Effect of Exercise on Left Ventricular Mechanical Efficiency in Conscious Dogs

Takashi Nozawa, MD; Che-Ping Cheng, MD, PhD; Toshiyuki Noda, MD; William C. Little, MD

Background We studied the effect of exercise (7.2 to 8.0 km/h) on the efficiency of the conversion of metabolic energy to external work or stroke work (SW) by the left ventricle (LV).

Methods and Results Energy use was calculated from LV myocardial oxygen consumption per beat (MV02). LV volume was calculated from orthogonal dimensions and coronary flow measured with ultrasonic flow probes. The total mechanical energy of the LV was calculated as the pressure-volume area (PVA). At rest, the MV02-PVA point fell on the MV02-PVA relation determined by steady-state changes in arterial pressure produced by graded infusions of phenylephrine. Exercise increased the slope (Ees) of LV end-systolic pressure-volume (PV) relation by 29%. During exercise, the MV02-PVA point shifted to the right only slightly above the control MV02-PVA relation by 0.007±0.005 mL O2 · beat⁻¹ · 100 g LV⁻¹. Despite the increase in ventricular contractility with exercise, the PVA/MV02 ratio was unchanged because of the marked increase in PVA. During exercise, the transmission of total mechanical energy to external work (SW/PVA) increased from 65±5% to 72±4% (P<.01) as the ratio of the arterial end-systolic elastance to Ees decreased from 1.1±0.2 to 0.8±0.1 (P<.05). Thus, LV mechanical efficiency (SW/MV02=SW/PVA · PVA/MV02) improved from 12.9±1.5% to 14.3±1.1% (P<.05) during exercise.

Conclusions Exercise increases the efficiency of conversion of metabolic energy to external work by the LV due to alteration in LV arterial coupling resulting in increased production of mechanical energy and enhanced transmission of mechanical energy to external work, which more than offsets any increased metabolic cost of the enhanced contractility. (Circulation. 1994;90:3047-3054.)

Key Words • oxygen consumption • stroke work • metabolic energy • contractility • exercise

In the absence of ischemia, the energy consumed by the left ventricle (LV) can be calculated from its oxygen consumption.1 The external work produced by the LV during each beat is the stroke work (SW), the area contained within the pressure-volume (PV) loop. Thus, the efficiency of the conversion of energy to useful work by the LV can be determined from the ratio of SW to myocardial oxygen consumption per beat (MV02). During exercise, the work of the LV increases as the cardiac output and arterial pressure both increase to meet the augmented metabolic needs of the body. This is manifest both as an increase in the SW produced by each beat and as an increased heart rate. It would be advantageous if this increased SW could be produced by the more efficient conversion of energy to external work (ie, increased mechanical efficiency). In previous studies by Lombardo et al2 and Gorlin et al,3 supine exercise increased measures of LV mechanical efficiency in healthy young subjects.

At a constant contractile state, MV02 is related to the LV PV area (PVA). PVA, which consists of SW and the area under the remaining portion of the LV end-systolic pressure-volume relation (ESPVR), represents the total mechanical energy produced by the LV.1,4 The mechanical efficiency (SW/MV02) of the LV can be expressed as the product of the ratio of PVA to MV02 (the conversion of metabolic energy to mechanical energy) and the ratio of SW to PVA (the conversion of mechanical energy to external work).5,6 The PVA and the portion of PVA expressed as SW depend on the LV end-diastolic volume, the contractile state (quantitated by the LV ESPVR), and the arterial system. We have recently found that the SW/PVA ratio increases during exercise.7 This effect should enhance LV mechanical efficiency. However, the increase in LV contractility during exercise would be expected to shift the MV02-PVA relation upward, increasing the MV02 associated with any PVA.8,9 This effect would tend to decrease mechanical efficiency during exercise. It is not known which effect predominates during exercise. Recently, Hayashida et al10 suggested that LV mechanical efficiency is nearly maximal for a given preload both at rest and during exercise. However, they did not directly measure MV02. Thus, the effect of exercise on LV mechanical efficiency is still unknown.

Accordingly, this study was undertaken to determine the effect of exercise on MV02, SW, and LV PV relations to assess the effect of exercise on the efficiency of LV conversion of metabolic energy to mechanical energy (PVA/MV02), the transmission of mechanical energy to external work (SW/PVA), and the resulting mechanical efficiency of the LV (SW/MV02).

Methods

Preparation

Seven healthy mongrel dogs weighing 23 to 38 kg were instrumented while they were under anesthesia induced with xylazine (2 mg/kg) and sodium thiopental (6 mg/kg IV) and...
maintained with halothane (0.5% to 2%). They were intubated and ventilated with oxygen-enriched room air to maintain arterial oxygen tension at more than 100 mm Hg. A sterile, left lateral thoracotomy was performed, and the pericardium was opened wide. A micromanometer pressure transducer (Konigsberg Instruments) and a polyvinyl catheter for transducer calibration and blood sampling were inserted into the LV through an apical stab incision. Three pairs of ultrasonic crystals (5 MHz) were implanted in the endocardium of the LV to measure the anteroposterior, septolateral, and base-apex (long-axis) dimensions, using the method we previously described.11-13 Ultrasonic time-transit flow probes (model 2R or 3R, Transonic System Inc) were placed on the left circumflex (LCx) and left anterior descending (LAD) coronary arteries for measurement of LV coronary flow. A catheter was inserted into the coronary sinus via the right atrium for coronary venous blood sampling. Two hydraulic occluder cuffs were placed around the superior and inferior venae cavae. The wires and tubing were tunneled subcutaneously and brought out through the skin of the neck.

Data Collection

Studies were performed after the animals made a full recovery from the instrumentation (1 to 2 weeks after the initial surgery). The LV catheter was connected to a pressure transducer (Statham P23Db) that was calibrated with a mercury manometer. The signal from the micromanometer was adjusted to match that of the catheter. The transit time of 5 MHz sound between the crystal pairs was determined and converted to a distance assuming a constant velocity of sound in blood of 1.55 m/s. The coronary flow probes were connected to an ultrasonic flowmeter (model T201, Transonic System Inc). The flow probe was calibrated at the factory with confirmed accuracy within ±10%. Coronary arterial and venous oxygen contents were measured with a hemoximeter (Co-Oximeter 482, Instrumentation Laboratory).

Protocol

Before exercise, steady-state data were collected during 12- to 15-second periods at rest to obtain heart rate, LV pressure and volume, and coronary flow while the dogs stood on a treadmill (Quinton, Inc). At the same time, LV and coronary sinus blood samples were taken for measurement of oxygen content. Then, transient vena caval occlusions were performed for 6 to 12 seconds to obtain the LV ESPVR. The animals were unsedated, and autonomic reflexes were intact.

After we collected the resting data, the dogs ran on a motorized treadmill. The treadmill speed was gradually increased from 4.0 km/h over 1 to 2 minutes to the submaximal level, 7.2 to 8.0 km/h, in steps of 0.8 km/h. We collected steady-state data and blood samples approximately 3 minutes after the last increase in exercise speed during steady-state submaximal exercise (Fig 1). Then, a transient caval occlusion was created for 6 to 12 seconds during exercise (Fig 2). This submaximal exercise level was sustained during data collection for both steady state and caval occlusion.

One or 2 days before the exercise study, the resting cardiac oxygen consumption per beat (MV02/PVA) relation was measured in all seven dogs. To avoid the influence of autonomic reflexes during changes in loading conditions, autonomic...
blockade was induced for this protocol with only hexamethonium chloride (5 mg/kg) and atropine sulfate (0.1 mg/kg), followed by their continuous infusions at rates of 0.1 mg/kg and 0.005 mg/kg per minute, respectively. LV loading conditions were widely varied with three or four graded doses of phenylephrine. After stabilization of all signals, steady-state data were recorded during 12 to 15 seconds, and thereafter the transient occlusions of venae cavae were created to obtain the ESPVR. The baseline data from five of these animals are reported elsewhere.9

Data Processing and Analysis

Data were digitized, using an on-line analog-to-digital converter (Data Translations Devices) at 200 Hz during each 12- to 15-second collection period. The LV volume was calculated as a modified general ellipsoid using the following equation:

$$V_{LV} = \frac{(\pi/6) \cdot D_{AP} \cdot D_{SL} \cdot D_{LA}}{}$$

where $V_{LV}$ is LV volume, $D_{AP}$ is the anteroposterior LV diameter, $D_{SL}$ is the septolateral diameter, and $D_{LA}$ is the long-axis LV diameter. We have previously demonstrated that this method gives us a consistent measure of LV volume despite changes in LV loading conditions, configurations, and heart rate.11,14,15

End systole was defined as the time of the peak instantaneous ratio of LV pressure to volume. The PVA was determined as the area under the ESPVR line and the systolic PV trajectory and above the end-diastolic PV relation curve. The ESPVR obtained during transient vena caval occlusion was superimposed on the steady-state PV loop that was averaged over the 12- to 15-second recording period (Fig 2). We assumed that end-diastolic PV relation curve was reasonably approximated by a third-order polynomial. Then, the small area of PVA between the straight line connecting the volume axis intercept ($V_0$) of ESPVR and end-diastolic point ($V_{ed}, P_{ed}$) and the downward convex end-diastolic PV curve was given by $P_{ed} \cdot (V_{ed} - V_0)/4$. To evaluate the effect of nonlinearity of the ESPVR, the end-diastolic points were also fitted by a quadratic function curve ($P_{ed}=a \cdot V_{ed}^2 + b \cdot V_{ed} + c [a, b, c$ are constants, respectively]), and PVA was calculated from this nonlinear ESPVR. The product of the coronary flow (mL/min) and coronary arteriovenous oxygen content difference (vol%) divided by 100 gives oxygen consumption rate in milliliters of O2 per minute. This was divided by the heart rate to yield oxygen consumption in milliliters of O2 per beat. Total LV oxygen consumption was calculated as (total LV weight)/ (perfused LV weight distal to both LCx and LAD flow probes) multiplied by the measured oxygen consumption. To make PVA/MVO2 and SW/MVO2 dimensionless, we converted mm Hg mL of PVA and SW and O2 mL of MVO2 to joules by using 1 mm Hg mL = 1.33 x 10^-6 J and 1 mL O2=20 J.

In the preliminary study with the same instrumentation, we measured arterial and coronary sinus lactate levels before and during exercise (7.2 km/h) in two dogs. The lactate extraction ratio (ie, coronary arteriovenous lactate difference/coronary artery lactate) was 35.8±1.5% at rest and 37.8±8.6% during exercise. A systemic adenosine infusion at rest increased both LCx and LAD coronary flows more than threefold. These results demonstrate that our instrumentation did not limit coronary flow or produce cardiac ischemia during exercise.

At the conclusion of the experiments, the dogs were anesthetized with sodium thiopental (6 mg/kg) and halothane (0.5% to 2%) and a thoracotomy was performed to determine the perfusion territories of LCx and LAD using saturated Evans’ blue solution. The dogs were euthanized with T-61 euthanasia solution (0.3 mL/kg IV) after the injection of the Evans’ blue solution. The hearts were examined to confirm the proper positioning of the instrumentation. Then, the stained

**Fig 2.** Variable loaded pressure-volume loops were produced by caval occlusion both at rest and during exercise (left panels). Top left corner of loops defines the left ventricular (LV) end-systolic pressure-volume relation (ESPVR). This ESPVR line was superimposed on the steady-state pressure-volume loop (right panels). The pressure-volume area (PVA) was calculated as the sum of the external/stroke work (SW) and the area under the remainder of the ESPVR (PE).
and nonstained portions of the LV were weighed. The ratio of the perfused LV weight to the total LV weight was 0.57±0.05.

**Statistical Analysis**

Data obtained from dogs at rest and during exercise were compared using two-tailed paired t tests. Linear regression analysis was applied to obtain the regression line of MVO₂ on PVA. Values are given as mean±SD.

**Results**

Fig 1 shows analog recordings of LV pressure (LVP), ventricular diameters (Dₘ, anterioposterior diameter; Dₛ, septolateral diameter; Dₐ, long-axis diameter), ventricular volume (LVV), coronary flows (for LCx or LAD) in dogs at rest and during exercise. During exercise, the heart rate, LVP, and both coronary artery flows increased.

Group values of hemodynamic, PV, and cardiac oxygen consumption indexes are summarized in Tables 1, 2, and 3. An example of PV loops at steady state and transient caval occlusion before and during exercise are shown in Fig 2. Exercise increased the heart rate by an average of 45 beats per minute and end-systolic pressure by an average of 20 mm Hg. During exercise, LV end-diastolic volume slightly but significantly increased.

### Table 1. Hemodynamic Data at Rest and During Exercise

<table>
<thead>
<tr>
<th></th>
<th>HR, bpm</th>
<th>EDV, mL</th>
<th>ESV, mL</th>
<th>SV, mL</th>
<th>EDP, mm Hg</th>
<th>ESP, mm Hg</th>
<th>dP/dtₑₓₘₐₓ, mm Hg/s</th>
<th>-dP/dt, mm Hg/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td>128±14</td>
<td>41.0±8.7</td>
<td>27.6±6.3</td>
<td>13.5±3.4</td>
<td>11±4</td>
<td>110±7</td>
<td>2872±261</td>
<td>-2536±166</td>
</tr>
<tr>
<td>Exercise</td>
<td>173±7*</td>
<td>43.7±8.0†</td>
<td>28.0±5.9†</td>
<td>15.7±3.2*</td>
<td>13±4†</td>
<td>130±6*</td>
<td>3940±252*</td>
<td>-3287±190*</td>
</tr>
</tbody>
</table>

HR indicates heart rate; bpm, beats per minute; EDV, end-diastolic volume; ESV, end-systolic volume; SV, stroke volume; EDP, end-diastolic pressure; and ESP, end-systolic pressure.

*P<.01, †P<.05, ‡NS.
whereas end-systolic volume was unchanged; thus, the
stroke volume increased significantly by 16%. Exercise
enhanced the LV contractile state as determined by a
29% increase in $E_a$ and a 37% increase in $dP/dt_{max}$.
Both external work (SW) and PVA increased signifi-
cantly during exercise.

Coronary flow at rest was 81.5±10.6 mL/min/100
g⁻¹·LV and increased significantly by 51% during ex-
ercise. Coronary artery hemoglobin concentration
increased from 11.0±0.9 to 11.8±0.6 g/dL during exercise
($P<.05$). Coronary artery oxygen saturation was
96.1±1.0% at rest and was unchanged. Coronary ven-
ous oxygen saturation decreased from 21.7±3.6% to
14.9±1.9% during exercise ($P<.01$). The coronary
arteriovenous oxygen content difference increased signif-
cantly during exercise due to a decrease in coronary
venous oxygen content and an increase in arterial
oxygen content. Exercise increased cardiac oxygen
consumption per minute by 72% and per beat ($MVo2$) by
25%.

As shown in Table 4 and Fig 4, the ratio of SW to
PVA (SW/PVA) was 65.0±5.0% at rest and increased
significantly to 72.1±3.9% during exercise. The ratio of
PVA to $MVo2$ was 19.8±1.3% at rest and unchanged
(19.9±1.3%) during exercise. The ratio of SW to $MVo2$
(SW/$MVo2$), i.e., the mechanical efficiency, was 12.9±1.5
% at rest and increased to 14.3±1.1% during exercise
($P<.05$). PVA values calculated from nonlinear ESPV
were only slightly smaller than those from a linear
approximation of its ESPVR. The SW/PVA ratio were
slightly (<5%) increased and the PVA/$MVo2$ ratio was
slightly decreased (<2%) at both rest and exercise
as compared with that obtained ($P=NS$) from the linear
ESPVR (Table 4).

The $MVo2$-PVA relation was obtained at rest by chang-
ing LV loading conditions with phenylephrine after auto-
nomic blockade to prevent reflex changes in contractile
state. $E_a$ was nearly constant ($P=NS$) regardless of
changes in loading conditions in each heart, and the
coefficient of variation (standard deviation/mean value)
was ±6%±0.3. The value of the blocked $E_a$ (8.3±1.7
mm Hg/mL) was almost identical to the value (8.2±2.1
mm Hg/mL) at rest before exercise. After blockade, $MVo2$
was linearly correlated with PVA in all dogs with the
correlation coefficient of 0.862±0.013 (Fig 3). The slope
and $MVo2$ intercept of the $MVo2$-PVA regression line was
(2.19±0.30×10⁻³ mL O₂·mm Hg⁻¹·mL⁻¹ and
0.0247±0.0038 mL O₂/100 g LV, respectively. The slope
and $MVo2$ intercept calculated from the nonlinear ESPV
were similar to the linear values, 2.32±0.31×10⁻³ mL O₂·mm Hg⁻¹·mL⁻¹ and 0.0246±0.0046 mL O₂/100 g
LV, respectively, with the correlation coefficient of
0.866±0.014.

Fig 3 shows the response to exercise in the
$MVo2$-PVA plane in all hearts. Before exercise, each
data point was on or near the blocked $MVo2$-PVA
relation. $MVo2$ at rest was 0.0731±0.0195 mL
O₂·beat⁻¹·100 g⁻¹·LV and was very close to the value
calculated from the $MVo2$-PVA regression line at the
same PVA value before exercise (0.0732±0.0179 mL
O₂·beat⁻¹·100 g⁻¹·LV, $P=NS$). In 6 of the 7 animals,
the resting $MVo2$-PVA point fell within the 95% confi-
dence intervals of the blocked $MVo2$-PVA relation.
During exercise, the $MVo2$-PVA point shifted to the
right with increases in both $MVo2$ and PVA. In 2
animals, the $MVo2$-PVA point during exercise was
above the 95% confidence limits of the blocked
$MVo2$-PVA relation, and in the other 5 it fell within the
95% confidence limits. $MVo2$ during exercise was
0.0916±0.0191 mL O₂·beat⁻¹·100 g⁻¹·LV and was
slightly but significantly ($P<.05$) higher than the value
calculated from the resting $MVo2$-PVA line at the same
PVA value (0.0851±0.0178 mL O₂·beat⁻¹·100 g⁻¹·LV).

Discussion
During exercise, both the LV SW and heart rate
increased. It would be advantageous if the increased SW

<table>
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<th>Table 2. Pressure-Volume Data at Rest and During Exercise</th>
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<td>$E_a$ mm Hg/mL</td>
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<tr>
<td>Rest</td>
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<td>Exercise</td>
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$E_a$ indicates slope of end-systolic pressure-volume relation line (ESPVR); $E_v$, effective arterial elastance; $V_o$, volume-axis intercept of ESPVR; SW, stroke work; and PVA, pressure-volume area.

Data in parentheses were calculated from nonlinear ESPVR.

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<table>
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<tr>
<th>Table 3. Cardiac Oxygen Consumption at Rest and During Exercise</th>
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<td>Flow, mL/min. 100 g⁻¹·LV</td>
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<tr>
<td>Rest</td>
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<td>Exercise</td>
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FLOW indicates coronary artery flow; A-COUNT, arterial oxygen content (CONT); V-COUNT, coronary venous CONT; A-V CONT, coronary arteriovenous CONT difference; and $MVo2$, oxygen consumption.

$P<.01$, †$P<.05$. 
During exercise, the increase in MVo2 and found exercise. In contrast, we directly measured PVA/MVo2 and found it to be unaltered during exercise. Thus, the increase in SW/PVA resulted in an enhancement of SW/MVo2 during exercise.

An increase in ventricular contractility, as occurs during exercise, is accompanied by an augmentation of the energy use for intracellular calcium handling. This is apparent as an upward shift of the MVo2-PVA relation, as expected to occur during exercise, would decrease the ratio of PVA to MVo2 at constant PVA, indicating an increase in the total energy cost to produce mechanical energy. During exercise, in the present study, LV contractility was enhanced as indicated by an increase in Eps of 2.3 ± 1.0 mm Hg/mL. Because the MVo2 intercept increases by 2.4 ± 10^{-3} mL/O2 for each mm Hg/mL increase in Eps in isolated canine hearts, we predict an upward shift in the MVo2-PVA relation by 0.005 mL · O2^{-1} · beat^{-1} during exercise in the present study. We found that the PVA/MVo2 point during exercise was 0.007 ± 0.005 mL O2 · beat^{-1} · 100 g^{-1} LV above the control MVo2-PVA line. Thus, our observation is consistent with a contractility-induced upward shift of the MVo2-PVA relation. However, the magnitude of the increase in MVo2 at constant PVA during exercise is small, and although the mean value was significantly increased, in only 2 of the 7 animals did the exercise point fall outside of the 95% confidence limits of the control MVo2-PVA regression line. Thus, the present study does not have the power to define whether the MVo2-PVA relation is shifted during exercise in conscious animals. However, the present study does clearly demonstrate that PVA/MVo2 is not altered during exercise. This ratio, PVA/MVo2, representing the overall efficiency of production of mechanical energy, should be differentiated from the slope of the MVo2-PVA relation. This slope indicates the increment of metabolic energy required to produce an increment of mechanical energy. The total MVo2 consumed at a given PVA consists of the sum of the MVo2 intercept and the incremental MVo2 to generate PVA. Thus, PVA/MVo2 increases with increasing PVA. The marked increase in PVA we observed during exercise offset any upward shift of the MVo2-PVA relation so that PVA/MVo2 (indicating the efficiency of conversion of metabolic energy to mechanical energy by the myocardium) was not altered during exercise (Fig 3).

The other factor that influences SW/MVo2 is SW/PVA, the portion of the total mechanical energy that results in external work. SW/PVA is determined by the coupling of the LV and the arterial system. This coupling can be quantitated as the ratio of the effective arterial elastance (Ea) to Eps. Ea is calculated as the end-systolic pressure/stroke volume and is approximated by the product of peripheral vascular resistance and heart rate. If the ejection portion of the LV PV loop is assumed to be flat and the end-diastolic pressure is negligible, Burkoff and Sagawa showed that SW/PVA = 1/(1 + 0.5 · Eps/Ea). We have recently found that despite the limitations of the simplifying assumptions, this prediction is a very accurate description of the intact cardiovascular system of conscious animals. In the present study, Eps increased during exercise, whereas Ea remained constant. Thus, during exercise SW/PVA increased. Because PVA represents the total mechanical energy produced by each LV contraction, this indicates that more of this mechanical energy is expressed as useful external work (SW) during exercise.

Thus, during steady-state, submaximal exercise, the efficiency of the conversion by the LV of metabolic energy (MVo2) to external work (SW) was enhanced by the increase in PVA and the expression of more of the mechanical energy (PVA) as external work (SW).

There are several methodological issues to be considered. First, we measured LAD flow distal to the large first septal branch and assumed that coronary flow per unit mass is uniform within the LV. Consistent with this assumption, both LCx and LAD flows changed in a parallel manner during exercise. The resting coronary flow was 81.5 mL · min^{-1} · 100 g^{-1} LV, consistent with earlier studies. Furthermore, we have found that adenosine increases both coronary flows more than three times and that coronary arteriovenous lactate
Effect of Exercise on Cardiac Mechanical Efficiency

Nozawa et al

We measured MVO₂ to calculate LV energy use. Different substrates may have different energy yields when oxidized. Exercise enhances myocardial glucose uptake and exogenous glucose oxidation, probably related to the catecholamine changes, although free fatty acid uptake is also enhanced during exercise. However, the maximum possible difference in energy output from MVO₂ of glucose and free fatty acids is about 4%. Thus, any alteration in substrate use during exercise should not have influenced our calculation of mechanical efficiency. Furthermore, a shift from fatty acid to glucose use would be expected, based on the study of Burkhoff et al, to produce a downward shift of the MVO₂-PVA relation. In contrast, we observed an upward shift with exercise.

We used three-dimensional sonomicrometry for LV volume measurement. This method has been extensively validated in past studies and accurately reflects LV volume under a wide variety of normal and pathological conditions. To account for respiratory changes in intrathoracic pressure, data were averaged over the 12- to 15-second recording period that spanned multiple respiratory cycles. The instrumentation may produce some depression of LV performance. This may be accounted for the relative low ejection fraction we observed, consistent with previous studies in conscious, instrumented dogs.

The full MVO₂-PVA relation cannot be determined in a conscious animal in the presence of intact reflexes because the required steady-state alterations in load result in reflex alterations in contractility. Accordingly, we defined the control MVO₂-PVA relation after autonomic blockade. The resting MVO₂-PVA point, determined with intact reflexes, fell close to the blocked MVO₂-PVA relation. This is consistent with previous observations of the lack of an effect of adrenergic tone on LV function in conscious animals at rest.

We studied the effect of submaximal steady-state exercise (7.2 to 8.0 km/h), so our results may not apply to more strenuous exercise. At higher levels of exercise, we would expect greater increases in heart rate, arterial pressure, Eₛ, SW, PVA, and MVO₂. We speculate that this would result in an even greater increase in LV mechanical efficiency as a more marked decrease in Eₑ/Eₑₑ, resulted in a higher SW/PVA and PVA, which would continue to offset the more marked upward shift of the MVO₂-PVA relation due to a greater increase in contractility.

We were only able to measure one MVO₂-PVA point during exercise, so we are not able to detect whether exercise altered the slope of the MVO₂-PVA relation. Burkhoff et al recently observed that an increase in ejection fraction in physiologically loaded isolated canine hearts decreased the slope. Such an effect may occur during exercise.

We conclude that exercise increases the efficiency of conversion of metabolic energy to external work by the LV due to alteration in LV arterial coupling resulting in increased production of mechanical energy and enhanced transmission of mechanical energy to external work, which more than offsets the increased metabolic cost of the enhanced contractility.

Acknowledgments

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