Energy Conversion Efficiency in Human Left Ventricle

Tomoki Kameyama, MD; Hidetsugu Asanoi, MD; Shinji Ishizaka, MD; Kazuo Yamanishi, MD; Masatoshi Fujita, MD; and Shigetake Sasayama, MD

Background. Left ventricular mechanical efficiency is one of the most important measures of left ventricular pump performance. Several clinical studies, however, have shown that mechanical efficiency does not fall substantially as the heart fails. To clarify the insensitivity of mechanical efficiency to the change in pump performance, we analyzed human left ventricular mechanical efficiency, applying the concept of left ventricular systolic pressure–volume area (PVA).

Methods and Results. PVA correlates linearly with myocardial oxygen consumption per beat (MVo2): MVo2=a·PVA+b, and represents the total mechanical energy of contraction. We determined MVo2–PVA relation and external work in 11 patients with different contractile states. We also calculated the energy transfer from MVo2 to PVA (PVA/MVo2 efficiency), that from PVA to external work (work efficiency), and mechanical efficiency (external work/MVo2). Left ventricular pressure–volume loops were constructed by plotting the instantaneous left ventricular pressure against the left ventricular volume at baseline and during pressure loading. The contractile properties of the ventricle were defined by the slope of the end-systolic pressure–volume relation (Ees). Pressure elevation raised external work by 41.4%, PVA by 71.2%, and MVo2 by 54.5%. These changes were associated with a decrease in work efficiency and an increase in PVA/MVo2 efficiency. The opposite directional changes in these two efficiencies rendered the mechanical efficiency constant. The slope, a, of the relation between MVo2 and PVA was relatively constant (2.46±0.33) over the range of 0.8–8.8 mm Hg/ml of Ees, but the oxygen axis intercept, b, tended to decrease with the reduction in Ees. PVA/MVo2 efficiency correlated inversely (r=−0.66, p<0.05) with Ees, whereas work efficiency correlated linearly with Ees (r=0.91, p<0.01).

Conclusions. Mechanical efficiency is not appreciably affected by changes in loading and inotropic conditions as long as the left ventricular contractility is not severely depressed. (Circulation 1992;85:988–996)

Key Words • pressure–volume relation • oxygen consumption • pressure–volume area

Mechanical efficiency, one of the most important parameters of the energy transfer system, designates the fraction of total consumed energy that is converted into external work (EW).1,2 Previous clinical studies,3–5 however, have documented that if the contractile state of the ventricle is not severely depressed, left ventricular mechanical efficiency does not change substantially in patients with cardiac dysfunction. The mechanism for the insensitivity of mechanical efficiency to cardiac function has not been systematically examined in clinical settings because of the lack of a suitable analytical framework. Sugato and Suga et al7 have shown that ventricular pressure–volume area (PVA) represents the total mechanical energy of contraction (Figure 1), and it correlated linearly with myocardial oxygen consumption per beat (MVo2). The PVA has a great advantage over the previous predictors of mechanical energy utilization in that left ventricular mechanical performance is integrated into the PVA and can be quantitatively related to MVo2 on the same dimensional basis.8 Therefore, the PVA concept provides a useful framework to analyze the relation between the loading and inotropic conditions and mechanical efficiency of the diseased left ventricle. So far, mechanical energy transfer using the PVA concept has been examined only under unphysiological states such as excised hearts or anesthetized animal models.6–9

In the present study, we have extended the analysis of efficiency of energy transfer6,8 for the first time to human left ventricles. To clarify the insensitivity of mechanical efficiency to cardiac deterioration, we have examined, in patients with normal and moderately depressed hearts, how the changes in contractile state and afterload influence the MVo2–PVA relation and mechanical efficiency.

Methods

Study Patients

The study was performed in 11 patients (mean age, 50 years; range, 36–62 years). Six patients had previous myocardial infarction, four had cardiomyopathy (two, idiopathic and two, after myocarditis), and one had atypical chest pain. Patients who showed clinical symp-
diagrams and signs of new myocardial ischemia on treadmill exercise test or exercise $^{59}$Tl myocardial perfusion scans were excluded from the study, as were patients with left ventricular aneurysm or mitral regurgitation. All patients were in normal sinus rhythm, and all medications were withheld 24 hours before the procedure. The study protocol was reviewed and approved by the Ethical Committee of our institute, and informed written consent was obtained from each patient. There were no complications as a result of the study.

**Catheterization Procedure**

Cardiac catheterization was performed by the right brachial approach with the patients in a fasting state. After conventional diagnostic right and left heart catheterization, coronary arteriography was performed by the Sones technique. Proximal coronary sinus catheterization was performed with a dual-thermistor thermodilution catheter (Webster Laboratory Inc.). A high-fidelity micromanometer-tipped catheter (Micro-Tip, Millar Instruments) was then introduced into the left ventricle, which allowed simultaneous high-fidelity pressure measurement during left ventriculography. After sufficient time was allowed after coronary arteriography, coronary sinus blood flow was determined in duplicate by the standard thermodilution method. The position of the catheter was checked frequently during the study by both fluoroscopic appearance and hand injection of small doses of contrast medium to confirm the proximal thermistor placed directly into the ostium of the coronary sinus. Arterial and coronary sinus blood samples were drawn simultaneously for the determination of oxygen saturation and lactate concentration. Left ventricular cineangiography was then performed in the 30° right anterior oblique projection with a Toshiba 9-in. (22.86-cm) image intensification system. Left ventricular opacification was achieved by injecting 35–40 ml of radiopaque nonionic contrast agent (iopamidol) through a Millar angiographic catheter at a rate of 12 ml/sec. Films were exposed at a rate of 60 frames/sec with an Arriflex 35 mm cine camera while the patient was gently holding his breath. During the cineangiographic study, high-fidelity left ventricular pressure, ECG, cineangiographic frame markers, and an injection marker were recorded simultaneously.

An adequate recovery time was allowed for the left ventricular pressure to return to baseline level. Phenylephrine (5 mg/100 ml) was started to increase the systolic left ventricular pressure by about 50 mm Hg. In nine patients, the heart rate was kept almost the same as in the control state by coronary sinus pacing. Measurements of coronary sinus flow and samplings of coronary sinus blood were taken again under steady-state conditions during pressure elevation. After these measurements, the second cineventriculogram was obtained in the same manner as in the control state. In seven patients, myocardial lactate uptake was calculated as follows: myocardial lactate uptake = (arterial lactate – coronary sinus lactate)×100/arterial lactate. There were no signs of pulmonary congestion, appearance of a transient mitral regurgitation, or other side effects during these procedures.

**Generation of Angiographic Pressure–Volume Diagrams**

The boundary of the ventricular silhouette was delineated manually by Oscon cine analyzer. Left ventricular

![Figure 1](http://circ.ahajournals.org/)

**Figure 1.** Schematic of pressure–volume (P–V) relations of left ventricle (left panel) and P–V area (PVA) (right panel). Three P–V loops of ejection contractions are shown in the left panel. The solid circles at the left upper corners of the loops are the end-systolic P–V points. The line through these points is the end-systolic P–V line, and its slope is Ees. Diastolic P–V curve consists of the diastolic segment of these P–V loops. Effective arterial elastance, Ea, is the slope of end-systolic pressure–stroke volume (P–SV) relation. The origin of the relation line, that is, its volume axis intercept, is a given end-diastolic volume. The stroke volume is represented on this ventricular volume axis as a distance to the left of this intercept. When the ventricle is coupled with the arterial system, the equilibrium is determined as the intersection between the arterial end-systolic P–SV line and the ventricular end-systolic P–V line. PVA is the area in the P–V diagram that is circumscribed by the end-systolic P–V line, the end-diastolic P–V relation curve, and the systolic segment of P–V trajectory (E-A-B-C-E, right panel). PVA consists of the external work (EW) performed during systole and the end-diastolic elastic potential energy (PE) stored in the ventricular wall at end systole. EW is the area within the P–V loop trajectory (A-B-C-D-A), and PE is the area between end-systolic P–V line and end-diastolic P–V relation curve to the left of EW (E-C-D-E).
Given a constant heart rate, arterial end-systolic pressure changes with stroke volume in a roughly linear relation. The slope of this relation is in proportion to the impedance that the arterial tree offers to the stroke flow. Thus, the arterial properties can be represented as a first approximation by the arterial end-systolic pressure–stroke volume relation. Sunagawa et al\(^4\) called this slope effective arterial elastance (Ea). We determined Ea as the ratio of end-systolic pressure to stroke volume both at rest and during increased afterload.

**Left Ventricular Work and Work Efficiency**

We measured PVA by planimetry at baseline and during increased afterload and defined it as the area within the straight line connecting volume axis intercept (V0) of the end-systolic pressure–volume line and the end-systolic point, end-diastolic pressure–volume relation curve, and systolic segment of the pressure–volume trajectory. PVA in an ejecting contraction consists of two parts (Figure 1, right panel).\(^6\) One is the area within the pressure–volume loop trajectory (A-B-C-D-A), which equals left ventricular EW. The other is the area between the end-systolic and end-diastolic pressure–volume relation curves to the left of EW (E-C-D-E). This area is considered equal to end-systolic elastic potential energy built during systole and stored in the ventricular wall. We analyzed the ratio of EW to PVA, which represents the efficiency of energy transfer from the ventricle to the arterial system. This ratio was termed work efficiency in the present article.\(^15\)

**Mechanical Efficiency and MVo2–PVA Relation**

Oxygen contents of arterial and coronary sinus blood were calculated as the product of the percent oxygen saturation, oxyhemoglobin binding capacity, and hemoglobin concentration. MVo2 was calculated as the product of coronary sinus blood flow and the arteriocor- nary sinus oxygen difference and was expressed on a per beat basis. Because the energy units of EW and MVo2 are expressed by mm Hg·ml and ml O\(_2\), respectively, these units were converted into a common unit of energy, joules (J), by use of the following conversions: 1 mm Hg·ml = 1.33×10\(^{-4}\) J and 1 ml O\(_2\) = 20 J.\(^16\) Then mechanical efficiency was expressed conventionally by the ratio of EW to MVo2. For each patient, MVo2 was plotted as a function of PVA in the basal and high-pressure states. The slope, a, and oxygen axis intercept, b, of the relation between MVo2 and PVA were determined from a straight line connecting these two points (i.e., MVo2 = a·PVA + b) in the same manner as determined by Burkhoff et al (Figure 2, lower panel).\(^17\)

**Statistical Analysis**

Data were expressed as mean±SD. Because the patients served as their own controls, the statistical significance of differences in hemodynamic variables was tested by paired \(t\) test. Values of \(p<0.05\) were considered to represent a statistical significance. The interrelations between efficiency parameters and basal inotropic state were quantified by linear regression analysis.

**Results**

The data for all patients are listed in Table 1. The left ventricular end-systolic pressure increased by 54.6±20.9
mm Hg with phenylephrine, associated with 35.2±22.9% increase in Ea. There were significant augmentations of end-diastolic volume and end-systolic volume and reduction in ejection fraction. Coronary sinus blood flow increased by 48.1±26.9%, but there were no significant changes in arteriocoronary sinus O$_2$ difference. Consequently, myocardial oxygen consumption per minute increased by 49.0±25.9%. Myocardial lactate uptake also increased in all seven patients, and there were no signs of myocardial ischemia on the ECG during these interventions.

Representative pressure-volume loops are shown in Figure 2. Individual data on the left ventricular work and energy transfer are listed in Table 2. With pressure elevation, left ventricular EW increased by 41.4±34.3%, and end-systolic potential energy was doubled. The PVA (sum of the area of EW and potential energy) increased by 71.2±33.6%. These changes were accompanied by a decrease in work efficiency (EW/PVA) but an increase in PVA/MVO$_2$ efficiency. Consequently, mechanical efficiency (EW/MVO$_2$) did not change appreciably.

Ees ranged from 0.81 to 8.8 mm Hg/ml, and left ventricular ejection fraction ranged from 30% to 70%. Only four of the patients had depressed ejection fraction (<50%), which did not necessarily correlate with Ees (r=0.48, p>0.10). Figure 3 shows the scatter diagram of slope coefficient, a, and oxygen axis intercept, b, of MVO$_2$-PVA relation against Ees. The reciprocal of the slope, 1/a, reflects the efficiency with which oxygen is utilized to generate mechanical energy. This value was constant, with a mean value of 41% over the wide range of Ees values. The intercept, b, tended to decline with depression of contractile state. Figure 4 shows PVA/MVO$_2$ efficiency and work efficiency plotted as a function of Ees. The former correlated inversely with Ees (r=-0.649, p<0.05) and the latter linearly (r=0.907, p<0.01). Consequently, because of counter-balance of these two efficiencies, mechanical efficiency (EW/MVO$_2$) remained unchanged (22.1±4.2%) within the range of Ees or ejection fraction of the patients studied (Figure 5).

**Discussion**

The primary determinants of MVO$_2$ are the preload and afterload and the contractile state of the left ventricle. PVA is the first and intermediate form of total mechanical energy, which allows us to relate these parameters of cardiac mechanical performance to MVO$_2$ on the same dimensional basis. Tension–time...
TABLE 2.  Left Ventricular Work and Efficiency

<table>
<thead>
<tr>
<th>Patient</th>
<th>EW (mm Hg·ml)</th>
<th>PE (mm Hg·ml)</th>
<th>PVA (mm Hg·ml)</th>
<th>PVA/MVO₂ (%)</th>
<th>EW/PVA (%)</th>
<th>EW/MVO₂ (%)</th>
<th>a (%)</th>
<th>1/a (%)</th>
<th>b (%)</th>
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<tr>
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<td>9,781</td>
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<td>1,937</td>
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<td>72</td>
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<tr>
<td>Mean C</td>
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<td>±1,115</td>
<td>±1,697</td>
<td>±7</td>
<td>±10</td>
<td>±4</td>
<td>±0.33</td>
<td>±6</td>
<td>±0.63</td>
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<tr>
<td>SD</td>
<td>±1,423</td>
<td>±1,115</td>
<td>±1,697</td>
<td>±7</td>
<td>±10</td>
<td>±4</td>
<td>±0.33</td>
<td>±6</td>
<td>±0.63</td>
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</table>

EW, left ventricular external work; PE, end-systolic potential energy; PVA, pressure-volume area; PVA/MVO₂, PVA/myocardial oxygen consumption efficiency; EW/PVA, work efficiency; EW/MVO₂, mechanical efficiency; a, slope of the PVA–MVO₂ relation; 1/a, reciprocal of slope a; b, oxygen axis intercept of PVA–MVO₂ relation; J, joule; C, control; P, phenylephrine.

index, peak pressure, and stroke work, which are proposed as clinical predictors of MVO₂, can change considerably despite an absence of change in MVO₂. Myocardial peak force and contractile state have been viewed as the major predictors of energy consumption. MVO₂ for a given peak force increases considerably, however, with shortening and ejection. Weber and Janicki showed that the time integral of total systolic force correlated linearly with MVO₂ for both ejecting and isovolumetric contractions in canine left ventricle. This parameter, however, is not equivalent to the total mechanical energy generated by ventricular contraction, as PVA is. Moreover, Suga et al have recently demonstrated that MVO₂ remained constant when the force–time integral was greatly changed by reciprocally changing afterloaded pressure and end-diastolic volume while keeping PVA constant in a stable contractile state. The linear muscle version of PVA, which was called force–length area, has also been shown to correlate closely with MVO₂ in ferret papillary muscles and with total heat generated in rabbit papillary muscles. All these results support the feasibility of PVA as a linear and close correlate of MVO₂.

As far as we know, this is the first study to quantitatively describe the influence of ventricular contractile state and afterload on efficiency of energy transfer in human left ventricle. The present study showed that, with the depression of contractile state, there arose an increase in energy conversion from MVO₂ to PVA and a decrease in that from PVA to EW. Mechanical efficiency (EW/MVO₂) remained unchanged over the range of contractile states of the patients studied. An increase in afterload produced similar effects on these energy conversion efficiencies. We used PVA as the total mechanical energy of ejecting contraction, as defined by Suga. According to the PVA concept, mechanical efficiency can be divided into two steps: the efficiency of energy conversion from MVO₂ to PVA as the first step and the efficiency of energy conversion from PVA to EW as the second step. Then PVA can be assumed to reflect an intermediate form of energy between MVO₂ and EW.

**Energy Conversion Efficiency From MVO₂ to PVA**

Suga and Suga et al have demonstrated a linear relation between MVO₂ and PVA with a nonzero positive intercept for PVA=0 (MVO₂=a·PVA+b, b>0). The intercept, b, the MVO₂ required for a mechanically unloaded contraction, represents the sum of the basal energy requirement and the energy required for excitation–contraction coupling. They also have shown that when inotropic state is enhanced, the MVO₂–PVA relation shifts upward (increase in b), and when inotropic state is depressed, the relation shifts downward (de-
crease in b), without significant change in the slope, a, of the relation. The reciprocal of the slope of the linear $MVO_2$–$PVA$ relation has been considered to represent the efficiency of chemomechanical transduction of the contractile machinery of the left ventricle. In excised dog hearts, this efficiency ranged from 30% to 50% regardless of the loading, heart rate, and inotropic conditions.\textsuperscript{25–27} The present study demonstrated that this efficiency averaged 41% in human left ventricle at any level of contractile state. Under these circumstances, $PVA/MVO_2$ efficiency increases with the depression of contractile state for a given $PVA$ and with an increase in $PVA$ accompanied by an increase in afterload (Figure 6, left panel).

**Left Ventricular Work Efficiency**

Work efficiency, $EW/PVA$, represents the efficiency of mechanical energy transfer from the ventricle to the arterial system.\textsuperscript{9} This efficiency decreased with the depression of the contractile state or with an increase in afterload. If we assume that the time-averaged ventricular pressure during ejection is close to end-systolic pressure and left ventricular diastolic pressure is negligible compared with the pressure during ejection, $EW$ can be approximated by the product of stroke volume and end-systolic pressure. Work efficiency can be formulated\textsuperscript{28} as $EW/PVA=1/[1+(Ea/Ees)/2]$, where $Ea$ represents the arterial input impedance properties (effective arterial elastance) expressed by the slope of arterial end-systolic pressure–stroke volume relation. This formula implies that work efficiency is a function of basal inotropic state ($Ees$) and afterload ($Ea$). In a previous study,\textsuperscript{15} we have demonstrated that in patients with variably depressed hearts, work efficiency was actually related to the $Ea/Ees$ ratio. This relation is shown schematically in Figure 6 (right panel), which illustrates two different inotropic conditions and two different effective arterial elastances. This conceptual framework shows that a depression of contractile state or an increase in afterload increases the $Ea/Ees$ ratio and end-systolic potential energy and reduces work efficiency.

**Left Ventricular Mechanical Efficiency**

According to earlier studies,\textsuperscript{1–4,29,30} cardiac mechanical efficiency varies between 0 and 45%, with normal functioning values of 10% to 25%. Our present data fell within this range. The mechanical efficiency is affected by ventricular preload, afterload, and contractile state. The influence of these factors on efficiency, however, has been difficult to analyze in quantitative terms. Evans
loading during cineventriculography. According to Baxley et al3 or Nichols et al4 most of the patients with ejection fraction less than 40% showed a significant reduction in mechanical efficiency. In patients with ejection fraction greater than 40%, however, mechanical efficiency did not always decrease to that extent. In the present study, mechanical efficiency did not correlate with left ventricular ejection fraction. The mechanism for relatively constant mechanical efficiency in moderately depressed hearts can be explained by the PVA concept (Figure 6). In these hearts, the reduction in non-work-related oxygen consumption, b, raises PVA/MVO2 efficiency, which counteracts the reduction in work efficiency so as to maintain mechanical efficiency. When the heart is severely depressed, however, the substantial fall in work efficiency would predominate over the increase in PVA/MVO2 efficiency, resulting in a mismatch in terms of mechanical efficiency. Similar mechanisms could also work in the response of mechanical efficiency to pressure loading.

The present study included six patients with previous myocardial infarction. In these patients, MVO2 might be augmented in maintaining increased regional systolic wall stress, which could not be expressed within the pressure–volume diagram. Therefore, if wall stress were used instead of pressure, different findings might occur in patients with regional wall motion abnormalities.

**Limitations**

Several methodological problems must be discussed. Because volume data are limited by left ventriculography, we derived the relation between PVA and MVO2 only for two pressure settings in each subject, one baseline pressure and another relatively high pressure. For this analysis, we took advantage of the fact that despite the great difference in heart size, a high correlation of the linear MVO2–PVA relation has been shown both in dog hearts and in rabbit hearts.31 Minor fluctuation of contractility caused by baroreflex-mediated alterations in sympathetic discharge cannot be excluded. In this regard, Suga et al32 reported that the changes in the slope of end-systolic pressure–volume line caused by either carotid sinus or aortic baroreceptor reflexes were only about 13% when arterial pressure was changed between 100 mm Hg and 150 mm Hg. In addition, Vatner et al33 found that in conscious animals, the baroreflex control of cardiac contractility was even weaker than in the anesthetized state. Therefore, the reflex change in the slope of end-systolic pressure–volume relation in our study could be negligible.

Pressure loading potentially causes a transient mitral regurgitation, which might modify the increase in MVO234 and the change in mechanical efficiency because mitral regurgitation increases left ventricular flow work but decreases potential energy. The present study, however, confirmed that there were no signs of mitral regurgitation either at rest or during pressure elevation.

The thermodilution technique for estimation of coronary blood flow has been used in the past several years. The accuracy of this method greatly depends on the catheter position within the coronary sinus, and only large changes (more than 30%) in coronary blood flow are likely to be qualitatively accurate.35 We paid meticulous attention throughout these studies to proximal positioning of the external thermistor. The change in

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**Figure 5.** Scatterplots show relation between the mechanical efficiency (EW/MVO2) and the basal inotropic state (Ees) (upper panel) or ejection fraction (EF) (lower panel). Mechanical efficiency remained unchanged (mean 22.1%) against different Ees or EF levels. EW, left ventricular external work; MVO2, myocardial oxygen consumption per beat; Ees, slope of end-systolic pressure–volume relation.

and Matsuoka1 first observed in Starling heart–lung preparations that mechanical efficiency decreased as the condition of the heart preparations deteriorated. They also found that cardiac efficiency was greater during volume loading than during pressure loading. Bing et al30 measured left ventricular efficiency in humans. Mean efficiency value for normal subjects was 22%, which compared remarkably well with the average value of 15% for patients with congestive heart failure. Baxley et al3 calculated mechanical efficiency in patients with variably depressed hearts resulting from valvular or myocardial disease. The range of efficiency varied from 4% to 40%, with the smallest value formed in patients with primary myocardial disease. They showed that mechanical efficiency was roughly correlated with ejection fraction. Recently, Nichols et al4 showed a similar relation between ejection phase indexes and left ventricular mechanical efficiency in patients with coronary artery disease.

The present study demonstrated that mechanical efficiency was comparable in patients with mildly to moderately depressed hearts. We consider that the differences between our results and those in earlier studies probably result from severity of cardiac depression, because we exclude patients with severe cardiac dysfunction, who may have a risk in pressure or volume

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coronary blood flow in this study was 42.2%. With each patient serving as his own control and with little change in heart rate, data thus collected should reliably reflect changes in coronary sinus flow.

Recent evidence indicates that the ionic contrast medium can significantly alter myocardial metabolism by increasing the free fatty acid uptake of the myocardium while decreasing the myocardial uptake of glucose and lactate. In the present study, we carried out left ventriculography using nonionic contrast agents, which exhibit considerably less myocardial toxicity. Sufficient time was also allowed between two left ventriculographies. Therefore, the effects of contrast medium on cardiac performance and metabolism would be minimized in this study.

Finally, because of the small numbers and heterogeneity of the patients, the data obtained were somewhat scattered and could contain modest quantitative error. However, hemodynamic changes seen in our study were dramatic and relatively uniform during pressure manipulation. Therefore, quantitative factors probably would little affect the present results.

In summary, we applied the PVA concept to evaluate energy conversion efficiency in human left ventricles. Our results showed that mechanical efficiency does not change appreciably with depression of contractile state or an increase in afterload as long as the left ventricular pump function is not severely compromised. The decreased sensitivity of mechanical efficiency can be accounted for by the counteraction of PVA/MVO₂ efficiency and work efficiency. Thus, the concept of PVA-mediated energy transfer would provide us a rational basis to gain insight into the energetic aspects of human left ventricle under normal and pathological conditions.

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