Equal Oxygen Consumption Rates of Isovolumic and Ejecting Contractions with Equal Systolic Pressure-Volume Areas in Canine Left Ventricle

HIROYUKI SUGA, TAKAKAZU HAYASHI, SHIGEFUMI SUEHIRO, RYUICHI HISANO, MACHIKO SHIRAHATA, AND ISHIO NINOMIYA

SUMMARY — Left ventricular systolic pressure-volume area (PVA) has been found to be highly linearly correlated with cardiac oxygen consumption rate per beat (VO₂) in a given canine heart with a stable inotropic background. PVA is a specific area in the pressure-volume (P-V) diagram that is bounded by the end-systolic and end-diastolic P-V relationship lines and the systolic segment of the P-V loop, consisting of the sum of external mechanical work and what is considered the end-systolic elastic potential energy in the ventricular wall. In this study, we compared VO₂'s of steady state entirely isovolumic and variously ejecting contractions that were made to have equal PVA's in the canine left ventricle. We found that VO₂'s of these isovolumic and ejecting contractions with equal PVA's (isovolumic vs. ejecting - 1008 ± 64 (SB) vs. 1022 ± 62 mm Hg ml/beat, n = 32 pairs in 10 hearts) were equal to each other (0.0376 ± 0.0021 vs. 0.0368 ± 0.0021 ml O₂/beat) regardless of the marked differences in stroke volume (0 vs. 9.8 ± 0.6 ml), end-diastolic volume (20.3 ± 0.8 vs. 23.7 ± 0.9 ml), end-systolic volume (20.3 ± 0.8 vs. 13.9 ± 0.7 ml), peak pressure (123 ± 5 vs. 88 ± 5 mm Hg), stroke work (0 vs. 636 ± 36 mm Hg ml/beat), and calculated peak total wall force (1688 ± 77 vs. 1077 ± 72 g). Therefore, we conclude that PVA can serve as a reliable predictor of VO₂ in a given canine left ventricle with a stable inotropic background whether the contraction mode is isovolumic or ejecting.


MANY cardiodynamic variables and indices have been proposed as primary determinants of cardiac oxygen consumption (Braunwald et al., 1976; Gibbs, 1978; Gibbs and Chapman, 1979). However, none of them alone or appropriately combined so far can consistently serve as a reliable predictor of cardiac oxygen consumption rate per beat (VO₂) under a variety of cardiac loading conditions. Even peak wall force of the ventricle, which has been widely accepted as the most important determinant of VO₂ in a constant inotropic state, cannot uniquely predict VO₂ of a given contraction because its VO₂ varies with ventricular ejection at a constant peak wall force (Coleman et al., 1969; Burns and Covell, 1972; Weber and Janicki, 1977).

In search of a more reliable predictor of VO₂, our recent experiments on canine excised cross-circulated hearts have shown that VO₂ is highly linearly correlated with the left ventricular systolic pressure volume area (PVA) (Suga, 1979; Khalafbeigui et al., 1979; Suga et al., 1981). PVA is a specific area in the pressure-volume (P-V) diagram that is bounded by the end-systolic and end-diastolic P-V relationship lines and the systolic segment of the P-V loop trajectory, as shown in Figure 1. PVA is therefore the sum of two areas; one for the external mechanical work and the other for what is considered the end-systolic elastic potential energy in the ventricular wall (Suga, 1979, 1980). PVA can be considered total mechanical energy required for one contraction of the ventricle (Suga, 1979). However, the feasibility of PVA as a reliable predictor of VO₂ still remains to be fully examined.

In the present investigation, we specifically studied whether or not VO₂'s of an entirely isovolumic and a variously ejecting contraction with equal PVA's were identical in a stable inotropic state. We used the left ventricle of the canine excised cross-circulated heart preparation and precisely servo-controlled left ventricular pressure and volume loads while accurately measuring VO₂. Since the mode of contraction was switched and the data were obtained before and after the switch, a change in the inotropic background during each experimental run was minimal. Results clearly indicated that the two steady state contractions with markedly differently loads but having equal PVA's were accompanied by virtually the same VO₂'s in spite of the significant differences in many cardiodynamic variables including peak total wall force. The peak

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Partly supported by Research Grants for Cardiovascular Diseases from the Ministry of Health and Welfare of Japan, Grant-in-Aid for Scientific Research from the Ministry of Education, Science and Culture of Japan, and a Japan Heart Foundation Research Grant for 1980.
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Received October 13, 1980; accepted for publication May 14, 1981.
wall force was about 33% smaller in the ejecting contraction than in the isovolumic contraction in spite of their equal PVA's and Vo's.

**Methods**

**Heart Preparation**

A pair of mongrel dogs (about 12 kg body weight) were anesthetized with sodium pentobarbital (30 mg/kg, iv) in each experiment. Blood was heparinized (1000 U/kg body weight) in both dogs. The common carotid artery and the external jugular vein were cannulated in one dog to be used as a support dog in preparation for the cross-circulation of the heart preparation.

In the heart donor dog, the chest was opened midsternally and the left subclavian artery and the right ventricle (via the atrial appendage) were cannulated to be connected with the cross-circulation tubings from the support dog. The heart-lung section was isolated by ligation at the descending aorta, brachiocephalic artery, superior and inferior caval veins and azygos vein. The lung lobes were removed after ligation at the pulmonary hili and the cross-circulation was started. The supported beating heart was excised from the chest.

The left ventricle was vented at the apex and the left atrium was opened widely. All the chordae tendineae were cut to free the mitral valves. A thin rubber balloon (unstressed volume = about 60 ml) which had been tied on a connector (18 mm i.d.) was placed in the left ventricle and the balloon end of the connector was secured at the mitral annulus by sutures. The electrical cable of a miniature pressure gauge (Konigsberg, P-7), placed inside the apical end of the balloon, was pulled out through the apical incision.

The intraventricular balloon was connected to the volume servo pump that was identical in design with the original one (Suga and Sagawa, 1977). The balloon and the pump were primed with water without leaving any air bubbles. The pump enabled us to precisely control and accurately measure left ventricular volume. Figure 2 illustrates the preparation connected to the pump system.

The arterial blood pressure of the support dog served as the coronary perfusion pressure. The mean level of the coronary perfusion pressure was relatively constant in each preparation, although it ranged between 70 and 125 mm Hg among dogs. In some preparations, the support dog's arterial blood pressure tended to decrease below 70 mm Hg and was maintained above 70 mm Hg by either canine blood transfusion or infusion of low molecular dextran solution. Furthermore in preparation nos. 8 and 10, phenylephrine (50-250 µg/min, iv) was administered to maintain the support dog's arterial pressure near 100 mm Hg.

The temperature of the heart was monitored and maintained as constant as possible between 35°C and 37°C with a heater on the coronary arterial perfusion tubing.

The heart beat at its regular sinus rhythm, which ranged between 90 and 150 beats/min among preparations. The left ventricular ECG was recorded and used to trigger the volume command signal of the servo pump. Occasionally, cardiac arrhythmias
were observed and intervened in steady state contractions. When the arrhythmias were too frequent, we did not run any experiment.

The support dog was artificially ventilated with room air mixed with O₂. Coronary pH, Po₂, and PCO₂ occasionally were measured. Po₂ and PCO₂ were relatively constant in each dog, but ranged between 70 and 150 mm Hg, and 20 and 35 mm Hg, respectively. Coronary pH tended to gradually decrease with time from 7.3-7.4 to 7.1-7.2 over a few hours under the cross-circulation. In some dogs, we corrected pH by administration of bicarbonate, but in the other dogs, we did not correct it. On the average among dogs, the pH was 7.36 during experimental runs.

We collapsed the right ventricle by draining all the coronary venous blood returning into it to minimize its oxygen consumption rate.

At the end of each experiment, we opened the left ventricle to examine the fitting of the balloon. Left and right ventricular weights were measured, with all the septum included as part of the left ventricle. In 10 hearts the weights were 65 ± 12 (sd) and 25 ± 5 g, respectively.

**Oxygen Consumption**

Total coronary perfusion flow was measured with an electromagnetic flowmeter in the coronary venous return tubing draining the right ventricle. Although the left ventricular Thesbian flow was excluded from the flow measurement, it is known to be a few percent of total coronary flow (Moir et al., 1963) and was in fact as little as 1-2 ml/min as opposed to total coronary flow of 40-150 ml/min in our preliminary experiments. We therefore considered it negligible in the measurement of the total coronary flow.

The oxyhemoglobin percent saturations of the coronary arterial and venous bloods were measured with two carefully calibrated oximeters (Waters Instrument, model 0-600A). From their readings, the arteriovenous oxygen saturation difference was obtained. To convert the saturation difference into oxygen content difference, coronary arterial blood of a known saturation was sampled immediately before the first experimental run in each dog, and its oxygen content was determined with a Lex O₂ Con oxygen content analyzer (Lexington Instruments). To minimize the conversion error from oxygen saturation to content, we maintained arterial Po₂ around 100 mm Hg so that the physically dissolved oxygen (0.3 vol %/100 mm Hg Po₂) could be negligibly small as compared to total oxygen content of coronary arterial blood. We did not add dextran solution during the present experimental runs so that arterial hemoglobin concentration changed little. Since we compared the oxygen consumption rates of contractions only a few minutes apart in the present study, we reasonably assumed that the conversion factor from oxygen saturation to content remained the same for the neighboring contractions. Although multiple experimental runs required about half an hour, we applied the same conversion factor, assuming that its change, if any, was relatively small. This assumption may have caused some error in the assessment of oxygen consumption rate, but the neighboring contractions were probably subjected to the same error. This kind of the stationary error did not affect our comparison of the oxygen consumption rates of neighboring contractions.

The product of the total coronary flow in ml/min and the coronary arteriovenous oxygen content difference in vol % gives the oxygen consumption rate per min. This quantity was divided by heart rate in beats/min to yield the oxygen consumption rate per beat, VO₂, in ml O₂/beat.

As described by Weber and Janicki (1977), both coronary flow and venous oxygen saturation tracings gradually changed to their new levels after each change in ventricular loading conditions. Therefore, we obtained their readings after their continuously recorded tracings had stabilized reasonably (Fig. 3). Although the mean levels of the coronary flow and oxygen saturation curves were stable, the curves fluctuated to some extent. We consider that the fluctuations probably were pro-
duced by the slightly pulsatile flow of the coronary venous blood drained from the right ventricle. We simply neglected these fluctuations and read their mean levels.

### Pressure-Volume Area

PVA, an abbreviation of the left ventricular systolic pressure-volume area, is the specific area in the pressure-volume (P-V) diagram that is bounded by the end-systolic and end-diastolic P-V relationship lines and the systolic segment of the P-V loop trajectory, as seen in Figure 1. This area, which may still be somewhat strange to many readers, was first proposed to be meaningful through a theoretical consideration of the time-varying elastance model of the ventricle based on its instantaneous P-V relationships (Suga and Sagawa, 1974; Suga, 1979). Later, PVA was found to be highly linearly correlated with VO₂ in a set of multiple isovolumic and ejecting contractions of a given left ventricle with a stable inotropic background (Khalafbeigui et al., 1979; Suga et al., 1981). PVA was assessed in this study in the same manner as before.

First, a preliminary set of isovolumic contractions at different volumes was produced in each left ventricle and the end-systolic P-V relationship line was determined by drawing a straight line through the end-systolic (i.e., peak isovolumic) P-V data points of these contractions. The applicability of a straight line to the end-systolic P-V relationship within the physiological ranges of ventricular pressure and volume has often been supported (Monroe and French, 1961; Suga et al., 1973; Suga and Sagawa, 1974; Weber et al., 1976; Sagawa, 1978), although there are limitations of its applicability to abnormally loaded contractions (Suga et al., 1979). The volume axis intercept, Vd, of the end-systolic P-V line was determined by extrapolation. Next, for each contraction whose PVA and VO₂ were to be studied, a straight line was drawn from the predetermined Vd to the end-systolic P-V data point, which was either the peak pressure point in the isovolumic contraction or the left upper corner for the ejecting contraction. This line was considered the end-systolic P-V line of the contraction, marking the upper boundary of PVA, as seen in Figure 1. The end-systolic P-V relationship curve was then determined by drawing a curve through multiple end-diastolic P-V points, and marked the lower boundary of PVA. However, in the low-volume region where the end-diastolic P-V curve ran under the volume axis, the volume axis instead of the end-systolic P-V curve bound PVA. The systolic segment of the P-V loop trajectory also bounded PVA. PVA was measured in mm Hg ml by planimetry on each P-V diagram pictured from a storage oscilloscope. In a preliminary study, the sample standard deviation of PVA values of a given PVA (750 mm Hg ml) was as small as 9 mm Hg ml (n = 10).

### Experimental Protocol

To compare VO₂'s of steady state entirely isovolumic and variously ejecting contractions with equal PVA values in the same inotropic state, we switched the mode of contraction from isovolumic to ejecting or in the opposite direction while equalizing their PVA's as much as possible. Since the shapes of two PVA's were different, it was difficult to make two PVA's identical. However, they were found to be very close to each other in the following analysis.

Both coronary flow and arterial venous oxygen saturations were recorded continuously, and their steady state readings were obtained when the transient changes following the switch of contraction mode had disappeared. It usually took a few minutes until we obtained data after each switch of contraction mode. Similar switches of contraction mode were carried out at one to seven different volumes in each heart. We considered that this protocol was appropriate for the present study in which any change in inotropic background for a pair of steady state isovolumic and ejecting contractions had to be minimized. The previous protocols consisting of comparison of VO₂'s and PVA's of many isovolumic and ejecting contractions in various sequences and with various volume steps were not appropriate for the present purpose because the inotropic background might have changed unexpectedly between a temporally distant pair of isovolumic and ejecting contractions even if their PVA's were equal (Khalafbeigui et al., 1979; Suga et al., 1981).

### Measured Parameters

In addition to coronary flow and its arterial and venous oxygen saturations for VO₂ and the P-V loop for PVA, we recorded left ventricular pressure and volume as a function of time. From these tracings, we obtained stroke volume (ml), end-diastolic and end-systolic volumes (ml), and peak ventricular pressure (mm Hg). Stroke work (mm Hg ml/beat) was obtained as the area within the P-V loop by planimetry.

In addition, we calculated peak total circumferential wall force (g) of the ventricle using a sphere model. The total wall force F(g) is equal to the product of the internal cross-sectional area A (cm²) and intraventricular pressure P (mm Hg, 1 mm Hg = 1.36 g/cm²). Since A = πr² (internal radius)² and ventricular internal volume V(ml) = (4πr³/3) (internal radius)³, A = 1.21 V²/³. Therefore, F = 1.64 P V²/³.

To determine peak wall force, we utilized the same graphical method as proposed previously (Suga and Sagawa, 1979). Figure 4 shows a family of isometric curves (or force isopleths) superimposed on a representative pair of isovolumic and ejecting P-V loop trajectories having equal PVA values. These isometric curves were drawn by relation P and V that satisfied F = 1.64 P V²/³ = a specified constant.
force incrementing from 500 to 4000 g in steps for multiple curves. By using this figure, we could easily determine the instant at which ventricular wall force became maximal and roughly estimate the peak wall force. We read ventricular pressure and volume values at the time of peak wall force and put them into $F = 1.64 \ P \ V^{2/3}$ to obtain the peak wall force for analysis.

**Results**

Figure 5 shows the pressure and volume tracings and the P-V loop trajectories of a representative pair of isovolumic and ejecting contractions with comparable PVA's in the left ventricle. They were steady state contractions in a stable isotropic state. The shapes of their PVA's are illustrated in the figure. Although their shapes are different, the two PVA's are virtually identical, indicating a successful equalization of PVA. Vo$_2$'s of these contractions were found to be virtually the same and are listed in the figure. Stroke volume and stroke work were zero in the isovolumic contraction, whereas they were considerable in the ejecting contraction. Endsystolic and end-diastolic volumes were different between the two contractions. Peak ventricular pressure was markedly higher in the isovolumic contraction. Calculated peak wall force also was markedly higher in the isovolumic contraction. Similar results were observed in all 32 pairs of isovolumic and ejecting contractions in a total of 10 hearts.

Table 1 lists PVA, Vo$_2$, and the other cardiodynamic parameters for each pair of isovolumic and ejecting contractions in 10 hearts and their means, sd's and se's. Both PVA's and Vo$_2$'s were very close to each other for all pairs of contractions. Their differences were statistically insignificant (paired $t$-test, $P > 0.2$ for PVA, $P > 0.05$ for Vo$_2$). Stroke volume and work existed only of ejecting contractions. End-diastolic and end-systolic volumes, peak ventricular pressures, and peak wall forces were significantly different (paired $t$-test, $P < 0.001$) from each other between the paired contractions.

Figure 6 plots the differences of Vo$_2$ against those of PVA (left) and peak wall force (right) between all the pairs of isovolumic and ejecting contractions. Evidently, equal amounts of oxygen were consumed in isovolumic and ejecting contractions having equal PVA's but different peak wall forces.

**Discussion**

The present results clearly indicate that isovolumic and ejecting contractions with equal PVA's...
are accompanied by equal VO2's in spite of marked differences in peak ventricular pressure and peak wall force as well as stroke volume, end-diastolic and end-systolic volumes, and stroke work in a given canine left ventricle with a stable inotropic background. Ventricular pressure and volume have been considered primary determinants of VO2 (Evans and Matsuoka, 1915; Starling and Visscher, 1926; Sarnoff et al., 1958; Monroe and French, 1961). Ventricular wall force was later shown to be better correlated with VO2 (McDonald, 1966; Graham et al., 1968; Burns and Covell, 1972; Gibbs and Chapman, 1979). However, there is obvious evidence against peak wall force as a reliable and consistent predictor of VO2 under a variety of ventricular loading conditions; that is, even if isovolumic and ejecting contractions have the same peak wall forces, the ejecting contraction is accompanied by a greater VO2 than the isovolumic contraction in a stable inotropic state. The difference in VO2 was proportional to the external mechanical work (Pool et al., 1968; Burns and Covel, 1972; Gibbs, 1978). This feature of cardiac energetics is considered a cardiac version of the Fenn effect (Fenn, 1923), although the underlying mechanism remains unknown (Gibbs, 1978). Our results support the view that peak wall force is not consistently a reliable predictor of VO2 even in a stable inotropic state and none of stroke volume, end-diastolic and end-systolic volumes, stroke work, and peak pressure can consistently serve as reliable predictors of VO2, as generally recognized at present (Gibbs, 1978). In contrast, PVA is shown to be a promising candidate as a reliable predictor of VO2. The present results thus corroborate our previous investigation (Suga, 1979; Khalafbeigui et al., 1979; Suga et al., 1980).

Since we obtained neither the tension time index (Sarnoff et al., 1958) nor time integral of wall force (Weber and Janicki, 1977) in the present study, we could not conclude quantitatively how they were different between a pair of isovolumic and ejecting contractions with equal PVA's. However, a glance at the ventricular pressure tracings of the two contractions, for example in Figure 5 gives us an impression that tension time index, i.e., time integral of ventricular pressure, or area under the pressure curve, is much smaller in the ejecting contraction. As for the comparison of PVA and the time integral of wall force, a separate study is needed in which instantaneous wall force is calculated and integrated over systole.

Let us now theoretically consider the relationship between PVA and peak wall force. Figure 7A illustrates a P-V loop trajectory of an ejecting contraction together with the end-systolic P-V line and an isotonic curve for the peak wall force. When the ventricular pressure during ejection is relatively constant, as usually is observed in normal contractions in vivo, peak wall force occurs at the end of isovolumic contraction phase or, in other words, the onset of the ejection phase, as shown in Figure 7. Define Pe = constant ventricular pressure during ejection, SV = stroke volume, Ved = end-diastolic volume, Ves = end-systolic volume, F = peak wall force, Emax = slope of the end-systolic P-V relationship line, and r = end-diastolic internal radius of the sphere model of the ventricle. The following equations can be derived:

\[ Pe = Emax(Ved - Vd) \]

\[ F = \pi^2 Pe \]

\[ Ved = \left(\frac{4\pi}{3}\right)^\frac{3}{2} \]

\[ SV = Ved - Ves \]

\[ PVA = Pe(Ved - Ves) + Pe(Ves - Vd)/2 \]

From these equations, the following two equations can be deduced.

\[ F = (Ved - Sv - Vd)[\pi(3/4\pi)^{2/3}Ved^{2/3}Emax] \]

\[ PVA = 0.5(Vod + SV - Vd)F/\pi(3/4\pi)^{2/3}Ved^{2/3} ] \]

These two equations allow us to relate PVA and F for a given SV. Since the elimination of Ved from these equations is not simple, we used numerical calculations to relate them. Figure 7B shows the solution. Evidently, PVA and peak wall force are curvilinearly related for a given stroke volume, but the PVA-peak force relation depends on stroke volume. For any given PVA, peak wall force is always greater in the isovolumic contraction than in any ejecting contraction. In other words, for a given peak wall force, PVA is always smaller in the isovolumic contraction than in any ejecting contraction. Since PVA has been shown to be highly linearly correlated with VO2 (Khalafbeigui et al., 1979; Suga et al., 1981), it is expected that a similar family of curves will be obtained when PVA on the ordinate is replaced by VO2 in Figure 7B. Weber and Janicki’s (1977) finding (Fig. 5 of their paper) as well as Burns and Covell’s (1972) finding (Fig. 3 of their paper) exactly support this prediction. These theoretical considerations support our view that ventricular peak wall force cannot always serve as a reliable predictor of VO2.

Our previous studies have indicated the linear relation: VO2 (ml O2/beat) = A-PVA (mm Hg ml/bt) + B. Coefficient A is the slope of the VO2-PVA linear regression line and indicates the oxygen cost of PVA. On the average, A = 1.53 \times 10^{-5} \pm 0.14 \times 10^{-5} (SE) (ml O2/mm Hg ml) (Khalafbeigui et al., 1979) or 1.64 \times 10^{-5} \pm 0.12 \times 10^{-5} (Suga et al., 1981) in the same type of canine heart preparations as used in the present study. Constant B is the VO2 for pressure and volume unloaded beating hearts and is 0.019 \pm 0.003 (SE) (ml O2/beat) or 0.015 \pm 0.002 in those previous studies. The present results together with the previous ones conclusively indicate that the VO2-PVA relationship is virtually the same for both isovolumic and ejecting modes of contraction in a stable inotropic state. We therefore...
### Table 1: Experimental Data for PVA, Vo₂, and Other Cardiodynamic Variables

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<th>Vo₂ (ml O₂/beat)</th>
<th>SV (ml)</th>
<th>EDV (ml)</th>
<th>ESV (ml)</th>
<th>Peak P (mm Hg)</th>
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**E**  
Mean: 1022  
SD: 0.0368  
SE: 0.0216
Table 1—continued

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<th>$V_o_2$ (ml O2/beat)</th>
<th>SV (ml)</th>
<th>EDV (ml)</th>
<th>ESV (ml)</th>
<th>Peak P (mm Hg)</th>
<th>SW (mm Hg ml)</th>
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Paired t-test: NS = statistically insignificant ($P > 0.05$), 0.1% = level of significance, i.e., $P < 0.001$.

Conclude that PVA is a very reliable predictor of $V_o_2$ in a given canine left ventricle with a stable ionotropic background.

PVA is an energy quantity (Suga, 1979). Therefore, PVA may be advantageous to a better understanding of the coupling between cardiac mechanics and energetics. For example, an empirical relation: $V_o_2$ (ml O2/beat) = $1.6 \times 10^{-6}$ PVA (mm Hg ml/beat) + 0.015 (ml O2/beat), can be converted into $V_o_2$ (joule/beat) = 2.4 PVA (joule/beat) + 0.3 (joule/beat), using the physical relation of 1 mm Hg = $1.333 \times 10^{-4}$ joule and the physiological relation of 1 ml O2 = 20 joules in normal cardiac metabolism. The new coefficient of PVA, 2.4, is dimensionless. Rearranging the above equation yields $V_o_2$ (joule/beat) = 0.3 (joule/beat) = 2.4 PVA (joule/beat), or PVA/$V_o_2$ = 0.3 = 0.42. The last equation indicates that the efficiency of energy conversion from oxidative metabolism (in excess of $V_o_2$ of unloaded beating heart) to PVA is 42%. Since external mechanical work is part (about 60%) in a normal ejecting contraction of PVA, the conventional mechanical efficiency of energy conversion from $V_o_2$ to external mechanical work is expected to be normally about 20% at best. This value is consistent with the values reported in a recent review by Gibbs (1978). What would then be the fate of the energy not used for PVA and external work? According to Gibbs (1978), it is probably converted finally into heat.

External mechanical work, or stroke work, of the heart is an energy quantity by itself. However, Evans and Matsuoka (1915) and Sarnoff et al. (1958) showed that the external work of the heart did not uniquely correlate with the cardiac oxygen consumption rate, but instead the oxygen consumption rate for a given external work was markedly greater in contractions whose external work was performed primarily by pressure load rather than volume load. The inferiority of external work as a predictor of cardiac oxygen consumption is now well recognized (Gibbs, 1978); our study supports this view.

Another energy quantity proposed as a determinant of cardiac oxygen consumption is the contractile element work CEW (Britman and Levine, 1964;
contrast to the PVA's elastic potential energy stored in the series elasticity, in PVA. CEW's internal work is also a kind of elastic and the end-systolic elastic potential energy in contrast to CEW and PVA is the internal work in CEW and the end-systolic elastic potential energy in PVA. CEW's internal work is also a kind of elastic potential energy stored in the time-varying elasticity. The series elasticity is a passive element, whereas the time-varying elasticity is an active element that is considered to consist of both the active contractile element and all the passive parallel and series elastic elements. Moreover, the oxygen cost of the CEW's internal work was found to be about three times as much as that of the external work for yet unknown reasons (Pool et al., 1968; Coleman et al., 1969; Burns and Covell, 1972; Gibbs, 1978), whereas the oxygen costs of PVA's elastic potential energy and external work are virtually the same (Suga et al., 1980). These comparisons of PVA and CEW may lead us to a better understanding of cardiac mechanics and energetics.

We have emphasized the importance of the stable inotropic background of the heart in each experimental run in the present study for the following reason. In a separate study (manuscript in preparation), administration of catecholamines and calcium to the heart markedly increased VO\textsubscript{2} for any given PVA. Therefore, if the inotropic background had not been stable for each pair of isovolumic and ejecting contractions having equal PVA's, these contractions would have had different VO\textsubscript{2}'s. The importance of the inotropic background as a modifier of the coupling between cardiac mechanics and energetics has been generally accepted (Braunwald et al., 1976; Gibbs and Chapman, 1979).

In summary, we conclude that PVA is a very reliable predictor of cardiac oxygen consumption rate, regardless of the contraction mode in a given canine left ventricle with a stable inotropic background.

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Equal oxygen consumption rates of isovolumic and ejecting contractions with equal systolic pressure-volume areas in canine left ventricle.

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doi: 10.1161/01.RES.49.5.1082

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