during upright exercise is due primarily...
to an increase in end-diastolic volume, which in athletes is accompanied by an increase in myocardial mass to maintain a normal relation between cavity size and wall thickness. There is little evidence that human intrinsic ventricular systolic performance is altered by endurance training.

The present study extends these observations by demonstrating a general physiological mechanism for enhancing cardiac pump performance. By functioning on the steep portion of the Starling curve, endurance athletes have increased diastolic reserve and a greater change in SV for a given change in filling pressure than untrained individuals. This difference results from increases in chamber compliance or distensibility as reflected by the dP/dV of the diastolic pressure–volume relation in the athletes.

How might this adaptation occur? Numerious studies have shown a consistent linear relation between VO₂ and cardiac output. Furthermore, Ekelund and Holmgren demonstrated a linear relation between cardiac output and PCW pressure both in athletes and sedentary individuals. While such cross-sectional studies do not allow a clear distinction between an innate characteristic of successful athletes and the end result of long-term, intensive endurance training, it seems likely that exercise in athletes, who train at high VO₂ and cardiac output, imposes a prominent volume load on the heart, causing elevated filling pressures during training and resulting in an increase in end-diastolic volume.

Acutely, the limit to ventricular dilation is probably dependent on both the compliance characteristics of the myocardium and pericardial or pulmonary mechanical restraint. In fact, removing the pericardium in dogs increases end-diastolic volume during exercise, increases maximal cardiac output, and therefore increases maximal aerobic power. It is difficult to sort out the relative contributions of myocardial compliance and pericardial restraint to limiting ventricular filling during exercise. However, the present study strongly suggests that the chronic volume loading of endurance training results in increased effective ventricular compliance and alters the pressure–volume relation and therefore Starling (SV/LVEDP) relation of the athlete’s heart. Such an increased chamber compliance and steep Starling curve is teleologically very beneficial to an athlete, facilitating the delivery of large volumes of blood to exercising skeletal muscle. However, it may be a distinct disadvantage during orthostasis, resulting in a large decrease in SV when filling pressure is reduced. In the present study, this hypothesis is supported by the observation that the steepness of the LVEDP/SV curve correlates significantly with tolerance to LBNP.

The issue of orthostatic intolerance in endurance athletes has been a controversial one since Stegmann et al first described depressed carotid baroreflex sensitivity in a group of trained athletes. Subsequent work has focused almost exclusively on the baroreflex regulation of heart rate (HR) and peripheral resistance (TPR), and has recently been reviewed. In our laboratory, we have recently emphasized the equal importance of SV in the “triple product” of blood pressure control (HR×SV×TPR). We demonstrated a large decrease in SV during LBNP in individuals with orthostatic intolerance (primarily athletes) and suggested that SV might be an important independent factor not under the exclusive control of the autonomic nervous system. The present study does not exclude autonomic influences on ventricular filling and contractility but does show that adaptations affecting mechanical diastolic properties of the athlete’s heart can explain a significant component of the orthostatic intolerance seen in this group. Furthermore, this ventricular adaptation would tend to magnify the effects of any impairment of baroreflex control of heart rate and peripheral resistance.

There are potential limitations to the present study that must be considered in interpreting the results. As has been pointed out by Nobel, the Starling curve relating LVEDP to SV may not actually describe a pressure–volume relation and may more precisely reflect the constancy of end-systolic volume for a given ejection pressure. However, the shape of this form of the Starling ventricular function curve depends critically on the configuration of the diastolic pressure–volume relation, which in turn is a function of the length-tension relation of individual sarcomeres. Furthermore, SV remains an almost constant fraction of end-diastolic volume throughout a wide range of left ventricular filling pressures. In the present study, we constructed both curves—SV as a function of PCW pressure using an acetylene rebreathing method to measure cardiac output, and PCW pressure as a function of LVEDV using two-dimensional echocardiography. The confirmation of the same phenomenon—that athletes have large changes in end-diastolic volume and SV with changes in left ventricular filling—by two independent techniques (echocardiography and C₂H₂ rebreathing) argues strongly that we are identifying a true physiological characteristic of endurance athletes.

We must emphasize that the maintenance of blood pressure during orthostatic stress depends on multiple components of cardiovascular regulation, including baroreflex control of heart rate and peripheral resistance. The relatively weak though statistically significant negative relation between the slope of the linear portion of the end-diastolic pressure/SV curve and LBNP tolerance argues for the importance of these other variables in predicting individual orthostatic tolerance. The present study thus provides one piece of the puzzle and complements previous investigations of blood pressure control in athletes by identifying nonautonomic control of SV as an important variable.

We have demonstrated that endurance athletes have greater effective left ventricular diastolic chamber compliance and distensibility than nonathletes and thus have a steeper slope of their Starling curve relating left ventricular filling pressure to SV. The
slope of this relation correlated significantly with LBNP tolerance, suggesting an important role for mechanical, nonautonomic factors in the orthostatic intolerance observed in endurance athletes.

Acknowledgments

We gratefully acknowledge the technical assistance of Mr. Willie Moore and the secretarial assistance of Mrs. Carolyn Donahue. We also must thank Dr. Jim Stray-Gundersen, Ms. Andrea Katz, and Mr. Wyman Schultz for performing the exercise tests and Ms. Debra Epstein and Ms. Margaret Morin for analyzing the echocardiograms. Rick Risser provided invaluable statistical consultation. We are indebted to Drs. Michael Zile and Eric Eichorn for their insight into the analysis of our diastolic pressure–volume curves and Drs. Ron Victor and F. Andrew Gaffney for their thoughtful reviews of the manuscript.

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KEY WORDS • Frank-Starling relation • orthostatic intolerance • athletes • pressure–volume relation • chamber stiffness, compliance
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Circulation. 1991;84:1016-1023
doi: 10.1161/01.CIR.84.3.1016

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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