Effect of Supine Exercise on Left Ventricular Volume and Oxygen Consumption in Man

By Richard Gorlin, M.D., Lawrence S. Cohen, M.D., William C. Elliott, M.D., Michael D. Klein, M.D., and Francis J. Lane, M.D.

RESPONSE to physical exercise in man has been studied extensively. For any given workload, a predictable change in blood pressure, heart rate, and cardiac output has been shown to occur. Rushmer\(^1\) has pointed out that the response of stroke volume to exercise, however, may be variable both in experimental animals and in man. He has also demonstrated that not only the amount, but also the direction of change in cardiac dimensions may vary in the exercising dog, thus indicating that the pattern of response cannot be readily predicted. In a preliminary report using radiocardiography, Cournand and associates\(^2\) demonstrated that the heart emptied more completely during physical exercise. In a recent study measuring cardiac dimensions by means of silver clips sewn on the heart of four postoperative subjects, Braunwald and associates\(^3\) showed that these dimensions diminished on the average of 3 to 5 per cent during supine leg exercise. It is the purpose of this report to present data obtained in 20 human subjects with minimal heart disease on the change induced by supine leg exercise in left ventricular volume, in the degree and speed of ventricular emptying, and to describe the relationship of this mechanical activity to myocardial oxygen consumption.

Case Material

Twenty patients were classified on the basis of clinical evidence and diagnostic cardiac catheterization. Ten subjects had no discernible heart disease; six had mitral stenosis of moderate degree with valve areas ranging from 1.2 to 2.2 cm.\(^2\); one patient had a small secundum-type atrial septal defect with a pulmonary:systemic blood flow ratio of 1.2:1; two patients had moderate, asymptomatic aortic stenosis with valve areas of 1.2 and 1.5 cm.\(^2\), respectively; one subject had mild systolic hypertension without recognizable cause. All were in sinus rhythm, and none had left ventricular hypertrophy or enlargement.

Methods

Catheters were placed in the left ventricle by either the retrograde or transseptal technic, and in the ascending aorta and a brachial artery. A beaded thermistor mounted on a radiopaque no.-4 vinyl catheter was threaded through the aortic catheter so as to protrude several millimeters into the blood stream immediately above the aortic valve. The thermistor was connected across a wheatstone bridge and thence to a recording circuit. Pressures were recorded via Statham P-23D strain-gages on a Sanborn no.-150 direct writer. Duplicate indocyanine green (dye) cardiac output determinations were made by injection into the left ventricle with sampling at a brachial artery. One output was carried out at the onset and the other at the termination of the aortic thermodilution curves. These outputs agreed within 9 per cent. In each state 5 to 15 thermodilution curves were measured by injecting 2 to 5 ml. of cool saline into the left ventricle (fig. 1). The relative change in temperature on successive beats \(\frac{T_n}{T_{n-1}}\) was obtained as an average of steps beginning with the fourth beat following indicator injection (to allow indicator mixing).\(^5\) In each instance the minimum number of ratios utilized was that required to yield a mean ratio within ±5 per cent of the true mean ratio (at 95 per cent confidence).\(^6\)

As described elsewhere,\(^4\) the data were analyzed to derive end-diastolic and end-systolic volumes, and end-systolic to end-diastolic volume ratios.

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End-diastolic and mean systolic forces per beat (dyne) were calculated as follows:

\[ F_e = P_e \times (r_e^2) \times \frac{2}{P_m} \times \frac{2}{\pi} \]

where \( P_e \) = left ventricular end-diastolic pressure (mm. Hg)
\( P_m \) = left ventricular systolic mean pressure (mm. Hg)
\( r_e \) = end-diastolic radius squared (cm.²)
\( \frac{2}{P_m} \) = mean index (cm.²)
\( \frac{2}{\pi} \) = mean factor to degrees

Pressure time per beat (PTB) and force-time per beat (FTB) were calculated as follows:

\[ PTB = P_m \times \text{sep} \times 1,332 \] dynes

where \( \text{sep} \) = systolic ejection period (sec./beat)

\[ FTB = PTB \times \frac{1,332}{\pi} \times \frac{r_m^2}{P_m} \] dynes

Mean systolic shortening distance (D) and rate (R) were calculated as follows:

\[ D_m = \frac{2}{r_m} \left( \frac{r_e}{r_m} - \frac{r_e}{r_e} \right) \]
\[ R_m = \frac{2}{\pi} \left( \frac{r_e}{r_m} - \frac{r_e}{r_e} \right) \times \text{sep} \]

where \( r_m \) = end-systolic radius (cm.)

External left ventricular work (Kg.M/min./M.²) was calculated by standard methods. Contractile element work index [⁷] was calculated (Kg.M/min./M.²):

\[ \frac{13.6 \times \text{HR} \times P_m \times (SV + \frac{V_m}{9.6})}{1,000} \]

where \( \text{HR} \) = heart rate (per min.)
\( SV \) = stroke volume (ml./M.²)
\( V_m \) = left ventricular systolic mean volume (ml./M.²)

The ratio of external to total* work (contractile element work index) was calculated.

In 10 of the 20 patients, catheters had been placed in the coronary sinus. Coronary flow was measured by the krypton-85 method [⁸] (ml./100 Gm. left ventricle/min.), and myocardial arteriovenous oxygen difference was determined manometrically (ml./L). Myocardial oxygen consumption (ml./100 Gm. left ventricle/min.) was calculated as the product of these two measurements. External myocardial efficiency was calculated as described elsewhere [⁹]. The ratios of force-time and contractile element work index to oxygen consumption per beat were calculated.

Observations were made at rest and then repeated during the second 5 minutes of a 10-minute exercise state, at a time when blood pressure and heart rate had become stable. All measurements were accomplished within 2 minutes when only volumes were determined, and within a 5-minute period when both ventricular volumes and coronary flows were measured. Exercise was performed in the supine position with a bicycle ergometer, at a rate sufficient to increase heart rate approximately 150 per cent of control and total body oxygen consumption 250 per cent of control.

Results

Left Ventricular Volume

Results are shown in table 1 and figures 1.

*External work represents useful work done by composite fiber (contractile element in series with an elastic component). Total work represents work done by the contractile element itself and includes internal work done stretching the series elastic as well as external or fiber shortening work.
and 2. When data from all 20 patients were analyzed there was no significant change in end-diastolic volume, while both the end-systolic fraction and end-systolic volume decreased significantly \((p < 0.001\) and \(p < 0.025\), respectively). Because prior studies in dogs\(^1\) had indicated a high individual variability of response of cardiac dimensions to exercise, the 20 patients were divided into three groups according to change in end-diastolic volume, to see if different patterns of response could be recognized.\(^4\) Seven patients showed less than 10 per cent change of end-diastolic volume from control; eight showed a significant decrease \((p < 0.005)\); and five had a significant increase \((p < 0.025)\). Such a subdivision revealed other differences between the groups (table 2). Stroke volume was augmented significantly more in the group with increased end-diastolic volume when compared with the other two subgroups \((0.025 < p < 0.05\)–double tail distribution of \(T)\). As a result, those with increased end-diastolic volumes during exercise had the greatest increase in cardiac output over control. This occurred despite a much less rise in heart rate. Thus, these subjects (group A) differed from the others by virtue of end-diastolic volume, stroke volume, heart rate, and cardiac output during exercise. No particular type of subject by diagnosis dominated any subgroup.

### Left Ventricular Pressure

Average systolic pressure rose 14 per cent and heart rate 47 per cent. Left ventricular

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**Table 1**

<table>
<thead>
<tr>
<th>Age</th>
<th>BSA* (M²)</th>
<th>Pressure (mm Hg)</th>
<th>Volume ml/M²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Systolic mean</td>
<td>End-diastolic</td>
</tr>
<tr>
<td>Rest</td>
<td>Mean</td>
<td>109</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>13</td>
<td>0.24</td>
</tr>
<tr>
<td></td>
<td>Mean</td>
<td>125</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>SD</td>
<td>20</td>
<td>3.4</td>
</tr>
<tr>
<td></td>
<td>P value</td>
<td>&lt;0.001</td>
<td>NS</td>
</tr>
</tbody>
</table>

*BSA = body surface area in meters square.

\(*P* values were derived by the “t” test, analyzing the average of the paired differences for each value by the null hypothesis.

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\(^1\)That the variability was not methodologic was suggested by two considerations. Rushmer’s studies on end-diastolic circumference in the exercising dog showed great variability in response.\(^1\) Other studies of left ventricular volume in man, by contrast, show more consistent changes for any group of subjects to any given intervention\(^4\) than reported herein.

Circulation, Volume XXXII, September 1965
<table>
<thead>
<tr>
<th>End-systolic fraction</th>
<th>Heart rate, per min.</th>
<th>Mean systolic force, cm., dyne x 10^6</th>
<th>Mean systolic force, cm., dyne x 10^6</th>
<th>End-diastolic force, cm., dyne x 10^6</th>
<th>Mean systolic shortening, (cm.)</th>
<th>Rate, (cm./sec.)</th>
<th>External work, (Kg./min./M.²)</th>
<th>Ejection period, (sec./beat)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.58 0.065 0.52 0.095</td>
<td>77 11 114 15</td>
<td>3.0 0.29 2.85 0.37</td>
<td>162 44 167 59</td>
<td>13 6 13 9</td>
<td>3.5 12.5 4.4 1.2</td>
<td>0.7 2.6 17.3 8.0</td>
<td>0.28 0.025 0.24 0.29</td>
<td></td>
</tr>
<tr>
<td>&lt;0.001 NS NS NS</td>
<td>&lt;0.001 NS NS NS</td>
<td>&lt;0.005 &lt;0.001 &lt;0.001 &lt;0.005</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.58 0.51 -0.05 0.58 0.53 &lt;0.025 0.59 0.51</td>
<td>73 104 &lt;0.001 85 117 &lt;0.001 75 117</td>
<td>3.1 3.0 &lt;0.1 3.0 3.0 NS</td>
<td>190 220 0.05 170 180 &lt;0.001 140 130</td>
<td>16 21 0.05 11 12 0.001 13 10</td>
<td>3.8 13.7 4.8 10.3 NS</td>
<td>13.7 18.3 10.3 0.25 NS</td>
<td>4.8 13.3 4.8 0.24 NS</td>
<td>0.27 0.25 0.26 0.24</td>
</tr>
<tr>
<td>&lt;0.001 NS NS</td>
<td>&lt;0.001 NS NS</td>
<td>&lt;0.001 &lt;0.005 &lt;0.005 NS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.59 0.51</td>
<td>75 117</td>
<td>2.9 2.6</td>
<td>140 130</td>
<td>13 10</td>
<td>3.3 3.8</td>
<td>11.1 16.3</td>
<td>3.7 6.4</td>
<td>0.30 0.23</td>
</tr>
<tr>
<td>&lt;0.001 &lt;0.001</td>
<td>&lt;0.001 &lt;0.001</td>
<td>&lt;0.001 &lt;0.001</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**RESPONSES OF EDV TO EXERCISE**

**Figure 2**

Left ventricular volume at rest and during exercise. The responses are subdivided into groups A (increased end-diastolic volume response to exercise); B (end-diastolic pressure was unchanged for the total group, and although average directional changes in pressure were similar to average change in end-diastolic volume in the three subgroups, these alterations were not significant. Figure 3 shows a plot of end-diastolic pressure versus diastolic volume. There were 12 concordant and four discordant directional responses. In four subjects the change in either value was too small to be properly assessed.

**Left Ventricular Force**

Systolic mean force showed essentially no change except in two of the five patients with increased end-diastolic volume (fig. 4). Changes in end-diastolic tension likewise were not significant except in the five in whom end-diastolic volume increased.

*(Note that with but few exceptions, the heart emptied more completely in all groups.)*

_Circulation, Volume XXXII, September 1965_
SUPINE EXERCISE

Table 2

Hemodynamic Responses of the Three Patient Groups

<table>
<thead>
<tr>
<th>Group</th>
<th>Rest Stroke volume (ml./M.²)</th>
<th>Heart rate (per min.)</th>
<th>Cardiac index L./min./M.²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>41</td>
<td>73</td>
<td>3.0</td>
</tr>
<tr>
<td></td>
<td>55</td>
<td>104</td>
<td>5.7</td>
</tr>
<tr>
<td>% Change</td>
<td>15</td>
<td>43</td>
<td>90</td>
</tr>
<tr>
<td>Group B</td>
<td>40</td>
<td>85</td>
<td>3.4</td>
</tr>
<tr>
<td></td>
<td>46</td>
<td>117</td>
<td>5.4</td>
</tr>
<tr>
<td>% Change</td>
<td>15</td>
<td>37</td>
<td>59</td>
</tr>
<tr>
<td>Group C</td>
<td>39</td>
<td>75</td>
<td>2.9</td>
</tr>
<tr>
<td></td>
<td>35</td>
<td>117</td>
<td>4.1</td>
</tr>
<tr>
<td>% Change</td>
<td>-10</td>
<td>50</td>
<td>29</td>
</tr>
</tbody>
</table>

Left Ventricular Mean Shortening Distance and Rate

The average shortening distance during systole increased significantly. This was true regardless of direction of change in end-diastolic ventricular volume. Similarly, the mean rate of shortening showed a highly significant increase during exercise (fig. 4). This augmentation occurred at an essentially unchanged systolic mean force. In two subjects in whom systolic force did increase, however, shortening did not increase in one and rose only 8 per cent in the other. Because the changes in mean systolic force and in mean systolic cardiac size were so minimal in most of these studies, and ejection period (and time to peak force) decreased in all, it could be inferred from these data that an increase in fiber shortening rate usually represented an increase in the shortening rate of the contractile element itself. It was possible to calculate the force-velocity relations during a single systole for one patient in group C according to the method of Levine and Britman (fig. 5). Note the shift of the exercise curve to the right of the rest curve, indicating increased inotropism, at a time when end-diastolic volume had decreased in relation to the control.

Left Ventricular Work

External mechanical work per minute increased. Contractile element work index also increased, but primarily in those subjects with increased mean systolic ventricular volume during exercise.

![Figure 3](http://circ.ahajournals.org/)

**Figure 3**

Left ventricular diastolic pressure-volume relationships with exercise. While a concordant change or virtually no change occurred in the pressure and volume in the majority of cases, there were notable exceptions.

*Circulation, Volume XXXII, September 1965*
Myocardial Dynamics and Energy Consumption during Exercise

<table>
<thead>
<tr>
<th>Total of 10 patients</th>
<th>O₂ consumption (ml. per min. per 100 Gm.)</th>
<th>Mean systolic force, dyne x 10⁶</th>
<th>Pressure-time per beat, dyne x 10⁵/cm²</th>
<th>Force-time per beat, dyne sec. x 10⁶</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>Mean</td>
<td>10.4</td>
<td>160</td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td>S.D.</td>
<td>1.1</td>
<td>40</td>
<td>0.07</td>
</tr>
<tr>
<td>Exercise</td>
<td>Mean</td>
<td>16.8</td>
<td>160</td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td>S.D.</td>
<td>2.8</td>
<td>50</td>
<td>0.09</td>
</tr>
<tr>
<td>P Value†</td>
<td>&lt;0.001</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Total = contractile element work.
†P values were derived by the "t" test, analyzing the average of the paired difference for each value by the null hypothesis.

Left Ventricular Oxygen Consumption

(Table 3)

This value increased significantly during exercise in the 10 patients in whom oxygen consumption was measured. Likewise, oxygen consumption per beat increased.

Indices of Oxygen Consumption in Relation to Cardiac Effort

Oxygen consumption was correlated with external work (R = 0.88), and the line of regression had a positive oxygen-consumption intercept (Y) if extrapolated to zero work* (fig. 6). External mechanical efficiency increased. Similarly, when contractile element work index was plotted against oxygen consumption, there was a correlation between the two (R = 0.84) (fig. 6). Furthermore, change in work index correlated with...
### SUPINE EXERCISE

<table>
<thead>
<tr>
<th>Mean systolic shortening</th>
<th>Contractile element work index, Kg. M./min./M.²</th>
<th>External left ventricular work, Kg. M./min./M.²</th>
<th>External mechanical efficiency index, %</th>
<th>Force time/ oxygen consumption ratio</th>
<th>Contractile element work index/O₂ consumption ratio</th>
<th>External/total work ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distance, cm.</td>
<td>Rate, cm./sec.</td>
<td>3.4</td>
<td>12.4</td>
<td>5.3</td>
<td>3.8</td>
<td>18</td>
</tr>
<tr>
<td>0.6</td>
<td>2.7</td>
<td>1.2</td>
<td>1.0</td>
<td>3</td>
<td>0.6</td>
<td>0.06</td>
</tr>
<tr>
<td>4.2</td>
<td>17.9</td>
<td>10</td>
<td>7.8</td>
<td>23</td>
<td>2.1</td>
<td>0.60</td>
</tr>
<tr>
<td>1.0</td>
<td>5.1</td>
<td>3.0</td>
<td>2.2</td>
<td>6</td>
<td>0.7</td>
<td>0.12</td>
</tr>
<tr>
<td><strong>&lt;0.05</strong></td>
<td><strong>&lt;0.05</strong></td>
<td><strong>&lt;0.05</strong></td>
<td><strong>&lt;0.005</strong></td>
<td><strong>&lt;0.025</strong></td>
<td><strong>&lt;0.05</strong></td>
<td><strong>&lt;0.05</strong></td>
</tr>
</tbody>
</table>

**Figure 7**

Ratio of external to total work plotted against external mechanical efficiency. Except for one case, as the ratio increased, efficiency increased and vice versa. The increase in ratio generally was related to a reduction in mean systolic ventricular volume (seven of ten instances).

**Figure 8**

Indices of force plotted against oxygen consumption (qO₂). Solid circle, rest open circle, exercise. Note the random response of each value to exercise despite a rise in oxygen consumption. There was no correlation between change in any of these indices of force and change in oxygen consumption. There was, however, a significant correlation between resting oxygen consumption and pressure-time per beat (0.86 ± 0.33), and a borderline correlation between resting oxygen consumption and force-time per beat (0.61 ± 0.33).
efficiency \(^\ddagger\) (fig. 7). Reduction in mean systolic heart size tends to increase the ratio of external to total work. Such a reduction occurred in seven of the 10 subjects, and thus would appear to play a partial role in the observed increase in external efficiency. Because of previous studies indicating correlations between indices of cardiac tensile force and oxygen consumption, certain of these values were plotted as shown in figure 8. Resting pressure-time per beat correlated with resting oxygen consumption \((0.87 \pm 3)\). However, there was no correlation between change in pressure-time, force-time or systolic mean force and change in oxygen consumption during exercise. As shown in table 3, none of these values changed significantly with exercise, although oxygen consumption did. On the other hand, change in mean shortening distance correlated with change in oxygen consumption \((R = 0.68 \pm 0.33)\). As shown in table 3, both mean shortening rate and distance increased significantly with exercise, and directionally similar to oxygen consumption in the majority of instances \((\text{fig. 9})\).

\(^\ddagger\)This directional relationship to external efficiency is in part to be expected because of the correlations between oxygen consumption and indices of work, but it was not seen in all subjects.

**Discussion**

**Response of Left Ventricle to Exercise**

The results of these studies suggest that there are three major mechanisms participating in the cardiac response to exercise in the supine position. The first is an increase in heart rate. The second is an increase in inotropism. Ordinarily, this should reduce cardiac systolic and diastolic dimensions and result not only in reduction in initial or end-diastolic ventricular volume, but also an increase in the fraction of blood ejected, as well as an increase in the speed at which this volume is ejected for any given force. This "pure" response was seen in 15 patients (groups B and C). Thus, as described by Rushmer \(^1\) and Braunwald and associates, \(^3\) the role of inotropic factors in the exercise response of nearly normal man would appear to be firmly established by these observations. On the other hand, under certain conditions, a third factor appears to be operative—an increase in initial diastolic tension of the heart, i.e., Starling's law. In five patients, end-diastolic volume increased and a much greater stroke volume \((\text{and minute output})\) was expelled during exercise than at rest, in contradistinction to the other two subgroups. In addition to inotropic action, the increase in diastolic cardiac size and tension further enhanced both cardiac filling and emptying. In this regard in Braunwald's series, \(^4\) only a minor augmentation of stroke volume occurred in those patients in whom cardiac dimension decreased during exercise. These individuals are similar to our subgroups B or C.

The Starling mechanism would seem to be essential in those individuals who ejected an unusually large stroke volume during exercise; this is illustrated most graphically in patient GM. Stroke volume increased from 42 to 65 ml./beat/M. \(^2\). This was accomplished by an increase in initial cardiac volume as well as by an increase in the per cent of this volume ejected. The per cent shortening of the left ventricle in the resting state amounted to 22 per cent of the initial diastolic length, and during exercise shortening increased to 29 per cent of initial length. Had end-
SUPINE EXERCISE

369

diastolic volume not increased, however, then per cent shortening would have had to increase to 44 per cent of initial length to deliver the unrealistic figure for fractional emptying of 82 per cent! This value for shortening would exceed the degree of shortening calculated to be maximum in isolated cat papillary muscle.11 Thus, marked increase in stroke volume most likely can occur only when end-diastolic volume is increased. The inference is that the exercise response is composed of alterations in heart rate, degree of inotropism and degree of diastolic filling and distention of the heart. Because these factors may vary from moment-to-moment for reasons as yet unclear, it is not at all surprising that prior workers have reported disparate and varying results from animal-to-animal and from experiment-to-experiment. Different responses may readily occur at different times depending on the balance among these three regulatory forces and the intensity of demand for increased blood flow.

Activity of Contractile Element

Because there is in series with the contractile element an elastic component with force-dependent distensibility, one cannot always infer changes in events within the contractile element from changes in fiber activity alone. Thus, for an increase in fiber shortening rate to represent an increase in contractile element shortening rate, one must know that force and its time derivative have remained constant or have increased. Once the direction of change in velocity of contractile element has been ascertained, one must decide if the change in velocity is due to change in force (as the two vary reciprocally), or to change in initial fiber length, or to change in inotropism. The last two interventions can shift the curve of relationship of force and velocity to the right or left.12

Although instantaneous events are essential for quantitative analysis, some approximate conclusions may be drawn from interpretation of average systolic events4 (fig. 4). In most studies, systolic mean force changed little or increased, and ejection period (and time-to-peak force*), decreased. Therefore, increased systolic shortening rate indicated increased contractile element velocity. In groups B and C with no change, or actual decrease in end-diastolic volume, this implied a rightward shift in the force-velocity curve due to increased inotropism (fig. 5). In group A, with increase in end-diastolic volume, the observed augmentation of contraction could have been due to the "Starling effect" as well as to inotropism.

Pressure Volume Relationships

Because true end-diastolic pressure could not be assessed during the stress of exercise, meaningful correlations between this pressure and changes in end diastolic volume are virtually impossible.† Nevertheless, it was striking that concordant alterations or no change in direction of response were seen in the majority of patients.

Myocardial Energy Requirements during Exercise

Previous investigators have demonstrated an increase in cardiac oxygen consumption in relation to various indices of force generation: tension-time index,18 cardiac-effort index,14 pressure-time,15 and force-time.16 Such a relationship existed among the resting data in this study wherein for any given subject, the higher the pressure-time or force-time at rest, the higher the oxygen consumption (fig. 8). However, in our patients, exercise imposed a different response upon this baseline resting relationship. Mean cardiac

*Indicating increased rate of stretch of series elastic component.10
†Mean intrathoracic pressure will decrease or increase during supine exercise depending on whether the patient has assumed the inspiratory mid-position or is performing a partial Valsalva maneuver. This may have profound effects on the recorded left ventricular end-diastolic pressure. As a result, when uncorrected for variation in baseline, this pressure cannot necessarily be expected to represent true intraventricular pressure and, therefore, cannot accurately be related to changes in ventricular volume. It is assumed that many of the discrepancies shown in figure 3 were probably caused by this factor alone.

Circulation, Volume XXXII, September 1965
size and mean systolic force did not change appreciably in the majority of patients, nor did pressure-time or force-time. On the other hand, mean fiber shortening distance, and in particular, the mean rate of shortening of the ventricle during systole, increased significantly. It would seem then that these experiments constitute indirect evidence that there is an energy cost related to the kinetics of cardiac contraction as well as to the force of cardiac contraction.

The increase in external mechanical efficiency seen in seven of 10 studies is consonant with previously reported observations in normal subjects. In an engine of unchanged characteristics, efficiency tends to be fixed over its range of operation. Apparently, this is not true of the heart. There are various possible reasons for this. Britman and Levine have indicated that work done by the contractile element is different from, and greater than that done by the composite muscle fiber, which contains a series elastic component. These workers showed that as the ratio of external to total (or contractile element work) increased, external mechanical efficiency increased, presumably because oxygen consumption is most closely related to total or contractile element work. The ratio of external to total work increased with exercise in all but one of the patients, as did external mechanical efficiency. Consideration of the equations presented by Britman and Levine reveals that the amount of contractile element work done in excess of external or fiber shortening work will depend on mean cardiac size during systole. Thus, the ratio of the two will rise (and external mechanical efficiency increase), whenever the mean systolic volume decreases—all other factors remaining the same. Seven of the 10 subjects had a reduction in mean ventricular volume during exercise, suggesting this to be an important factor in explaining augmented cardiac efficiency herein observed. Consideration of activation energy (constant per beat), or of energy of shortening, only introduces added factors, theoretically increasing energy consumption in the exercising heart (which shortens through a greater distance more frequently per minute). This should lower rather than raise external efficiency, however.

Finally, the increase in efficiency may be more apparent than real, and may be related in part to the basal energy costs of the heart involved in metabolic synthesis and repair, etc. Kohn and Szymanski have reported oxygen consumption in the fibrillating heart of $3.63 \pm 15 \text{ ml./100 Gm.}/\text{min}$. This could be reduced further by asystolic arrest, presumably eliminating energy of activation. Positive intercepts on the “Y” or oxygen consumption axis of the work graphs (external and total work), have also been reported by others. The fact that oxygen consumption may not be zero for zero work will obviously affect the slope of the efficiency curve as the basal energy requirement becomes a progressively smaller fraction of total energy consumption.

Conclusions

Twenty subjects with nearly normal left ventricles have been studied during rest and supine leg exercise, by left heart catheterization and use of aortic thermodilution technics. It was demonstrated that the average end-diastolic volume for the group did not change during exercise, but that the ventricle emptied more completely to a smaller end-systolic volume and accomplished this in less time per beat than at rest.

The group could arbitrarily be subdivided into five subjects with significantly increased end-diastolic volume, seven with no significant change, and eight with reduction in end-diastolic volume. Stroke volume was increased most frequently in the group with increased end-diastolic volume. It is suggested that the response to supine exercise involves primarily increase in rate coupled with an inotropic stimulation of the heart, but that in some instances the Starling mechanism plays a distinct and important part by augmenting ventricular filling and emptying.

Energy consumption of the exercising
heart could be correlated with those indices reflecting an increase in kinetics of fiber shortening rather than force or tension development, which changed little in these experiments.

The increase in external mechanical efficiency during exercise would seem to be due to reduction in mean systolic heart size (with increase in ratio of external to total work of fiber), and to the fact that the heart has a basal oxygen cost (which becomes a progressively smaller fraction of the total).

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Effect of Supine Exercise on Left Ventricular Volume and Oxygen Consumption in Man

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