CLINICAL STUDIES

DISUSE ATROPHY OF THE LEFT VENTRICLE IN CHRONICALLY BEDRIDDEN ELDERLY PEOPLE

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In the elderly cardiac size and function are determined by their level of physical activity. In this study, we assessed by echocardiography, the anatomic and physiologic changes of the heart in 28 elderly patients who had no cardiac disease and who were chronically bedridden. The data obtained were compared to those obtained from a control group of 38 age and sex matched elderly people whose activities had not been restricted.

Chronically bedridden patients had markedly smaller left ventricular dimensions in both end-diastole and end-systole and smaller left atrial dimensions than did control subjects (3.7 ± 0.7 vs 4.7 ± 0.6 cm, p < 0.001, 2.4 ± 0.8 vs 2.9 ± 0.7 cm, p < 0.02 and 3.2 ± 0.5 vs 3.8 ± 0.9 cm, p < 0.01, respectively). Though the wall thickness of the interventricular septum did not differ between the study groups, the left ventricular posterior walls of the bedridden group were significantly thinner than in the control group (0.8 ± 0.2 vs 1.0 ± 0.2 cm, p < 0.01). The bedridden group had a significantly lower stroke index (26.9 ± 6.2 vs 47.0 ± 11.1 ml/min/m², p < 0.001) and cardiac index (1.84 ± 0.52 vs 3.15 ± 0.63 l/min/m², p < 0.001) than did the control group. Left ventricular mass index and left ventricular systolic stress were significantly lower in bedridden patients than in control subjects (88.0 ± 18.1 vs 143.5 ± 30.9 g/m², p < 0.001, and 135.9 ± 4.9 vs 186.6 ± 35.7 10³ dynes/cm², p < 0.001, respectively). The shortening fraction, however, did not differ between the two groups. The peak trans-aortic flow velocity of bedridden patients was not different from control subjects. The peak trans-mitral flow velocity in early diastole (R) of bedridden patients was significantly lower than in control subjects (0.44 ± 0.15 vs 0.57 ± 0.20 cm/s, p < 0.01), while the decrease in peak trans-mitral flow velocity during atrial systole (A) was not significant. The ratio of A to R was significantly larger in the bedridden group than in the control group (1.8 ± 0.7 vs 1.5 ± 0.5, p < 0.05).

We conclude that long term bed rest causes disuse atrophy of the heart in the elderly by decreasing venous return, left ventricular work and wall stress.

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The changes which occur in the structure of the heart with aging have not been defined. One report claims that the human heart increases in weight by 1 to 1.5 g per year1 and age-related left ventricular wall thickening has been demonstrated by echocardiogram²,³ Other studies, however, have shown that heart weight is not related

Key words:
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TABLE I

<table>
<thead>
<tr>
<th></th>
<th>age (yr)</th>
<th>height (cm)</th>
<th>weight (kg)</th>
<th>body surface area (m²)</th>
<th>heart rate (/min)</th>
<th>blood pressure (mmHg)</th>
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<tbody>
<tr>
<td>bedridden</td>
<td>mean</td>
<td>81</td>
<td>151.1</td>
<td>42.2</td>
<td>1.37</td>
<td>69</td>
</tr>
<tr>
<td>± SD</td>
<td></td>
<td>6</td>
<td>5.6</td>
<td>5.9</td>
<td>10.10</td>
<td>12</td>
</tr>
<tr>
<td>control</td>
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<td>80</td>
<td>151.3</td>
<td>48.8</td>
<td>1.46</td>
<td>70</td>
</tr>
<tr>
<td>± SD</td>
<td></td>
<td>6</td>
<td>7.5</td>
<td>8.9</td>
<td>0.14</td>
<td>13</td>
</tr>
<tr>
<td><strong>p value</strong></td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.02</td>
<td>&lt;0.05</td>
<td>NS</td>
<td>&lt;0.01: &lt;0.001</td>
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</table>

SD: standard deviation, NS: difference not significant

TABLE II

<table>
<thead>
<tr>
<th></th>
<th>AOD (cm)</th>
<th>LAD (cm)</th>
<th>Dd (cm)</th>
<th>Ds (cm)</th>
<th>IVST (cm)</th>
<th>PWT (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>bedridden</td>
<td>mean</td>
<td>3.0</td>
<td>3.2</td>
<td>3.7</td>
<td>2.4</td>
<td>1.0</td>
</tr>
<tr>
<td>± SD</td>
<td></td>
<td>0.4</td>
<td>0.5</td>
<td>0.7</td>
<td>0.8</td>
<td>0.2</td>
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<tr>
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<td>mean</td>
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<td>3.8</td>
<td>4.7</td>
<td>2.9</td>
<td>1.0</td>
</tr>
<tr>
<td>± SD</td>
<td></td>
<td>0.4</td>
<td>0.9</td>
<td>0.6</td>
<td>0.7</td>
<td>0.2</td>
</tr>
<tr>
<td><strong>p value</strong></td>
<td>NS</td>
<td>&lt;0.01</td>
<td>&lt;0.001</td>
<td>&lt;0.02</td>
<td>NS</td>
<td>&lt;0.01</td>
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AOD: aortic dimension, LAD: left atrial dimension, Dd: end-diastolic left ventricular dimension, Ds: end-systolic left ventricular dimension, IVST: interventricular septal wall thickness, PWT: left ventricular posterior wall thickness, SD: standard deviation, NS: difference not significant

We studied 28 consecutive elderly patients who were chronically bedridden for at least 1 year, and who had suffered from cerebrovascular disease or orthopedic dysfunction of their lower extremities. Age, height, body weight, body surface area, heart rate and blood pressure were recorded for each patient. Patients who had anatomical cardiac abnormalities due to myocardial infarction, hypertrophic cardiomyopathy, dilated cardiomyopathy, valvular heart disease or serious arrhythmias were excluded.

Thirty-eight hospitalized patients of similar age who had lived normally just before their hospitalization for a non-cardiac illness were selected as controls. Reasons for hospitalizations included cataracts, prostatic hypertrophy and early cancer of the stomach or uterus.

Echocardiograms

Commercially available real time 2-D Doppler echocardiographic instruments (Toshiba SSH-160A) were used. The chamber sizes and the left ventricle wall thick-
TABLE III

<table>
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<tr>
<th></th>
<th>SVI (ml/m²)</th>
<th>CI (l/min/m²)</th>
<th>Shortening Fraction (%)</th>
<th>LV Mass Index (g/m²)</th>
<th>LV Systolic Stress 10⁵ dynes/cm²</th>
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<tbody>
<tr>
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<td>mean 26.9</td>
<td>1.84</td>
<td>36.3</td>
<td>88.0</td>
<td>135.9</td>
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<td></td>
<td>± SD 6.2</td>
<td>0.52</td>
<td>9.1</td>
<td>18.1</td>
<td>14.9</td>
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<tr>
<td>control</td>
<td>mean 47.0</td>
<td>3.15</td>
<td>38.0</td>
<td>142.5</td>
<td>186.6</td>
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<tr>
<td></td>
<td>± SD 11.1</td>
<td>0.63</td>
<td>10.7</td>
<td>30.9</td>
<td>35.7</td>
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</table>

P value <0.001 <0.001 NS <0.001 <0.001

SVI: stroke index, CI: cardiac index, LV: left ventricle, SD: standard deviation, NS: difference not significant

Fig. 1. Left ventricular dimensions in end-diastole (De) and end-systole (Ds), interventricular wall thickness (IVST), left ventricular posterior wall thickness (PWT) and left ventricular mass index (LVMI) in chronically bedridden elderly subjects (open column) and control elderly subjects (striped column). ** and * signify p<0.001 and p<0.02, respectively, and NS shows not significant difference.

Peak velocities during early diastole (R) and atrial contraction (A) of mitral and aortic flow signals were measured on the paper without angle correction. Recordings were made at the mid-expiratory phase of respiration.

From the echocardiographic data, the following parameters were measured: 1) left ventricular end-systolic and end-diastolic dimensions (Ds and Dd); 2) thicknesses of the interventricular septum and left ventricular posterior wall (IVST and PWT); 3) left atrial dimensions (LAD); 4) aortic dimensions (AOD); 5) shortening fraction of the left ventricle ((Dd–Ds)/Dd); 6) stroke index as the difference between the left ventricular end-diastolic volume and end-systolic volume indices (SVI=EDVI−ESVI); 7) cardiac index (SVI×HR); 8) left ventricular mass index ((1.05×((Dd+IVST+PWT)³−Dd³)+3×(IVST+PWT)2+Dd))/10; 10) peak mitral flow velocities (R: during early diastole and A: during atrial systole); 11) ratio of the peak mitral flow velocities during early diastole and atrial systole (A/R); and 12) peak aortic flow velocity (AOF).

Statistical Analysis

Stroke Volume, cardiac output and left ventricular mass are corrected for body surface area and expressed as an index. We compared the samples using Student's unpaired t distribution. A probability value of p<0.05 was regarded as significant.
TABLE IV

<table>
<thead>
<tr>
<th></th>
<th>TAF</th>
<th>TMF</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>m/sec</td>
<td>m/sec</td>
</tr>
<tr>
<td>bedridden</td>
<td>mean</td>
<td>1.12</td>
</tr>
<tr>
<td>± SD</td>
<td>0.64</td>
<td>0.15</td>
</tr>
<tr>
<td>control</td>
<td>mean</td>
<td>1.01</td>
</tr>
<tr>
<td>± SD</td>
<td>0.41</td>
<td>0.20</td>
</tr>
</tbody>
</table>

*p value* NS < 0.01 NS < 0.05

TAF: trans-aortic flow velocity, TMF: trans-mitral flow velocity, R: rapid filling phase, A: atrial contraction phase, SD: standard deviation, NS: difference not significant

RESULT

1. Bedridden patients (Table I): The chronically bedridden patients and control subjects were matched for age and sex. The mean body surface areas were significantly smaller (1.37 ± 0.10 vs. 1.46 ± 0.14 m², p < 0.05) and the blood pressures in systole and diastole were significantly lower in bedridden patients than in control subjects (128 ± 14 vs. 147 ± 27 mmHg, p < 0.01, and 69 ± 8 vs. 81 ± 17 mmHg, p < 0.001), while there was no difference in heart rates between the two groups.

2. Echocardiographic findings (Table II): The chronically bedridden patients had left ventricular dimensions in both end-diastole and end-systole, and left atrial and aortic dimensions which were markedly smaller than the control subjects (3.7 ± 0.7 cm vs. 4.7 ± 0.6 cm, 2.4 ± 0.8 vs. 2.9 ± 0.7 cm, 3.2 ± 0.5 vs. 3.8 ± 0.9 cm and 3.0 ± 0.4 cm vs. 3.2 ± 0.4 cm, respectively). Though the interventricular septum wall thickness did not differ between the study groups, the left ventricular posterior wall of bedridden patients was significantly thinner than in control subjects (0.8 ± 0.2 vs. 1.0 ± 0.2 cm, p < 0.01).

3. Derived hemodynamic indices (Table III): The bedridden group had significantly lower stroke indices (26.9 ± 6.2 vs 47.0 ± 11.1 ml/m², p < 0.001) and cardiac indices (1.84 ± 0.52 vs 3.15 ± 0.63 l/min/m², p < 0.001) than did the control group. The left ventricular mass index was significantly lower in bedridden patients than in control subjects (88.0 ± 18.1 vs 143.5 ± 30.9 g/m², p < 0.001) (Figure), and meridional left ventricular systolic stress was significantly lower in the bedridden group than in the control group (135.9 ± 14.9 vs 186.6 ± 35.7 10² dynes/cm², p < 0.001). The shortening fraction, however, did not differ between the two groups.

4. Doppler echocardiographic findings (Table IV): The peak trans-aortic flow velocities did not differ between bedridden patients and control subjects. The peak trans-mitral flow velocity in early diastole (R) of bedridden patients was significantly smaller than in control subjects (0.44 ± 0.15 vs. 0.57 ± 0.20 m/s, p < 0.01). The peak trans-mitral flow velocity in atrial systole (A) was smaller in the bedridden than in controls, but this difference was not significant. The ratio of A to R was significantly larger in the bedridden patient group than in the control group (1.8 ± 0.7 vs 1.5 ± 0.5, p < 0.05).

DISCUSSION

The heart is a pump which delivers a volume of oxygenated blood to the body in proportion to the body's metabolic requirements. The demands for oxygenated blood by the body are transmitted through the neuro-humoral system to the cardiovascular system. The heart responds to the neuro-humoral stimuli functionally at first, then, if the stimulus persists, it responds morphologically. It has long been known that systolic blood pressure increases with age. Any rise in blood pressure increases left ventricular wall stress. Left ventricular wall thickening is a mechanism of adaptation which returns

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elevated left ventricular wall stress to normal. In this respect, the earlier data may be correct in suggesting that the left ventricular wall thickness increases with age, independent of other age-related changes in the left ventricular cavity. However, the reports which attempt to correlate heart weight with age provide varied results. Such differences in the reported sizes or weights of aged hearts may be due to differences in the physical activity level of each study population.

In humans, immobilization reduces the general metabolic need, decreases total blood volume and, therefore, decreases venous return. Chronic decreases in cardiac chamber size, stroke volume and cardiac output are proportional to the reductions in venous return which in turn decrease left ventricular wall stress. The cardiac mass decreases to adapt to the decrease in left ventricular wall stress. These findings have been observed in wheel chair-bound quadriplegic patients with spinal cord injuries.

In our study, the echocardiographic characteristics of chronically bedridden elderly patients were compared to control elderly subjects. We found that there were decreases in left ventricular dimensions in both end-diastole and end-systole, left atrial dimensions, aortic dimensions, left ventricular posterior wall thickness, stroke index, cardiac index, left ventricular systolic wall stress and in left ventricular mass. These findings are consistent with those seen in quadriplegic patients who had decreased activity in combination with functioning skeletal muscle mass.

The body surface area of the bedridden group was significantly less than that of the control group, because bedridden patients had lower body weights. This loss of body weight was thought to be induced by the atrophy of functioning skeletal muscle. This atrophy is associated with a reduction in the number of skeletal muscle capillaries and a decrease in the size of the vascular bed to be perfused by the heart. The workload of the heart, therefore, decreases.

Another cause of cardiac atrophy is malnutrition. This was reported by St. John Sutton and his colleagues who observed that in anorexia nervosa patients the heart is decreased in size. The nutritional state of patients in our study may be insufficient, as compared to controls but we believe that the principal cause of cardiac atrophy in immobilized patients is cardiac muscle disuse. The animal experiment by Cooper and his co-workers revealed that disuse atrophy of the unloaded cardiac muscle was established easily.

Using Doppler echocardiography, we found that our elderly study group had decrease a R-wave and increased A-wave velocities of trans-mitral blood flow, and increased A/R ratios. The R wave of chronically bedridden patients was significantly smaller than that of control elderly people but the A-wave velocity did not differ between these 2 groups. Miyatake and his co-workers reported that left ventricular diastolic function is impaired with aging and that the contribution of atrial contraction to left ventricular filling is augmented to compensate. Choong and his group found that nitroglycerin decreased the peak velocity of the E wave (equal to R wave in this paper) but did not alter the peak velocity of the A wave, and decreased the ratio of the peak velocity of the E wave to the peak velocity of the A wave. These changes did not depend on the impairment of left ventricular diastolic function, but on the reduction of left ventricular preload. Changes in the Doppler mitral flow velocity pattern in the bedridden group account for the impairment of left ventricular filling that occurs with aging and the reduced preload due to decreased cardiac filling.

This study demonstrates that immobility in the elderly causes disuse atrophy in functioning skeletal muscles which leads to reduced cardiac preload, low cardiac output, decreased left ventricular systolic stress and ultimately disuse atrophy of the heart muscle.

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