The Effect of Exercise on Coronary Blood Flow, Myocardial Oxygen Consumption and Cardiac Efficiency in Man

By Thomas A. Lombardo, M.D., Leonard Rose, M.D., Max Taeschler, M.D., S. Tuluy, M.D. and R. J. Bing, M.D.

It has been known that exercise causes an increase in the coronary blood flow in animals. The present work has been carried out to study the effect of exercise on coronary blood flow and myocardial oxygen consumption of the human heart in vivo. The results indicate that the heart responds to the increased load of exercise with a rise in coronary blood flow. Since the arteriovenous coronary oxygen difference shows little change, the increase in oxygen consumption of the heart muscle is primarily the result of an increased coronary blood flow. As the cardiac work rises more than the myocardial oxygen consumption, the left ventricular efficiency increases. The response of the failing heart muscle to acute increases in load produced by exercise does not differ from that of the normal heart or of the isolated heart.

CATHETERIZATION of the coronary sinus in man in conjunction with the nitrous oxide method has made it possible to determine coronary blood flow and myocardial oxygen consumption in man. Until now, the method has been used to follow the behavior of human heart muscle of resting individuals only. A study of the coronary circulation and myocardial oxygen consumption during exercise would appear to be of interest, because exercise constitutes a temporary increase in load to which the heart must adjust itself by coronary circulatory and metabolic changes. The alterations in the coronary circulation and myocardial oxygen consumption are of particular importance since it is during increased cardiac activity that deficiencies of the coronary circulation become most apparent. It is the purpose of this paper to present data on changes in coronary blood flow, myocardial oxygen consumption and myocardial efficiency in man occurring during moderate exercise and to discuss their significance.

METHODS

Selection of Patients

Thirteen patients with diseases of various etiologies contributed data which form the basis of this report. All patients were studied for diagnostic purposes and coronary blood flows were obtained only after diagnostic catheterization studies had been completed. The nature of the procedure was explained to the patient and his written consent obtained. Two subjects (E. S., R. B.) were mildly anemic (table 1), and three (O. Hr., A. M., W. M.) had hypertensive cardiovascular disease. One of these (O. Hr.), who also was slightly anemic, was on hexamethonium chloride therapy (table 1). Two subjects (A. A., W. R.) had aortic insufficiency and the others suffered from angina pectoris (O. S.), rheumatic heart disease with mitral stenosis (J. Dl.), senile heart disease with auricular fibrillation (J. Df.), thyrotoxicosis (C. T.) and congestive failure, cause unknown (O. Hk.). All patients except O. Hk. were well compensated.

Procedures

The test was performed in the morning after most of these individuals had eaten a small breakfast. Following a one hour rest, respiratory gases were collected in a Douglas bag over a period ranging.
### Table 1.—Findings Obtained before and after Exercise on 13 Patients with Various Conditions Affecting the Circulation

<table>
<thead>
<tr>
<th>Subject</th>
<th>Diagnosis</th>
<th>Cardiac Output cc./min.</th>
<th>Cardiac Index cc./min./M²</th>
<th>Coronary Arteriovenous O₂ Difference vol. %</th>
<th>Ox Content Coronary Sinus vol. %</th>
<th>Coronary Vascular Resistance mm.Hg/cc./100 Gm/min.</th>
<th>Coronary Left Ventricular O₂ Consumption cc./100 Gm/min.</th>
<th>Left Ventricular O₂ Consumption cc./min.</th>
<th>Aerobic Energy Up-take of Left Ventricle Kg. Meters</th>
<th>Mean Arterial Pressure mm. Hg</th>
<th>Work of Left Ventricle Kg.</th>
<th>Mechanical Efficiency of Left Ventricle %</th>
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</thead>
<tbody>
<tr>
<td>X. W.</td>
<td>Normal</td>
<td>Before</td>
<td>8,100</td>
<td>3,850</td>
<td>11.0</td>
<td>6.4</td>
<td>101</td>
<td>0.92</td>
<td>11.2</td>
<td>22.4</td>
<td>44.8</td>
<td>93</td>
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<tr>
<td></td>
<td></td>
<td>After</td>
<td>10,400</td>
<td>4,950</td>
<td>12.1</td>
<td>5.7</td>
<td>101</td>
<td>12.3</td>
<td>24.5</td>
<td>49.0</td>
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<td>E. S.</td>
<td>Mild anemia</td>
<td>Before</td>
<td>5,000</td>
<td>3,380</td>
<td>8.5</td>
<td>3.1</td>
<td>69</td>
<td>1.2</td>
<td>5.8</td>
<td>6.1</td>
<td>12.2</td>
<td>85</td>
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<tr>
<td></td>
<td>Hgb. 9.5 Gm.</td>
<td>After</td>
<td>6,260</td>
<td>4,230</td>
<td>9.2</td>
<td>2.8</td>
<td>89</td>
<td>8.2</td>
<td>8.6</td>
<td>17.2</td>
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<tr>
<td>R. B.</td>
<td>Mild anemia</td>
<td>Before</td>
<td>3,870</td>
<td>1,990</td>
<td>7.4</td>
<td>3.0</td>
<td>162</td>
<td>0.55</td>
<td>12.0</td>
<td>19.5</td>
<td>39.0</td>
<td>90</td>
</tr>
<tr>
<td></td>
<td>Hgb. 9.4 Gm.</td>
<td>After</td>
<td>11,550</td>
<td>5,920</td>
<td>8.1</td>
<td>2.9</td>
<td>267</td>
<td>21.8</td>
<td>35.4</td>
<td>70.8</td>
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<tr>
<td>A. M.</td>
<td>H. C. V. D.</td>
<td>Before</td>
<td>5,120</td>
<td>3,160</td>
<td>8.1</td>
<td>4.0</td>
<td>111</td>
<td>1.80</td>
<td>9.0</td>
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<td>23.4</td>
<td>107</td>
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<td></td>
<td>After</td>
<td>9,789</td>
<td>6,000</td>
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<td>2.7</td>
<td>278</td>
<td>1.45</td>
<td>23.6</td>
<td>28.2</td>
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<tr>
<td>W. M.</td>
<td>H. C. V. D.</td>
<td>Before</td>
<td>7,400</td>
<td>3,900</td>
<td>11.8</td>
<td>5.1</td>
<td>80</td>
<td>1.72</td>
<td>9.4</td>
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<td>26.4</td>
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<td></td>
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<td>After</td>
<td>13,900</td>
<td>7,300</td>
<td>12.2</td>
<td>4.6</td>
<td>124</td>
<td>1.10</td>
<td>15.1</td>
<td>21.2</td>
<td>42.4</td>
<td>138</td>
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<td>O. Hr.</td>
<td>H. C. V. D.</td>
<td>Before</td>
<td>8,255</td>
<td>4,390</td>
<td>6.5</td>
<td>2.6</td>
<td>126</td>
<td>0.85</td>
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<td>13.1</td>
<td>26.2</td>
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<td></td>
<td>Mild anemia</td>
<td>After</td>
<td>16,276</td>
<td>8,700</td>
<td>7.0</td>
<td>1.7</td>
<td>137</td>
<td>1.04</td>
<td>9.6</td>
<td>15.5</td>
<td>31.0</td>
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<td>A. A.</td>
<td>Aortic Insufficiency</td>
<td>Before</td>
<td>3,240</td>
<td>1,890</td>
<td>9.6</td>
<td>4.4</td>
<td>89</td>
<td>1.13</td>
<td>8.5</td>
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<td>27.4</td>
<td>101</td>
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<tr>
<td></td>
<td></td>
<td>After</td>
<td>4,720</td>
<td>2,720</td>
<td>9.3</td>
<td>4.6</td>
<td>142</td>
<td>1.01</td>
<td>13.2</td>
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<td>W. R.</td>
<td>Aortic Insufficiency</td>
<td>Before</td>
<td>5,150</td>
<td>2,750</td>
<td>10.3</td>
<td>7.5</td>
<td>80</td>
<td>1.00</td>
<td>8.2</td>
<td>13.6</td>
<td>27.2</td>
<td>80</td>
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<tr>
<td></td>
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<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<tr>
<td>J. Df.</td>
<td>Mitral</td>
<td>Before</td>
<td>15,400*</td>
<td>7,120</td>
<td>13.0</td>
<td>1.2</td>
<td>112</td>
<td>0.55</td>
<td>14.6</td>
<td>31.0</td>
<td>60.0</td>
<td>95</td>
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<td>Stenosis</td>
<td>After</td>
<td>20,000*</td>
<td>9,260</td>
<td>13.0</td>
<td>1.2</td>
<td>149</td>
<td>0.75</td>
<td>19.4</td>
<td>41.5</td>
<td>82.0</td>
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<tr>
<td>O. S.</td>
<td>Angina</td>
<td>Before</td>
<td>7,250</td>
<td>4,050</td>
<td>11.2</td>
<td>4.3</td>
<td>83</td>
<td>0.92</td>
<td>9.3</td>
<td>14.8</td>
<td>30.4</td>
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<td></td>
<td>Pectoris</td>
<td>After</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
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<td>—</td>
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<tr>
<td>J. Df.</td>
<td>Auricular</td>
<td>Before</td>
<td>4,700</td>
<td>2,790</td>
<td>10.2</td>
<td>3.9</td>
<td>67</td>
<td>1.14</td>
<td>6.9</td>
<td>8.2</td>
<td>16.4</td>
<td>76</td>
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<td></td>
<td>Fibrillation</td>
<td>After</td>
<td>6,900</td>
<td>4,100</td>
<td>9.7</td>
<td>3.6</td>
<td>80</td>
<td>1.14</td>
<td>6.9</td>
<td>8.2</td>
<td>16.4</td>
<td>76</td>
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<tr>
<td>O. Hk.</td>
<td>Congestive Heart</td>
<td>Before</td>
<td>5,100</td>
<td>2,800</td>
<td>13.1</td>
<td>5.2</td>
<td>45</td>
<td>1.71</td>
<td>5.9</td>
<td>8.2</td>
<td>17.1</td>
<td>77</td>
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<td></td>
<td>Failure</td>
<td>After</td>
<td>6,300</td>
<td>3,500</td>
<td>14.7</td>
<td>4.0</td>
<td>72</td>
<td>1.16</td>
<td>10.6</td>
<td>14.9</td>
<td>30.9</td>
<td>72</td>
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<tr>
<td>C. T.</td>
<td>Thyrotoxicosis</td>
<td>Before</td>
<td>9,428</td>
<td>5,290</td>
<td>12.2</td>
<td>7.1</td>
<td>65</td>
<td>1.55</td>
<td>7.9</td>
<td>12.3</td>
<td>24.5</td>
<td>101</td>
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<tr>
<td></td>
<td></td>
<td>After</td>
<td>12,198</td>
<td>6,840</td>
<td>12.3</td>
<td>6.9</td>
<td>86</td>
<td>1.63</td>
<td>10.5</td>
<td>16.3</td>
<td>32.7</td>
<td>140</td>
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</table>

* Benedict-Roth apparatus used.
from two to three minutes for the determination of oxygen consumption and carbon dioxide production. In one patient (J. D.) the oxygen consumption was determined with a Benedict-Roth spirometer. The right ventricle was catheterized via the left median antecubital vein and a sample of blood was obtained for the determination of the cardiac output.\(^1\)

Following this, the patient was placed in the right lateral position and the coronary sinus was catheterized. The presence of the catheter in the coronary sinus was determined by the fluoroscopic position of the catheter, the dark color of the blood withdrawn, the shape and height of the pressure,\(^1\) and by absence of cardiac irregularities.\(^2\) Successful intubation of the coronary sinus was possible in about 50 per cent of the patients in whom it was attempted. This low incidence is probably the result of anatomic variations in the structure of the right atrium or the coronary ostium.\(^3\) Although untoward effects have been reported to follow catheterization of the coronary sinus,\(^4\) none developed in our patients. This is due to the fact that intubation was attempted only with the patient in the right lateral position. Thus, the correct position of the catheter would be accurately ascertained and the entrance of the catheter into the right ventricle could be avoided. After the catheter had been correctly introduced the patient was placed on his back and an indwelling arterial needle was introduced into the right brachial artery.

With the catheter placed in the coronary sinus, nitrous oxide was administered by the method previously described.\(^5\) The patient was allowed to breathe the gas mixture for 12 minutes to permit saturation of the heart muscle with nitrous oxide. Just before the end of the saturation period, samples of both coronary sinus and arterial blood were collected for the determination of the nitrous oxide content at full saturation. The patient was then suddenly disconnected from the respiratory system and permitted to breathe room air. Four one-minute samples were drawn simultaneously from the coronary sinus and brachial artery. All blood samples were drawn in Luer-Lok syringes of 10 cc. capacity which had been oiled and which contained 10 drops of heparin.

After the resting observations had been completed, nitrous oxide was again administered for 10 minutes. At the beginning of the eighth minute of nitrous oxide inhalation, exercise was started. It was continued during the collection of the blood samples, until collection of expired air and sampling of right atrial blood had been completed. The total duration of exercise was slightly less than 10 minutes. Exercise was performed in the supine position. It was not mechanically controlled in some subjects (X. W., E. S., O. Hk., O. S., J. Dl., J. Df., A. A., and R. B.), consisting of alternating bicycle motion of the legs against a resistance imposed by the hands of one of us. In the other subjects (O. Hr., W. R., C. T., A. M., W. M.) exercise was performed on a bicycling apparatus by causing the patients to push their feet against two weighted pedals. Using this apparatus, the work performed amounted to approximately 19 kilogram meters per minute.\(^6\)

Blood pressures were measured with a sphygmomanometer or the strain gauge. In the latter case the pressures were optically recorded. Mean pressures were obtained by adding one-third of the pulse pressure to the diastolic pressure, or by planimetric integration of the area under the pressure curve.

### Calculations

Cardiac output was calculated according to the Fick equation:

\[
\text{Cardiac output} = \frac{\text{oxygen consumption (in cc.)}}{\text{oxygen content arterial blood (vol. %) minus oxygen content mixed venous blood (vol. %)}} \times 100
\]

The coronary blood flow was calculated according to the method previously described.\(^1\)\(^4\)\(^5\) Gregg has shown that the coronary sinus drains primarily left ventricular muscle. Consequently, the nitrous oxide method measures primarily the flow through a unit (100 Gm.) of left ventricular tissue.\(^7\) Furthermore, Visscher has stated that the oxygen content of the coronary sinus blood does not necessarily represent that of other venous channels draining the myocardium.\(^8\) Therefore, the nitrous oxide method measures blood flow through that unit of muscle only which drains into the coronary sinus. Gregg and his co-workers have found an average variation between the coronary flow per minute per 100 Gm. as determined with the nitrous oxide method and with the rotameter of \(\pm 12.4\) per cent.\(^9\)

Coronary vascular resistance was calculated according to the equation:

\[
\text{Coronary vascular resistance} = \frac{\text{mean aortic pressure (mm. Hg) - coronary blood flow (cc./100 Gm./min.)}}{\text{coronary blood flow (cc./100 Gm./min.)}}
\]

The resistance calculated in this manner refers to a unit (100 Gm.) of the coronary vascular bed which drains into the coronary sinus.

The oxygen consumption per 100 Gm. of left ventricular muscle was obtained with the equation:

\[
\text{Oxygen consumption (cc.)/100 Gm. of left ventricular muscle/min.} = \frac{\text{arterial oxygen content (vol. %) - coronary sinus oxygen content (vol. %)}}{\text{coronary vascular oxygen flow (cc./100 Gm. left ventricular muscle/min.)}} \times \text{left ventricular coronary flow (cc./100 Gm. left ventricular muscle/min.)}
\]

* Furnished by Respiration Aids Company, New York.
Oxygen consumption of the total left ventricle was obtained with the formula:

\[ \text{Oxygen consumption of left ventricle (cc. oxygen/min.)} = \frac{\text{left ventricular weight}}{100} \times \frac{\text{oxygen consumption/100 Gms.}}{\text{cardiac output (cc./min.)}} \]

Normal heart weight was calculated from tables of Smith\(^{19}\) and the left ventricular weight was assumed to be 53 cent of the total heart weight.\(^{14}\)

Since each cubic centimeter of oxygen corresponds to about 2 kilogram meters of energy,\(^{11}\) the aerobic energy uptake of the left ventricle was calculated as follows:

**Aerobic energy uptake of left ventricle (Kg. meters) = oxygen usage in cc./min. \times 2.**

The work of the left ventricle was obtained from the formula of Starling\(^{21}:\)

\[ \text{Work (Gm. cm./min.) = cardiac output (cc./min.) \times mean aortic pressure (cm. Hg) \times 13.6 (specific gravity of mercury).} \]

Gram centimeters per minute were converted to kilogram meters through division by 100,000. Only pressure energy was calculated because the velocity energy of the left ventricle is relatively small.

The relationship of aerobic energy uptake to work of the left ventricle (the mechanical efficiency of the left ventricle) was calculated from the formula:

**Mechanical efficiency (per cent)**

\[ = \frac{\text{work of left ventricle (Kg. meters/min.)}}{\text{aerobic energy uptake of left ventricle (Kg. meters/min.)}} \]

Because the weights of hypertrophied hearts could not be accurately observed, left ventricular efficiency could not be calculated in the presence of cardiac hypertrophy. However, by using normal heart weights in patients with hypertrophied left ventricles, maximal values for efficiency were obtained; therefore, if the maximal efficiency was low, true values were even lower. This was shown to be the case in myocardial failure.\(^{3}\) In assessing the effect of exercise on the heart, individual percentages of efficiency are immaterial but the changes in percent following exercise are valid.

**Analysis**

The manometric method of Van Slyke and Neil was used for the determination of oxygen and carbon dioxide in blood.\(^{13}\) Nitrous oxide was determined according to the method of Kety and Schmidt.\(^{14}\) The oxygen and carbon dioxide in expired air was analyzed according to the method of Scholander.\(^{15}\)

**Results**

Exercise produced a rise in cardiac output of 4066 cc. per minute, which represents a rise of 60 per cent above resting levels (table 1). Similar results were obtained by Hickam and his associates.\(^{16}\) In one patient (J. Dl.) the cardiac outputs were calculated from oxygen consumptions obtained with a closed circuit method (Benedict-Roth apparatus). It is possible that this accounts for the extremely high value obtained in this individual (table 1).

Previous studies on normal resting subjects have shown that the oxygen content of coronary sinus blood varies from 3.9 to 6.9 volumes per 100 cc.\(^{14,2,2}\) In this study, only two subjects (C. T., W. R.) exhibited resting values higher than 6.9 volumes per 100 cc. (table 1). All three patients with anemia (E. S., R. B., O. Hr.) and one patient with mitral stenosis (J. Dl.) demonstrated lower than normal values for oxygen content of coronary venous blood at rest (table 1). The oxygen content of coronary sinus blood was determined in 12 of the 13 subjects after exercise (table 1). Nine of this group exhibited a decline in oxygen content ranging from 0.1 to 1.3 volumes per 100 cc.; two showed no change (O. S., J. Dl.), and one (A. A.), revealed a slight rise (table 1).

The left ventricular oxygen extraction (coronary arteriovenous oxygen difference) increased in 10 of the 12 subjects with exercise (table 1). The increase ranged from 0.1 to 1.6 volumes per 100 cc. with an average increase of 0.6 volumes per 100 cc. The largest extraction on exercise occurred in the patient with congestive heart failure (O. Hk., table 1). One subject (J. Dl.) showed no change with exercise, and two (J. Dl., A. A.) showed a slight decrease. The resting coronary oxygen extraction was below normal (12 volumes per 100 cc.) in the group with mild anemia (E. S., R. B., O. Hr., table 1). It was normal in the patient with thyrotoxicosis (C. T.), slightly elevated in the subject with mitral stenosis (J. Dl.) and highest in the patient with congestive heart failure (O. Hk.). (See table 1.) Similar findings have been described in a previous publication.\(^{16}\)

In all subjects except one (X. W.), exercise produced an increase in the blood flow through
100 Gm. of left ventricular muscle of from 11 to 167 cc. per 100 Gm. per minute, with a mean increase of 43 cc. (table 1). This figure represents an average rise of 45 per cent. Large increments in coronary blood flow were also found by Essex in exercising dogs. It is of interest that increments in coronary blood flow were particularly great in those subjects who exhibited a large rise in cardiac output during exercise (R. B., A. M., W. M., A. A.). In one patient (O. Hr.) the cardiac output increased 97 per cent but the coronary blood flow rose only 8 per cent. This patient was receiving hexamethonium chloride as hypertensive therapy.

The coronary vascular resistance per 100 Gm. declined in seven out of nine patients with exercise (table 1). It is of interest that the coronary vascular resistance also fell in two patients suffering from hypertension (A. M., and W. M., table 1). This indicates that the coronary vascular resistance in this disease may not be fixed. A rise in mean arterial pressure with exercise was noted in a patient with thyrotoxicosis (C. T.) and in one patient with hypertension (O. Hr.). This rise was out of proportion to the increase in coronary blood flow and consequently, coronary vascular resistance increased (table 1). However, one of the patients (O. Hr.) was receiving hexamethonium chloride, which may have prevented further coronary vasodilatation through ganglionic blocking.

Exercise induced a rise in left ventricular oxygen consumption in all patients studied. The increase ranged from 1.1 to 14.6 cc. per 100 Gm. per minute, with an average of 4.9 cc. per 100 Gm. per minute or 65 per cent (table 1). In one patient with anemia (R. B.), one with hypertensive heart disease (A. M.), and in the patient with congestive heart failure (O. Hk.), the increase in myocardial oxygen consumption was especially marked (table 1). It is in these subjects that increments in coronary blood flow with exertion were large. The increase in oxygen consumption per unit of heart weight is of interest because it indicates that an increase in load which occurs with exercise is commensurate with an increase in oxygen usage by the heart.

The increase in left ventricular aerobic energy uptake averaged 48 per cent. The rise was particularly significant in two subjects with hypertension (A. M., W. M.), in one patient with anemia (R. B.), and in the individual with congestive heart failure (O. Hk.). (See table 1.)

Mean arterial pressure was determined in 9 of the 13 subjects before and after exercise. The average elevation in the systemic pressure of five patients was 30 mm. Hg, no change was observed in two, two showed a slight fall with exercise (table 1). These findings are in agreement with those of Ellis and Bock and co-workers.

As the cardiac output rose in all patients, the work of the left ventricle increased. In patients in whom all data were available, the rise in left ventricular work averaged 106 per cent. One patient with hypertension and anemia (O. Hr.) and one with aortic insufficiency (A. A.) showed a very large increase in work (table 1).

In five of seven patients the mechanical efficiency of the left ventricle rose with exercise (table 1). The average increase was 25 per cent. Two patients, one with hypertension (A. M.) and one with congestive failure (O. Hr.) demonstrated a decrease in left ventricular efficiency (table 1). The fall in efficiency noted in the patient with myocardial failure is of particular interest because it indicates that in myocardial failure, as in normal individuals, the oxygen consumption of the left ventricle increases as the load rises. The work of the heart, however, fails to increase proportionately. This results in a disproportionate rise in aerobic energy uptake and therefore in a fall in left ventricular efficiency. The greatest increase in efficiency was noted in the subject with hypertension and anemia who was under treatment with hexamethonium chloride (O. Hr.), and the patient with aortic insufficiency (A. A.). Exercise in these individuals produced large increments in cardiac work with only slight increases in energy uptake.

**DISCUSSION**

The studies reported in this communication show that moderate exercise increases coronary blood flow without significant change in oxygen
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extraction (table 1). This indicates that increases in load are met primarily by a rise in coronary blood flow. This result can be anticipated from previous studies on the hearts of normal resting subjects. It has been stated that the average coronary blood flow in normal resting subjects is 77 cc. per 100 Gm. per minute, the average oxygen consumption is 9.4 cc. per 100 Gm. per minute, and the average oxygen extraction is 12 volumes per 100 cc. Therefore, the total coronary blood flow for a heart weighing 300 Gm. is approximately 240 cc. or 5 per cent of the cardiac output. This is a small figure when compared with the larger volume of blood which perfuses the kidney or the liver. This indicates that the hearts of resting subjects are already extracting a very large amount of oxygen and increases in metabolic demands must be met primarily by a rise in coronary blood flow.

It is of some interest that in a patient (O. S.) who had previous anginal attacks with exertion, pain did not develop during the test. As the oxygen extraction did not change in this subject, the increase in coronary blood flow must have been sufficient for the increased metabolic demands of the heart muscle during exercise.

Associated with increases in coronary blood flow due to exercise, a decline in coronary vascular resistance was noted in seven of nine individuals in whom blood pressures were recorded during exercise (table 1). In two of the seven, hypertension was present. It has been previously stated that in essential hypertension the coronary vascular resistance is increased. A decline in coronary vascular resistance during exercise observed in these individuals illustrates that the increase in resistance is functional rather than anatomic. This is in accordance with work previously published by Smith, Scheinberg, Prinzmetal, Pickering and Wilkins, who found that the vascular resistances of the renal, cerebral, muscular and hepatic beds are not fixed.

The increase in oxygen consumption per unit of left ventricular muscle noted with exercise averaged 65 per cent. The rise in cardiac oxygen consumption is in line with the findings of Starling and Visscher and Evans on the heart-lung preparation. These investigators found that an increase in diastolic volume of the heart in vitro is accompanied, within limits, by an increase in the oxygen usage of the heart. It has been stated previously that a chronic increase in diastolic volume, such as is seen in patients with congestive heart failure, does not lead to a rise in oxygen uptake of the heart per unit weight. In line with this observation is the finding that the oxygen usage per 100 Gm. of left ventricular muscle in patient O. H., who suffered from congestive heart failure, is not elevated (5.9 cc. oxygen per 100 Gm., as compared with a normal of 7.8 cc., table 1). It is of particular significance that in this patient exercise did result in increased oxygen uptake of the myocardium (from 5.9 to 10.6 cc. per 100 Gm. of left ventricular muscle, table 1).

This demonstrates that the response of the failing heart muscle to acute increases in load does not differ from that of the normal human heart in vivo or the isolated heart in vitro. These findings again show the difference in response of the heart to acute and chronic changes in diastolic volume.

The mechanical efficiency of the left ventricle increased in five of seven patients in whom all data for the calculations are available (table 1). Although the aerobic energy uptake of the left ventricle rises with exercise, the work of the left ventricle rises to a greater degree. This denotes that an increase in load of the normal heart leads to a more effective conversion of oxidative energy into useful work. This is in agreement with findings obtained in this laboratory which showed that as the work of the heart increases, the ratio of mechanical work to energy derived from the aerobic breakdown of glucose rises also. It is possible therefore that the cause of a more effective conversion of aerobic energy by the heart working with an increased load may be a more efficient utilization of glucose.

The left ventricular efficiency of the patient with myocardial failure declined during exercise. This is the result of both the considerable rise in myocardial oxygen consumption (80 per cent) and of a relatively small increase in cardiac output (22 per cent). Apparently the myocardium had reached the state where any fur-
ther acute increase in load failed to elicit a proportional augmentation of stroke volume; evidence of the existence of myocardial depression or failure.

A decline in mechanical efficiency during failure has been previously noted. The observation that exercise leads to a further fall in mechanical efficiency denotes that the conversion of aerobic energy into useful work in failure becomes increasingly more impaired as the load progresses. In line with this is the finding that in myocardial failure the energy equivalent of the glucose extracted by the heart muscle is considerably greater than the work performed. Thus, the failing heart appears to be unable to make full use of its aerobic energy as well as the energy derived from the breakdown of glucose.

**Summary**

The effect of exercise on the coronary circulation, myocardial oxygen consumption and efficiency was studied in 13 subjects by means of the nitrous oxide method in conjunction with catheterization of the coronary sinus.

The coronary blood flow per 100 Gm. of left ventricular muscle increased after exercise without significant changes in the oxygen extraction. The rise in coronary blood flow per 100 Gm. of left ventricular muscle averaged 45 per cent. An average increase of 63 per cent in cardiac output during exercise was observed.

The oxygen consumption per 100 Gm. of left ventricular muscle rose an average of 65 per cent. The rise in myocardial oxygen consumption with exercise noted in a patient with congestive heart failure demonstrated that the response of the failing heart muscle to acute increases in load does not differ from that of the normal human heart or the isolated heart.

The increase in left ventricular work observed during exercise was proportionately greater than the rise in left ventricular oxygen consumption. Consequently, the left ventricular efficiency increased. In the patient with myocardial failure, the left ventricular efficiency declined.

A fall in coronary vascular resistance was observed in most of the patients including those with hypertension. This indicated that the increase in coronary vascular resistance in hypertension is functional.

The significance of these findings is discussed.

**SUMARIO ESPAÑOL**

Se sabe que en los animales el ejercicio produce un incremento en la circulación coronaria. El trabajo presente se condujo para estudiar el efecto del ejercicio en la circulación coronaria y la consunción de oxígeno del miocardio en el corazón humano vivo. Los resultados indican que el corazón responde al incremento en carga de ejercicio con un aumento en circulación coronaria. Como la diferencia de oxigenación arteriovenosa muestra muy poco cambio, el aumento en consunción de oxígeno del miocardio es primeramente el resultado de un aumento en circulación coronaria. A medida que el trabajo del corazón aumenta más que la consunción de oxígeno por el miocardio, la eficiencia del ventrículo izquierdo también aumenta. La manera en que un corazón que está decompensándose responde a incrementos agudos en trabajo producidos por ejercicio no difiere en nada del corazón normal o del corazón aislado.

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THOMAS A. LOMBARDO, LEONARD ROSE, MAX TAESCHLER, S. TULUY and R. J. BING

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