Ventricular efficiency predicted by an analytical model

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BURKHOFF, DANIEL, AND KIICHI SAGAWA. *Ventricular efficiency predicted by an analytical model.* Am. J. Physiol. 250 (Regulatory Integrative Comp. Physiol. 19): R1021–R1027, 1986.—Based on the recent data from the literature, we derived analytic expressions for ventricular stroke work (SW), ventricular O_2 consumption, and the ratio of the former to the latter, defined as ventricular efficiency, in terms of ventricular contractile properties and arterial afterloading properties. The ventricular properties were quantified by E_{es} and V_0, the slope and volume axis intercept, respectively, of the linear end-systolic pressure-volume relationship. Ventricular afterload was represented by the effective arterial elastance (E_a), a parameter that is dependent on aortic input impedance parameters. O_2 consumption was assessed by estimating ventricular pressure-volume area, which has been shown to be linearly related to O_2 consumption. Model analysis indicated that *1*) SW is maximum when $E_a = E_{es}$, *2*) the afterload that results in the greatest efficiency is always less than that which provides the maximum SW, *3*) the SW and efficiency of a weak heart are more sensitive to changes in afterload than in a strong heart, and *4*) there is a sigmoidal relation between ventricular efficiency and end-diastolic volume that reaches its maximum at volumes outside the upper limit of the physiological range. Further analysis of the model indicated that under physiological conditions ventricular and arterial properties may be adjusted more toward optimization of efficiency than SW.

end-systolic pressure-volume relationship; pressure-volume area; effective arterial elastance; myocardial oxygen consumption; aortic input impedance; isolated canine heart; cardiovascular model

THERE HAS BEEN LONG-STANDING INTEREST in understanding the interaction between the heart and vasculature, particularly with regards to the determinants of cardiac work or flow output (4, 5, 15, 18, 20, 21, 33–36, 38) and to the determinants of myocardial O_2 consumption (1, 3, 6–8, 14, 25–28). In most of the previous undertakings to model the energetics of the circulatory system, complex models requiring computer numerical solution have been devised [e.g., see Piene and Sund (18)]. Such models offer the advantage of essentially limitless complexity in their model design. The disadvantage is their inability to convey intuitive understanding of the interaction between the components of the system. Of course, when detailed and precise predictions of system behavior are required, such models are invaluable. Frequently, however, simpler models can provide equally useful information more quickly and easily. In particular, when these models can be solved analytically, the solution provides intuitive understanding of the behavior of the system over a wide range of operating conditions.

In the present study we derived a simple analytic model that relates the properties of the vascular system and the left ventricle to the mechanical work done by the heart and the amount of chemical energy consumed by the heart to perform that work. The basic experimental data on which the model was formulated were obtained in this laboratory and in the laboratory of Suga (27–29, 31, 32). The implications of the model predictions are discussed within the framework of the design features of the cardiovascular system.

THEORETICAL BACKGROUND

The efficiency of the ventricle as a pump is standardly defined as the ratio between mechanical work and energy consumption; however, because several definitions of both of these parameters exists, our definitions need to be clarified first. We will deal with only normal aerobic conditions, so that the source of the heart's energy can be considered to be proportional to O_2 consumed by the myocardium from coronary blood flow. The total mechanical work performed by the ventricle with each cardiac cycle is the sum of the external stroke work and the unexpressed mechanical potential energy stored in the ventricle at the end of ejection (27). However, the only component of ventricular work seen by the arterial vascular system is that work performed to propel blood from the ventricle to the aorta, i.e., the ventricular stroke work (SW). Therefore, from the point of view of the cardiovascular system as a whole, an appropriate definition of ventricular pump efficiency is the ratio between ventricular SW and O_2 consumption.

Both the amount of external SW performed by the ventricle and the amount of O_2 the ventricle consumes while performing that work vary with the loading conditions imposed on the ventricle and the contractile state of the heart. Below, we derive analytic expressions relating ventricular preload volume, afterload impedance, and ventricular contractile state to the resulting ventricular stroke work, O_2 consumption, and finally the ratio between these two, ventricular efficiency. This was accomplished by extending a previously proposed and validated analytic approach to ventricular-aortic coupling [Suna-

gawa et al. (33)] and incorporating the results of recent studies of Suga et al. (28, 29, 32) relating ventricular loading conditions and O_2 consumption.

Ventricular stroke work. In a previous study, Sunagawa et al. (33) analyzed the coupling of the left ventricle to systemic arterial system and derived an analytic expression that related stroke volume (SV) to ventricular contractility, preload volume, and arterial input impedance. Ventricular contractile properties were quantified by E_{es} and V_0 parameters, the slope and volume axis intercept, respectively, of the ventricular end-systolic pressure-volume relation (ESPVR) (22–24, 30). Suga and Sagawa (30) have shown that

$$P_{es} = E_{es}[V_{es} - V_0] \quad (1)$$

where P_{es} and V_{es} are the ventricular end-systolic pressure and volume, respectively. This relation is depicted in the bottom panel of Fig. 1.

The arterial system was represented by the three-element modified Windkessel model depicted in the top panel of Fig. 1. The impedance spectrum of this circuit has been shown to closely match the experimentally measured aortic input impedance of the arterial system except for the fluctuation of the modulus over the high-frequency range (37). It was shown that when a real ventricle and computer model of the arterial system with given properties were physically coupled the resulting

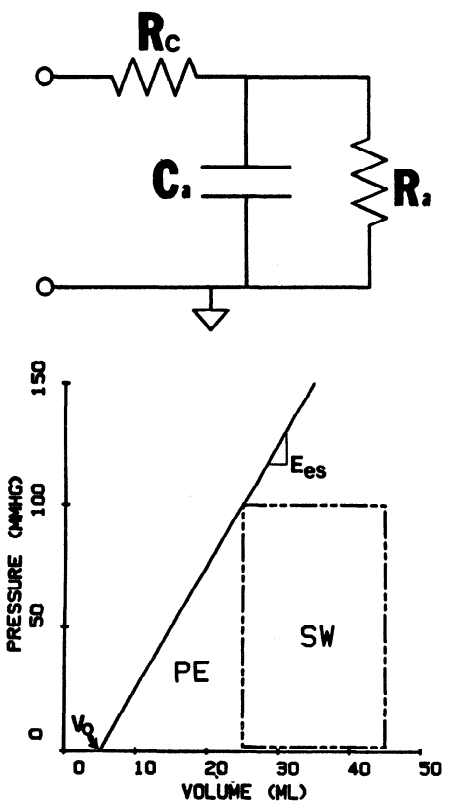

FIG. 1. *Top*: analogue equivalent of Windkessel model of aortic input impedance characterized by characteristic impedance (R_c), arterial compliance (C_a), and peripheral arterial resistance (R_a). *Bottom*: ventricular pressure-volume diagram. Ventricular end-systolic pressure-volume relation is linear and characterized by slope (E_{es}) and volume axis intercept (V_0). Pressure-volume area (PVA) is sum of external stroke work (SW) and end-systolic potential energy (PE).

SV could be predicted accurately by a single equation

$$SV = \frac{V_{ed} - V_0}{1 + E_a/E_{es}} \quad (2)$$

where V_{ed} is the end-diastolic ventricular volume. E_a (called the effective arterial elastance) represents the slope of the arterial end-systolic pressure-stroke volume relationship. It is a lumped characterization of the arterial impedance (with simplifying assumptions) and incorporates the influences of heart rate in terms of durations of ejections (t_s) and arterial diastole (t_d)

$$E_a = \frac{R_c + R_a}{t_s + T[1 - \exp(-t_d/T)]} \quad (3)$$

where R_c is the characteristic impedance, R_a is the peripheral resistance, and T is the product of R_a and arterial compliance C_a (i.e., $T = R_aC_a$). Under most physiological conditions this expression can be further approximated by

$$E_a = R_T/t_T \quad (3a)$$

where R_T is the total systemic resistance (i.e., $R_T = R_c + R_a$), and t_T is the total period of a heartbeat (i.e., $t_T = t_s + t_d$). This can be seen by expanding the exponential in the denominator by its Taylor series and disregarding the second and higher order terms, which is a reasonable assumption at physiological heart rates. Thus, to a first approximation, for a constant heart rate, E_a can be thought of as being essentially proportional to R_T. The validity of this model was demonstrated in an isolated supported canine left ventricular preparation that ejected against a computer-simulated arterial system (33).

If we assume that the time-averaged ventricular ejection pressure is close to P_{es}, ventricular SW can be approximated by the product of SV and P_{es}

$$SW = SV \times P_{es} \quad (4)$$

This simplified definition of SW includes the work done to the ventricle by the venous system and atrium during ventricular filling and thus makes the assumption that ventricular end-diastolic pressure is negligible compared with pressure during ejection; this assumption is reasonable in the normal heart but may be invalid in diseased states where end-diastolic compliance is frequently decreased. The definition also disregards the small inertia-related work performed by the heart.

Combining *Eqs. 1–4* and noting that $V_{es} = V_{ed} - SV$, the following analytic expression for SW can be derived

$$SW = E_{es}(V_{ed} - V_0)^2 \frac{E_a/E_{es}}{(1 + E_a/E_{es})^2} \quad (5)$$

Myocardial O_2 consumption. In many recent studies, Suga and co-workers (28, 29, 32) have demonstrated a linear relation between left ventricular O_2 consumption and pressure-volume area (PVA). The PVA is defined as the area on the ventricular pressure-volume (P-V) diagram circumscribed by the end-systolic and end-diastolic P-V relations and the systolic portion of the P-V trajectory (Fig. 1, bottom).

PVA can therefore be expressed as the sum of SW and

the end-systolic potential energy (PE), which is represented by the area of the triangle defined by the ESPVR, the end-diastolic P-V relation, and the isovolumic relaxation portion of the ventricular P-V trajectory

$$PE = P_{es}(V_{es} - V_0)/2 \quad (6)$$

again, assuming that end-diastolic ventricular pressure is small relative to end-systolic pressure. Substituting *Eqs. 1* and *2* into *Eq. 6* the following expression for PE is obtained

$$PE = E_{es}(V_{ed} - V_0)^2 \frac{(E_a/E_{es})^2/2}{(1 + E_a/E_{es})^2} \quad (7)$$

PVA, which is equal to the sum of SW and PE, is therefore approximated by

$$PVA = E_{es}(V_{ed} - V_0)^2 \cdot \frac{E_a/E_{es}}{(1 + E_a/E_{es})^2}[1 + (E_a/E_{es})/2] \quad (8)$$

The linear relation that Suga et al. (28, 29, 32) demonstrated between PVA and myocardial O_2 consumption (MVO_2) per beat is characterized by a slope (A) and an intercept (B) so that

$$MVO_2 = A[PVA] + B \quad (9)$$

The complete expression for MVO_2 is obtained by substituting *Eq. 8* into *Eq. 9*.

Ventricular efficiency. In the present study we defined ventricular efficiency (Eff) as the ratio between external SW and MVO_2. Accordingly, the following analytic equation for the mechanical efficiency is obtained by combining *Eqs. 5, 8,* and *9*

$$Eff = \frac{1}{A(1 + E_a/2E_{es}) + \frac{B(1 + E_a/E_{es})^2}{E_a(V_{ed} - V_0)^2}} \quad (10)$$

In calculating the efficiency, the values of the parameters in *Eq. 10* must be scaled appropriately so that both SW and MVO_2 are expressed in the same units (see below).

Parameter values. The values of E_{es}, E_a, V_0, V_{ed}, A, and B parameters were varied over a range considered to be physiological for a 20-kg dog with a left ventricle weighing 100 g. All parameter values were varied independently except for E_{es} and B (the y-axis intercept of the PVA-MVO_2 relation), which have been shown to be interdependent (29). Studies in our laboratory (2) indicate on average

$$B(\text{ml } O_2/\text{beat}) = 0.0032 \left(\frac{\text{ml } O_2}{\text{beat}} \cdot \frac{\text{ml}}{\text{mmHg}}\right) E_{es}(\text{mmHg/ml}) + 0.0104 \text{ (ml } O_2/\text{beat}) \quad (11)$$

The approximate physiological ranges of the other parameter values are E_{es} = 4–9 mmHg/ml; E_a = 4–7 mmHg/ml; V_0 = 5–10 ml; V_{ed} = 25–40 ml; and A = 1.75–2.10 × 10^{-5} ml $O_2 \cdot mmHg^{-1} \cdot ml^{-1}$. In determining ventricular efficiency, MVO_2 and PVA were converted to the same energy units, Joules, according to the following conventional equivalences (29): 1 mmHg·ml = 1.33 × 10^{-4} J and 1 ml O_2 = 20 J.

RESULTS

Equations 5, 9, and *10* provide quantitative descriptions of the dependence of SW, MVO_2, and efficiency on the six system parameters (E_{es}, V_0, V_{ed}, R_c, C_a, and R_c) that describe the properties of the left ventricle and the load imposed on it.

As an example of the predictions of these equations, consider the graphs in Fig. 2 in which SW (Fig. 2*A*), MVO_2 (Fig. 2*B*), and efficiency (Fig. 2*C*) are plotted as a function of E_a, the quantifier of afterload impedance. In graphing these relations, it was assumed that E_{es} = 7 mmHg/ml, V_{ed} = 45 ml, V_0 = 5 ml, A = 1.9 × 10^{-5} ml

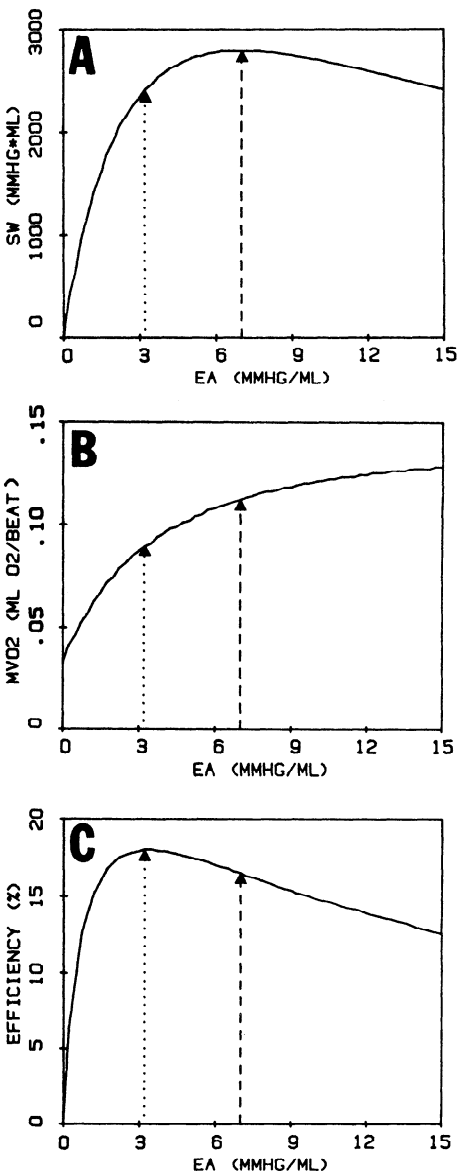

FIG. 2. Relation between afterload impedance as quantified by effective arterial elastance (E_a) and stroke work (A; SW), myocardial O_2 consumption (B; MVO_2), and ventricular efficiency (C). *Dashed lines,* E_a value that produces greatest SW; *dotted lines,* E_a value that produces greatest efficiency. See text for parameter values.

$O_2 \cdot mmHg^{-1} \cdot ml^{-1}$ and $B = 0.0328$ ml O_2/beat, which are within the physiological range of values (see above). E_a was varied between 0 and 15 mmHg/ml.

With increases in E_a, SW initially increases, reaches a plateau, and then decreases (Fig. 2*A*). Sunagawa et al. (34) showed that the maximum SW occurs when arterial and ventricular properties are matched; i.e., when $E_a = E_{es}$. This matched condition is indicated in Fig. 2 by the dashed line that is positioned in all the panels at $E_a = 7$ mmHg/ml.

MVO_2 (Fig. 2*B*) steadily increases with increases in E_a to a plateau. MVO_2 at zero afterload (i.e., $E_a = 0$) corresponds to the value of B. The plateau level of this relation corresponds to O_2 consumption during isovolumic contractions at the specified preload volume.

Ventricular efficiency (Fig. 2*C*), as defined in *Eq. 10*, initially rises with increases in E_a, reaches a maximum of ~18.5%, and then decreases. Whereas maximal SW occurs when $E_a = E_{es}$, optimum efficiency occurs when E_a is less than E_{es} (at $E_a = 3.4$ in this case). This optimal efficiency E_a value is indicated by the dotted lines in Fig. 2*C*. It is also evident that ventricular efficiency exhibits a greater dependence on E_a than does SW. At the E_a value that provides the greatest efficiency, SW is ~90% that of the peak SW; in contrast, at the E_a that provides the greatest SW, ventricular efficiency is only 70% that of its maximal value.

In Fig. 3, the same relations shown in Fig. 2 and discussed above are shown for several different levels of ventricular contractile state (the values of E_{es} are specified in Fig. 3*A*). Again, all other parameters are kept constant except for B, which is changed with E_{es} as indicated in *Eq. 11*.

Note that with increasing E_{es} the E_a that results in the greatest SW increases, always occurring when $E_a = E_{es}$ (arrows). The E_a that results in the greatest efficiency also increases with increasing E_{es} but is always less than the E_a that results in the greatest SW. The maximal attainable efficiency increases with E_{es}. Finally, it is evident that both the SW and the efficiency of a weak heart (i.e., a low E_{es} value) are more greatly influenced by afterload than that of a strong heart (i.e., a higher E_{es} value).

The dependence of SW, MVO_2, and efficiency on preload volume are shown in Fig. 4. The values of the model parameters used in calculating these relations were $E_{es} = 7$ mmHg/ml, $V_0 = 5$ ml, $E_a = 4$ mmHg/ml, $A = 1.9 \times 10^{-5}$ ml $O_2 \cdot mmHg^{-1} \cdot ml^{-1}$, $B = 0.0328$ ml O_2/beat. As indicated in *Eq. 5* and depicted in Fig. 4*A*, there is a parabolic relation between SW and EDV. Similarly, the relation between MVO_2 and EDV is also parabolic (*Eqs. 8* and *9*, Fig. 4*B*). There is a sigmoidal relation between ventricular efficiency and EDV (*Eq. 10*, Fig. 4*C*). The plateau of the sigmoid, however, is approached at volumes far greater than the upper limit of the physiological range of EDVs. Consequently, efficiency increases roughly linearly over the physiological range of EDVs.

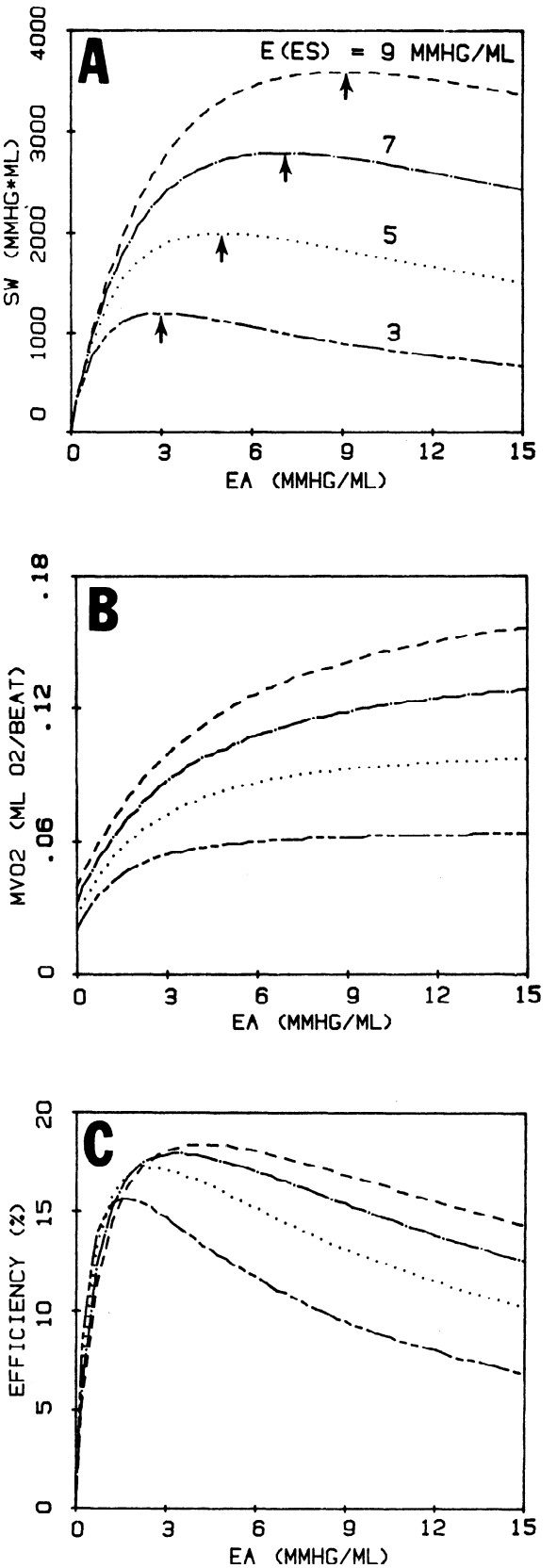

FIG. 3. Relation between afterload impedance as quantified by effective arterial elastance (E_a) and stroke work (*A*; SW), myocardial O_2 consumption (*B*; MVO_2), and efficiency (*C*) for different values of end-systolic elastance (E_{es}) as indicated in *A*. *Arrow heads in A*, E_a value equal to set E_{es} value. This is point at which SW is maximum. See text for other parameter values.

DISCUSSION

In the present analysis we derived analytic expressions for ventricular SW, ventricular O_2 demand, and ventric-

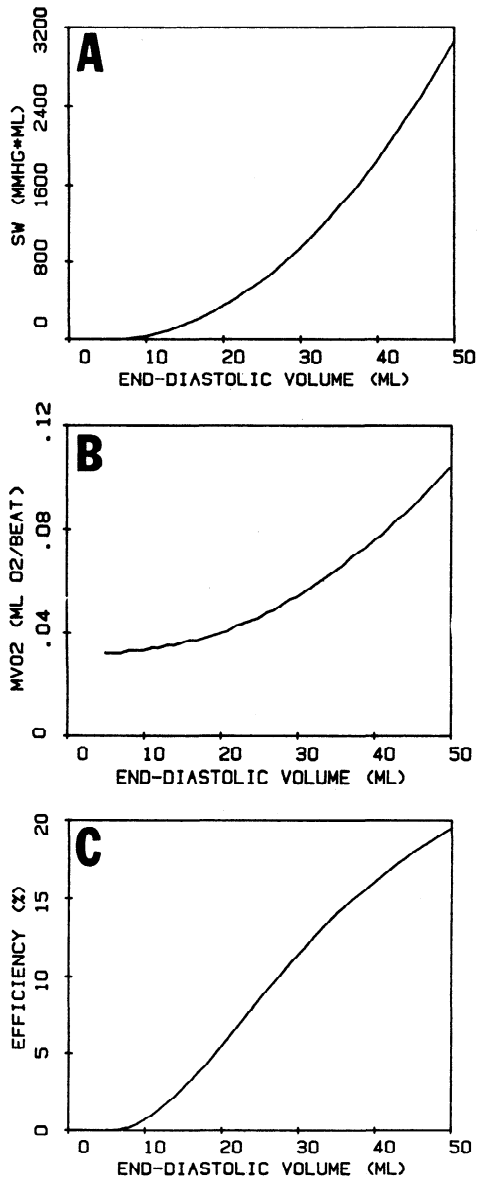

FIG. 4. Relation between end-diastolic volume and stroke work (*A*; SW), myocardial O_2 consumption (*B*; MVO_2), and efficiency (*C*). See text for parameter values.

ular efficiency, in terms of those parameters that characterize the properties of the heart and of the arterial system. The ventricular contractile state was quantified by E_{es} and V_0 (27). The arterial properties were defined, as in many previous investigations, by aortic input impedance (9, 12, 13, 16, 17, 38), which we represented by the three-element Windkessel model (37). To match the elastance parameterization of ventricular properties, the aortic impedance was also expressed as an effective elastance, E_a, a parameter that incorporates the values of the Windkessel model elements and heart rate (33, 34). Finally, the experimentally established linear correlation between O_2 consumption and ventricular PVA provided a convenient means to relate O_2 consumption to the working conditions of the ventricle as they are depicted on the P-V diagram (30).

Many definitions of ventricular efficiency have been considered in the past (1, 6, 7, 19, 32). Each definition deals with a separate aspect of myocardial or cardiovascular energetics, and no one definition is more correct than the others; the choice of definition is dependent on the particular question being addressed. In the present analysis we used the most conventional definition of ventricular efficiency, specifically, the ratio of the external mechanical work done by the ventricle (i.e., SW) to the ventricular O_2 consumption. Our reasoning for the choice of SW as the numerator in the efficiency equation is that this quantity represents the useful fraction of ventricular energy transferred to the arterial system. From the point of view of the body tissues, SW is the important measure of the service that the heart provides in transporting O_2; the amount of potential energy associated with a given amount of external work is, in a sense, inconsequential to the rest of the body organs.

There are five main points brought out by the present analysis: *1*) SW is maximum when $E_a = E_{es}$; *2*) the afterload that results in the greatest efficiency is always less than that which provides the greatest SW; *3*) efficiency is more sensitive to changes in afterload than is SW; *4*) the SW and efficiency of a weak heart are more sensitive to changes in afterload than in a strong heart; and *5*) there is a sigmoidal relation between ventricular efficiency and end-diastolic volume that reaches its maximum at volumes outside the upper limit of the physiological range.

The results of this theoretical analysis are in concordance with previous experimental work. Wilcken et al. (38) demonstrated in an open-chest canine preparation that when afterload resistance was either increased or decreased from its physiological value that SW decreased. Consistent with those results, Van den Horn et al. (35) more recently showed in an in situ cat model that the physiological load on the heart was such that its stroke work was maximal. Piene and Sund (19) have shown in isolated supported cat hearts that, under physiological conditions, pulmonary arterial properties and right ventricular properties are matched such that right ventricular efficiency will be maximal. Maximization of stroke work with matched ventricular and arterial properties (i.e., with $E_{es} = E_a$) has been theoretically predicted and experimentally validated by Sunagawa et al. (34) in the isolated physiologically loaded canine heart. Finally, it has been demonstrated experimentally by Elzinga and Westerhof (6) in isolated physiologically ejecting cat hearts that the maximal SW occurs at a higher afterload resistance than that which yields the maximal efficiency (defined in an identical manner as in the present study).

Thus the concept of matching of ventricular and arterial properties has gained some popularity in the literature. The results of earlier studies suggest that, under physiological conditions, SW may be the performance index for optimal coupling, whereas others suggest that efficiency may be the optimization criterion. We found some evidence that the ventricular and arterial properties may be matched to maximize efficiency under physiological conditions; this evidence emerges from the model employed in the present study as follows.

For a 20-kg dog at rest the cardiac output is ~2 l/min. If we assume a resting heart rate of 75 beats/min, the

SV will be 26.7 ml. V_0 is estimated to be 5 ml, and end-diastolic volume is ~45 ml. Finally, we estimate that a reasonable value for end-systolic ventricular pressure is ~90 mmHg. Given these constraints on the system, we find illustrated in Fig. 5*A* that E_{es} must have a value of 6.8 mmHg/ml and E_a must have a value of 3.3 mmHg/ml. The value of E_a is obtained from the P-V diagram by taking the absolute value of the slope of the line connecting the end-systolic pressure point and the end-diastolic volume point of the volume axis. The explanation of this method of determining E_a is presented elsewhere by Sunagawa et al. (34). Obviously, with the given restraints, E_a does not equal E_{es} and the SW is ~90% that which occurs under the matched conditions (i.e., with $E_a = E_{es} = 6.8$ mmHg/ml).

If, on the other hand, we set both E_a and E_{es} at the same value, e.g., 5 mmHg/ml (again with $V_0 = 5$ ml), then to obtain a SV of 26.7 ml, end-diastolic volume must be 58.4 ml, and end-systolic pressure would be 133.5 mmHg as illustrated in Fig. 5*B*. Both the end-diastolic volume and end-systolic pressure values are much higher than expected physiologically.

Thus, according to this model, it does not seem possible to obtain simultaneously a physiological SV, end-diastolic volume, and end-systolic pressure with the condition that $E_a = E_{es}$. E_a must always be smaller than E_{es} to obtain physiological conditions.

Furthermore, under the nonmatched conditions depicted in Fig. 5*A*, the ejection fraction will be 59%, which is a reasonable value under physiological conditions. This is in contrast to the less than 50% ejection fraction that would result when $E_a = E_{es}$.

Finally, it can be calculated that, with an E_{es} of 6.8 mmHg/ml, peak efficiency occurs at an E_a value of 3.3 mmHg/ml that is exactly the same as found above necessary to provide physiological operating conditions. Thus we conclude from this analysis that under physiological conditions both ventricular SW and efficiency are near their maximal values, but the arterial system load is adjusted closer to the value that would produce the peak efficiency rather than the maximal SW.

The result that the efficiency is more greatly influenced by afterload than is the SW also favors the concept that ventricular-aortic matching is governed by the efficiency. For a given system variable to qualify as a performance index for optimal coupling between subsystems, that variable should be sensitively influenced by the parameters of the subsystems. As shown in Fig. 2, ventricular SW is relatively insensitive to afterload over a wide range of E_a values, whereas ventricular efficiency is more greatly influenced by afterload; especially, as afterload is reduced below the optimum value there is a rather sharp drop off of efficiency. Also, with increases in afterload above the optimum, the efficiency falls off more rapidly than does SW.

Understanding what the performance index for optimal control of cardiovascular coupling is under physiological conditions may reveal the forces that dictate how ventricular and arterial properties change under conditions of stress and disease states. For example, what governs the vascular response in chronic congestive heart failure (10, 12, 17)? What determines the extent to which the heart hypertrophies in chronic hypertension? The answers to these and many other fundamental questions about heart disease are not known.

Limitations. Several limitations of the present model should be recognized. First, each of the three main assumptions of the present model (linear ESPVR, linear MVO_2-PVA relation, and E_a representation of afterload impedance) has only been thoroughly validated in the isolated canine heart, although most available data from intact animals support these concepts. Nevertheless, the extent to which the present model would apply to the intact conscious animal cannot be assessed with certainty. Second, we have excluded ventricular diastolic properties in the calculation of ventricular SW as a simplifying assumption. This assumption may be reasonable under normal conditions, but under diseased states when ventricular diastolic compliance is reduced or when ventricular volume is very high the error introduced will be larger. Third, we have assumed that the ESPVR is unique and independent of loading conditions. Several studies of isolated canine hearts have shown that the ESPVR is influenced to a small degree by the nature of the afterload (11, 31).

Summary. In the present analysis we presented an

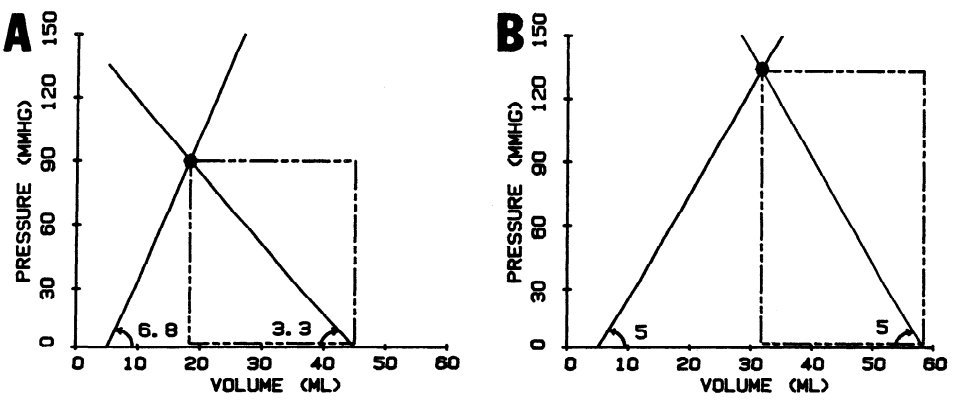

FIG. 5. Hypothetical pressure-volume loops (*dot-dashed lines*). Effective arterial elastance (E_a) can be represented on pressure-volume plane by line connecting end-diastolic and end-systolic pressure-volume points. *A*: we constrain stroke volume (SV) to be 26.7 ml, end-diastolic volume (EDV) to be 45 ml, and end-systolic pressure (ESP) to be 90 mmHg. Then it is required that end-systolic elastance (E_{es}) be 6.8 mmHg/ml and E_a be 3.3 mmHg/ml. *B*: we constrain SV to be 26.7 and $E_{es}=E_a=5$ mmHg/ml. Then EDV will be 58.4 ml and ESP will be 133.5 mmHg. Resulting EDV and ESP in *B* are outside of normal physiological limits. This analysis suggests that, under normal physiological conditions, E_a and E_{es} may not be equal, and thus stroke work may not be optimized.

integrated view of the cardiovascular system, accounting for mechanical and metabolic properties of the ventricle and the properties of the arterial system as they dictate the hydraulic load imposed on the ventricle. This approach relied heavily on the pressure-volume representation of ventricular contractile property. The advantage of the proposed model over more complex models is twofold. First, the model takes the form of analytic expressions that are simple to work with. Second, the model predictions can be easily depicted on the ventricular pressure-volume plane, which facilitates an intuitive understanding of the model predictions. The results of the model analysis are consistent with previous experimental work and suggest that cardiovascular properties are set more toward optimization of ventricular efficiency than stroke work under physiological conditions.

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