Augmentation of external work of the heart due to an increase of blood pressure is accompanied by a greater increase of oxygen consumption (QO₂) than increase of work due to an elevation of cardiac output. Thus the calculated cardiac efficiency, or ratio between the external cardiac work and oxygen consumption, decreases in the first case and either remains unchanged or increases in the second. It is suggested that this difference might be explained as follows: 1. The type of metabolism during pressure-work could be different from that during flow-work. 2. The usual formula for calculation of cardiac efficiency takes into account only the external work performed during the isotonic period while the oxygen consumption is calculated for the whole cardiac cycle, including the isovolumic period. It was assumed therefore that it is necessary to relate the QO₂ to the total amount of energy developed by the left ventricle throughout the isotonic as well as the isovolumic period.

An attempt to separate the energy expenditure into static and dynamic effort was described by Wiggers and Katz in 1928. A poor correlation between stroke work and QO₂ has been emphasized during the last few years by several authors. Good correlations of oxygen consumption were found, however, with the product of the mean systolic pressure and the heart rate, with the time-integrated systolic pressure and with tension. However, these investigations do not explain the differences in oxygen consumption mentioned above. In addition, the importance of the geometry of the heart for the estimation of mechanical performance has been re-emphasized by Burch and others who have directed attention to an earlier paper of Woods.

In view of these findings the following assumptions have been made: The oxygen consumption has to be related not only to the stroke work (E₂) but also to the energy expenditure during the isovolumic period (E₁ being its function), necessary for sustaining the pressure at a given volume during the isovolumic period. It could be calculated as follows:

\[ E₁ = \frac{\int_{t₀}^{t₁} P \, dt}{t₁ - t₀} \]

where \( P \) = intraventricular pressure from time \( t₀ \) (the beginning) to the end of the isovolumic period \( t₁ \) and \( V \) = volume of the left ventricle during the isovolumic period. Instead of the expression,

\[ \int_{t₀}^{t₁} P \, dt \]

it is possible to write \( P₁ \) expressing thus the mean intraventricular pressure during the isovolumic period. In order to simplify the calculations we assumed the left ventricle to be a sphere.

The oxygen consumption could then be re-
lated not only to the external work but to the sum of $E_1$ and $E_2$, which can be defined as the compound energy expenditure during the whole cardiac cycle. Since $E_1$, as a function of the energy expenditure is expressed in calories, $E_2$ was calculated in the same units.

To test this hypothesis these values have been calculated during increased peripheral resistance (aortic occlusion) and during increased cardiac output (induced muscular activity). The validity of the hypothesis that $E_1$ is an important factor in the estimation of cardiac energy has been tested by comparing the correlation between $QO_2$ and $E_2$, and $E_1 + E_2$ respectively. To ascertain that $QO_2$ is not altered by changes in the metabolic pattern, $RQ$ and consumption of basic metabolites, and substrates were also estimated.

**Methods**

Two series of experiments were performed on 27 mongrel dogs (18 to 28 kg) anaesthetized with pentobarbital sodium, 30 mg/kg iv. The chest was opened in the fourth intercostal space and the heart exposed in a pericardial cradle. Respiration was maintained by a pressure pump. The contractility of the left ventricle was recorded quantitatively by an inductance cardiomyographic method developed for this purpose. As shown in figure 1, a small high frequency coil (0.5 g) was sutured to the anterior wall of the left ventricle with a ligature. An iron-ferrite core moved inside the coil and was attached to the anterior wall by means of a nylon fish line. The core displacement was calibrated during and after the experiment. An additional measurement of the circumference of the left ventricle made it possible to extrapolate the recorded values and to calculate the external radius of the left ventricle. After completion of the experiment the internal volume of the left ventricle was estimated by measuring the volume of fluid in the heart under standard conditions while the displacement of the iron-core was recorded simultaneously. After calculating the internal radius, the mean radius $r = \frac{1}{2}(r_{int} + r_{ext})$ was used for further calculations.

Catheters for measuring pressure were introduced into the left ventricle and into the arch of the aorta. The outflow of the coronary sinus was measured by means of the local thermodilution method (LTD). The LTD catheter was introduced into the coronary sinus under X-ray fluoroscopic control. One estimation of coronary sinus outflow consisted of three injections of 2 ml saline at room temperature. The mean value was then calculated. The catheter placed into the left ventricle was used also for injections of cold saline for the estimation of cardiac output. A thermistor catheter was introduced into the arch of the aorta, and recorded the thermodilution curve resulting from the cold bolus, which was injected into the left ventricle. A balloon, fixed to the same catheter, made it possible to occlude the upper part of the descending aorta.

Blood samples were taken simultaneously from the coronary sinus and from the aorta for estimation of oxygen saturation by hemoreflexometry according to Zijlstra or by Natelson's volumetric micromethod. Analyses included also carbon dioxide, glucose, lactic acid and non-protein nitrogen. Hemoglobin was determined photometrically. Correlation coefficients were calculated in the usual way and the significance of changes of efficiency and of the indexes was determined by the Student t-test.

Two types of experiments were done. a) Peripheral resistance was increased by inflating the balloon in the thoracic aorta. b) Cardiac output was increased by muscular contractions produced by stimulating directly the muscles of both hind extremities with supramaximal shocks, 120 times per minute, for 10 minutes.

**Results**

1. **METABOLIC CHANGES DURING AORTIC OCCLUSION AND INDUCED MUSCULAR ACTIVITY**

Table 1 shows that no significant changes were found in $RQ$, consumption of glucose, lactic acid, and in the values of nonprotein nitrogen. The percentage of oxygen, used for...
ESTIMATION OF CARDIAC PERFORMANCE AND EFFICIENCY

CONTROL VALUES

oxidation of carbohydrates, was also similar during aortic occlusion and induced muscular activity.

2. CHANGES IN OXYGEN CONSUMPTION AND ENERGY OUTPUT DURING AORTIC OCCLUSION

On the other hand, figure 2 shows that the correlation coefficient, $r$, between oxygen consumption ($QO_2$) and energy output in the left ventricle during the isotonic period ($E_2$) is not very high ($r = 0.513, P < 0.05$). However, there is a much closer correlation between $QO_2$ and the sum of $E_1 + E_2$

![Figure 2](http://circres.ahajournals.org/)

**TABLE 1**

Consumption of Oxygen, Glucose, Lactic Acid, and the Values of Nonprotein Nitrogen (NPN) and Respiratory Quotient (RQ) in Cardiac Muscle Under Normal Conditions, During Aortic Occlusion and Induced Muscular Activity. Values Given in ml, or mg/100 g Tissue/min Respectively

<table>
<thead>
<tr>
<th>Experimental situation</th>
<th>Oxygen</th>
<th>Glucose</th>
<th>Lactic acid</th>
<th>NPN</th>
<th>RQ</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\bar{x}$</td>
<td>11.08</td>
<td>8.64</td>
<td>5.17</td>
<td>0.79</td>
<td>0.82</td>
</tr>
<tr>
<td>$\text{se}$</td>
<td>0.58</td>
<td>0.42</td>
<td>0.72</td>
<td>0.35</td>
<td>0.006</td>
</tr>
<tr>
<td>$n$</td>
<td>39</td>
<td>41</td>
<td>39</td>
<td>40</td>
<td>34</td>
</tr>
<tr>
<td>Aortic occlusion</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\bar{x}$</td>
<td>14.041</td>
<td>9.34</td>
<td>5.95</td>
<td>-0.96</td>
<td>0.77</td>
</tr>
<tr>
<td>$\text{se}$</td>
<td>0.98</td>
<td>1.58</td>
<td>1.01</td>
<td>0.95</td>
<td>0.072</td>
</tr>
<tr>
<td>$n$</td>
<td>17</td>
<td>16</td>
<td>16</td>
<td>16</td>
<td>12</td>
</tr>
<tr>
<td>Muscular activity</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\bar{x}$</td>
<td>13.641</td>
<td>10.9</td>
<td>4.09</td>
<td>0.23</td>
<td>0.77</td>
</tr>
<tr>
<td>$\text{se}$</td>
<td>0.88</td>
<td>1.59</td>
<td>1.54</td>
<td>1.22</td>
<td>0.051</td>
</tr>
<tr>
<td>$n$</td>
<td>14</td>
<td>14</td>
<td>13</td>
<td>14</td>
<td>10</td>
</tr>
</tbody>
</table>

$\bar{x}$: mean values; $\text{se}$: standard error; $n$: number of estimations. Values different significantly from normal denoted by $t$. 

*Circulation Research, Vol. XVI, June 1965*
Values of Energy Expenditure ($E_1$, $E_2$, and $E_1 + E_2$), Oxygen Consumption ($QO_2$) in cal·10⁻⁶ per beat; Coronary Sinus Outflow (SC) in ml/100 g/min; Mean Intraventricular Pressure During Isovolumic Period ($P_1$); Mean Aortic Pressure During Isotonic Period ($P_2$); Stroke Volume (SV); Cardiac Output (CO); and Radius of the Left Ventricle During the Isovolumic Period ($r$) Under Normal Conditions, During Aortic Occlusion and Induced Muscular Activity

<table>
<thead>
<tr>
<th>Experimental condition</th>
<th>$E_1$</th>
<th>$E_2$</th>
<th>($E_1 + E_2$)</th>
<th>$QO_2$</th>
<th>SC</th>
<th>$P_1$</th>
<th>$P_2$</th>
<th>SV</th>
<th>CO</th>
<th>$r$</th>
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</thead>
<tbody>
<tr>
<td>Normal</td>
<td>10.61</td>
<td>4.83</td>
<td>15.44</td>
<td>37.44</td>
<td>120.8</td>
<td>51.4</td>
<td>158.8</td>
<td>11.61</td>
<td>2230.2</td>
<td>2.42</td>
</tr>
<tr>
<td></td>
<td>1.02</td>
<td>0.48</td>
<td>1.43</td>
<td>2.69</td>
<td>8.67</td>
<td>3.1</td>
<td>5.1</td>
<td>1.14</td>
<td>222.1</td>
<td>0.05</td>
</tr>
<tr>
<td>Aortic occlusion</td>
<td>15.37</td>
<td>4.92</td>
<td>20.62</td>
<td>50.86</td>
<td>157.8</td>
<td>72.7</td>
<td>168.1</td>
<td>9.88</td>
<td>1803.0</td>
<td>2.48</td>
</tr>
<tr>
<td></td>
<td>1.15</td>
<td>0.42</td>
<td>1.50</td>
<td>4.27</td>
<td>9.92</td>
<td>1.99</td>
<td>4.8</td>
<td>0.85</td>
<td>173.2</td>
<td>0.044</td>
</tr>
<tr>
<td>Muscular activity</td>
<td>11.07</td>
<td>6.69</td>
<td>17.06</td>
<td>44.27</td>
<td>135.9</td>
<td>63.5</td>
<td>145.7</td>
<td>14.29</td>
<td>2830.2</td>
<td>2.34</td>
</tr>
<tr>
<td></td>
<td>1.23</td>
<td>0.79</td>
<td>1.87</td>
<td>2.67</td>
<td>10.64</td>
<td>5.35</td>
<td>11.2</td>
<td>1.36</td>
<td>335.0</td>
<td>0.006</td>
</tr>
</tbody>
</table>

Correlation between $QO_2$ and $E_1$, and ($E_1 + E_2$) respectively during aortic occlusion.

Correlation between $QO_2$, $E_1$, and $E_2$ during induced muscular activity.

Changes in Oxygen Consumption and Energy Output during Induced muscular activity.

Correlation between $QO_2$ and $E_1$, and $E_2$ during induced muscular activity.

Changes in Oxygen Consumption and Energy Output during induced muscular activity.

Correlation between $QO_2$, $E_1$, and $E_2$ during induced muscular activity.

CHANGES IN OXYGEN CONSUMPTION AND ENERGY OUTPUT DURING INDUCED MUSCULAR ACTIVITY

Figure 5 shows that, as in previous comparisons, the correlation between $QO_2$ and $E_2$ during induced muscular activity is not close ($r = 0.456$, $P < 0.001$). This difference is even more distinct during occlusion of the thoracic aorta ($r = 0.720$, $P < 0.001$).


table 2

$r = 0.456$ ($P < 0.001$). This difference is even more distinct during occlusion of the thoracic aorta ($r = 0.720$, $P < 0.001$). This difference is even more distinct during occlusion of the thoracic aorta ($r = 0.720$, $P < 0.001$).
relation between $QO_2$ and the sum of $E_1 + E_2$ is much closer ($r = 0.7596, P < 0.01$). This means that during increased cardiac output the energy expended during the isovolumic period participates significantly in the compound energy expenditure of the cardiac muscle.

4. CHANGES OF CARDIAC EFFICIENCY
In addition to the usual calculation of the mechanical efficiency, $\eta_m = E_2/QO_2$, it was

**OCCLUSION**

**FIGURE 4**
Correlation of changes in $QO_2$ ($\Delta QO_2$) and energy output, $\Delta E_2$ and $\Delta (E_2 + E_4)$ during aortic occlusion.

**INDUCED ACTIVITY**

**FIGURE 5**
Correlation between $QO_2$ and $E_2$ and $(E_2 + E_4)$ during induced muscular activity.
also possible to calculate a compound index of the chemomechanical turnover of the cardiac muscle, \( i_c = (E_1 + E_2)/QO_2 \), by taking into account the energy expended during the isovolumic period. Relating the amount of energy, delivered to the periphery, \( E_2 \), and considering the total compound energy released by the left ventricle, it is possible to estimate the pumping index of the left ventricle \( i_p = E_2/(E_1 + E_2) \).

During aortic occlusion (fig. 6) the mechanical efficiency and the pumping index, \( \eta_m \) and \( i_p \), respectively, are decreased. The compound index, \( i_c \), however, is not changed significantly, while during induced muscular activity neither \( \eta_m \) nor \( i_c \) changed, but \( i_p \) improved (fig. 6).

**Discussion**

The discrepancy between the greater increase of cardiac oxygen consumption during pressure-work in comparison with flow-work is evidently not caused by any marked change in the metabolic pathway of oxygen utilization. On the other hand, it was shown that the energy released during the isovolumic period plays a significant role in the total amount of energy expenditure of the cardiac muscle.

As has been stated above, the expression \( E_1 \) is considered to represent a function of energy dissipated during the isovolumic period. The definition of \( E_1 = V \cdot P \) may attract some criticism. Although the expression \( E_2 \) as external work presents no difficulty in applying the usual definitions taken from mechanics, difficulties arise when one wants to express the amount of energy developed during isovolumic conditions. Equations using tension or force seem to be inadequate, because the necessity of a dimension-correcting constant arises.

Therefore, \( E_1 \) was calculated as a function of energy expenditure during the isovolumic period; but this energy is nevertheless necessary for the subsequent ejection, during the isotonic period. When one calculates the compound energy expenditure as \( E_1 + E_2 \), it is possible that this value is overestimated, since a portion of \( E_1 \) might appear in \( E_2 \). It can be surmised, however, that this overestimation will not be very great, because if

![Aortic Occlusion and Muscular Activity](Attachment)

**FIGURE 6**

Cardiac efficiency during aortic occlusion and induced muscular activity.

*Circulation Research, Vol. XVI, June 1965*
the contraction cycle were stopped at the end of the isovolumic period, no blood would be ejected. This would imply that the energy required to eject blood is produced during the isotonic period, especially when taking into account the fact that despite the opening of the aortic valves the intraventricular pressure is steadily rising. In a recently published paper, Britman and Levine,25 using Hill’s series elastic model for muscle, derived an index for the contractile element work resembling the above expression.

As shown previously, the expression \( E_i + E_2 \) has a much closer correlation with \( QO_2 \) than \( E_2 \) only. It is therefore obvious that a considerable amount of the oxygen that is consumed during the whole cardiac cycle is used during the isovolumic period. This implies that \( E_i \) has to be considered when estimating cardiac performance. While the classical expression for efficiency, \( \eta_m = E_2/QO_2 \), expresses the overall mechanical external efficiency of the left ventricle, the “compound” index, \( i_c = (E_i + E_2)/QO_2 \), expresses the efficiency of the chemomechanical turnover. These two ratios (\( \eta_m \) and \( i_c \)) do not necessarily change in the same direction.

As is well known, mechanical efficiency decreases during increased peripheral resistance, e.g., aortic occlusion, without indicating if the change was due to impairment of the chemomechanical turnover of the cardiac muscle or by producing an adjustment of the source of energy to the peripheral load. An unchanged compound index, \( i_c = (E_i + E_2)/QO_2 \), during aortic occlusion indicates that the chemomechanical turnover was not affected in this case. When the cardiac output is increased by muscular activity, neither the mechanical nor the compound index changed. This is a result of a proportional increase of \( QO_2 \) and \( E_i, E_2 \) respectively. The ratio of the work delivered to the periphery, \( E_2 \), and the total amount of energy released by the left ventricle makes it possible to find an expression for the so-called “pumping index” of the left ventricle, \( i_p = E_2/(E_i + E_2) \). It indicates what amount of the total energy released by the left ventricle is being utilized for transport of blood to the periphery. Any disproportionate change of \( E_i \) or \( E_2 \) must be reflected in a change of \( i_p \). During aortic occlusion the increase of \( E_i \) was much higher than that of \( E_2 \) and consequently \( i_p \) was decreased (fig. 6). This decrease indicates that the matching between the heart (as a source of energy) and the periphery is affected. On the other hand, during induced muscular activity there is an increase of this ratio (fig. 6), which can be considered as an indication of an improved mutual matching, while the \( \eta_m \) and \( i_c \) do not disclose any change.

**Summary**

1) Changes of oxygen uptake and energy output by the left ventricle were investigated during increased peripheral resistance (aortic occlusion) and increased cardiac output (induced muscular activity) in open-chest dogs under pentobarbital anaesthesia.

2) Evidence has been provided to show that energy expenditure during the isovolumic period (\( E_i \)) participates significantly in the total oxygen consumption of the left ventricle (\( QO_2 \)). The correlation of \( E_i + E_2 \) (compound energy expenditure during the whole cardiac cycle) to \( QO_2 \) was closer than the correlation of the stroke work (\( E_2 \)) to \( QO_2 \) under the experimental conditions studied. The increase of \( QO_2 \) during increased peripheral resistance is due to a proportional increase of \( E_i \).

3) The determination of the total amount of compound energy released by the left ventricle, makes the estimation of the performance of the heart possible in a more specific way. Besides the classical mechanical efficiency, \( \eta_m = E_2/QO_2 \), an index of the chemomechanical turnover (compound index), \( i_c = (E_i + E_2)/QO_2 \), and the so-called pumping index of the left ventricle, \( i_p = E_2/(E_i + E_2) \), have been calculated. During increased peripheral resistance \( i_p \) did not change while \( i_p \) decreased, indicating that it is not the index of the chemomechanical turnover but the matching between the heart and the periphery which is impaired. On the other hand, during induced muscular activity, only \( i_p \) in-
creased, thus indicating an improvement of this matching.

Acknowledgment

The authors thank Dr. C. Kubie for valuable advice while preparing the project and Dr. L. H. Peterson and Ing. V. Rohlicka for valuable discussions of the paper. The help of Dr. A. Novakova, Mrs. A. Herbrychova, and Miss J. Salakova in different stages of the study is appreciated. The authors gratefully acknowledge the statistical calculations done by Ing. B. Burianova and Ing. K. Zvolankova.

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Estimation of Cardiac Performance and Efficiency During Aortic Occlusion and Induced Muscular Activity

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Circ Res. 1965;16:545-552
doi: 10.1161/01.RES.16.6.545

Circulation Research is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7330. Online ISSN: 1524-4571

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