

IMPEDANCE MATCHING BETWEEN VENTRICLE AND LOAD

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Impedance matching in the cardiovascular system is discussed in light of two models of ventricle and load: [1] a Thevenin equivalent consisting of a hydromotive pressure source and an internal, source resistance and compliance in parallel; and [2] a time-varying compliance filled from a constant pressure source and ejecting into a load of three components, a central resistor, a compliance, and a peripheral resistance. According to the Thevenin analog, the energy source and the load are matched when the load resistance is T/t times the internal source resistance (T is total cycle length, t is systolic time interval). Both from this model and from the variable compliance model it appears that optimum matching between source and load depends on the compliance of the Windkessel, as low compliance shifts the matching load resistance to a low value. Animal experiments (isolated cat hearts) indicated that both left and right ventricles at normal loads work close to their maxima of output hydraulic power, and, according to experiments in the right ventricle, maximum power output is related to load resistance and compliance as predicted by the above models. From an experimentally determined relationship among instantaneous ventricular pressure and volume (right ventricle of isolated cat hearts), an optimum load impedance was calculated on the basis of the assumption that the ratio between stroke work and static, potential energy developed in the ventricular cavity is maximum. The optimum load impedance found by this procedure closely resembles the normal input impedance of the cat lung vessel bed.

Keywords — Ventricular Thevenin equivalent, Ventricular pressure-volume relationships, Load Windkessel, Load impedance, External power, Ventricular efficiency.

INTRODUCTION

Transfer of energy from an energy source to an external load depends on the impedance of the load. In any such source-load system there must exist a load at which maximum energy is extracted from the source. When the load is such that maximum energy output is achieved, the load and the source and the load constitute, by definition, a matched system. The conditions for source-load matching depend on the characteristics of both the source and the load. In this article the coupling between ventricle and arterial system is treated under

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the hypothesis that a ventricle and its arterial load constitute a matched system. Matching is discussed on a theoretical basis, and experimental evidence that seems to indicate that ventricle-load matching is indeed a characteristic of the cardiovascular system is presented.

THEORETICAL CONSIDERATIONS

A general energy source can be represented by Thevenin's equivalent circuit, which consists of a driving "force" and an internal impedance in which some energy loss will occur when energy is extracted from the source to an external load. In the simple example of a load resistor R_l connected to the poles of a battery (Fig. 1), the current will depend on both the unloaded battery voltage (U_s), R_l , and an internal battery resistance, R_s . The voltage, U_l , over the load resistor is given by $U_l = U_s - R_s I$; hence the relationship between U_l and I describes a straight line of slope $-R_s$.

As $I = U_l/R_l$, it follows that

$$U_l = U_s R_l / (R_l + R_s) . \quad (1)$$

Dissipated power in R_l is given by

$$W = U_s R_l^2 / (R_l + R_s) .$$

By derivation of W with respect to R_l and putting the derivative equal to zero, we obtain the condition for maximum W :

$$R_l = R_s ,$$

which in this case represents the condition for source-load matching. At

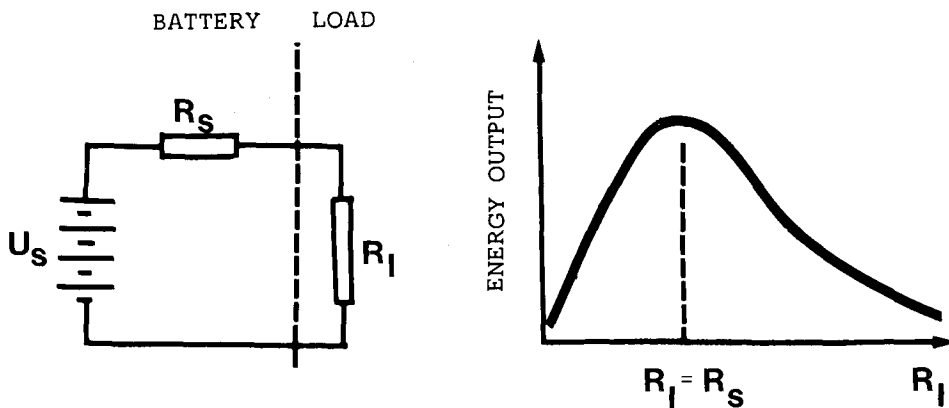


FIGURE 1. Simple example of matching between energy source (electrical battery, U_s , with internal resistance R_s) and load (resistance R_l). The energy output increases as R_l is increased from zero value, goes through a maximum at $R_l = R_s$, and declines when R_l is further increased. The position of maximum energy output defines the condition of matching between source and load.

matching, the power dissipated in R_l will be $W_m = U_s^2/4R_s$, and the current through R_l will be $U_s/2R_s$, which is exactly half the maximum current that can be drawn from the battery of $R_l = 0$.

The heart and its load can not be analyzed in the simple framework of Fig. 1, because there is a nonlinear element—the semilunar valves—between the ventricle and its periphery and because the “driving force” is pulsatile. Still, however, one may assume the validity of Thevenin’s general model and represent the ventricle by a (now oscillatory) “force” and an internal impedance. Above all, Elzinga, Westerhof and collaborators have investigated this model in thorough experiments (1-3). The internal impedance of the Thevenin equivalent can be characterized by relating ventricular pressure to ventricular flow over a range of different external loads. The DC part of the internal impedance, the internal resistance, is given by the relationship between mean ventricular pressure and mean flow (2), just as in the simple example above. The AC part of the internal impedance may be calculated by regression analysis of moduli and phases of the ventricular pressure and flow signals (14). Complete theoretical analysis of the matching conditions based on this representation of heart and load is yet to be performed, but more simple considerations may as well give us some idea how the load influences the energy output from the heart. In Fig. 2, a “hydromotive pressure” source (2) is connected to an internal resistance, R_s , via a unidirectional valve to the external load. The “hydromotive pressure” is here

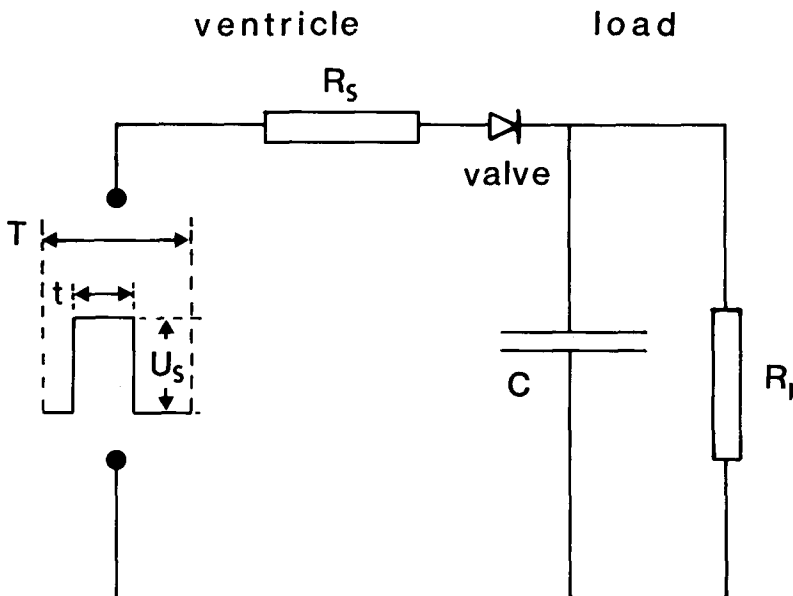


FIGURE 2. Thevenin equivalent of ventricle and load. Ventricle is modeled as pressure source (block pulse of height U_s , width t) connected to internal resistance (R_s) via unidirectional valve to the load. The load is modeled by a simple Windkessel of compliance C and resistance R_l .

assumed to be a block pulse of height U_s and width t of the total cycle length, T , and the external load to be a simple Windkessel of compliance C and resistance R_l . When C is high, the load “pressure,” U_l , will be almost nonoscillatory and have the value

$$U_l = U_s R_l / (R_l + T/t R_s) . \quad (2)$$

This follows from putting “volume” out of the ventricle over the period t equal to “volume flow” through R_l over the period T (i.e., $t \cdot (U_s - U_l) / R_l = T \cdot U_l / R_l$). The expressions of U_l of the simple (Fig. 1, Eq. 1) and the more elaborated (Fig. 2, Eq. 2) models are similar, with the exception that $T/t \cdot R_s$ replaces R_s when the unidirectional valve and the compliant load are introduced. It therefore follows directly that calculation of the matching conditions yields maximum output of energy from the source when

$$R_l = T/t \cdot R_s .$$

Mean “flow” when ventricle and load are matched will be $1/2 \cdot t/T \cdot U_s / R_s$, which is again half the maximum flow (obtained at $R_l = 0$). The load pressure, U_l , will be $U_s/2$, and mean ventricular pressure, U_v , will be $t/T \cdot U_s/2$. Hence at matching, R_l/R_s is also equal to U_l/U_v (mean load pressure/mean ventricular pressure). This result is identical to that of Horn *et al.* (4). The energy dissipated in R_l will generally be both oscillatory and nonoscillatory. At high C , the oscillatory energy will be small and may be neglected. The maximum dissipated energy in R_l will therefore be

$$W_m = t/T \cdot U_s^2 / 4R_s .$$

If $C = 0$ is assumed for the Windkessel, flow from the ventricle and load pressure will both occur in phase with the hydromotive pressure. In this situation, the unidirectional valve loses its significance, as there will never be any pressure gradient directed from the load to the ventricle in any phase of the cycle. Therefore, the conditions for matching will be as in the first example—source and load will be matched when $R_l = R_s$. The total (oscillatory plus nonoscillatory) maximum energy delivered to the external load in the case of low C will be equal to that at high C . The nonoscillatory energy will, however, be at t/T fraction of the total maximum energy:

$$W_m = (t/T)^2 U_s^2 / 4R_s .$$

The above considerations were based on a specific model of ventricle and load. One may ask whether the conclusions are a mere product of the model or whether other models give similar results. In Fig. 3A, an entirely different model of the ventricle is presented. Here the ventricle is represented by

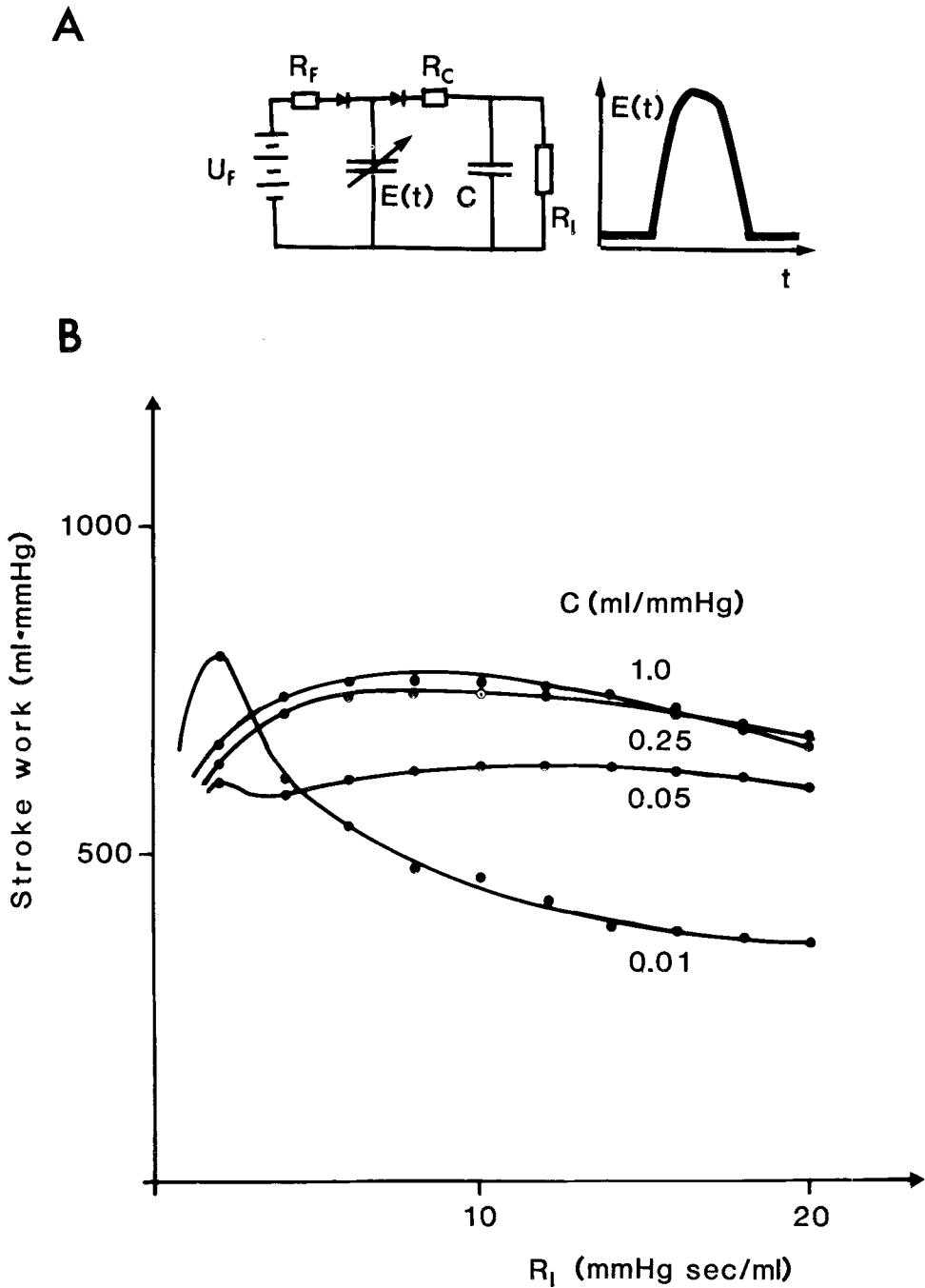


FIGURE 3. A: Variable elastance model of ventricle and load. Time-varying volume elastance $[E(t)]$ was filled from constant pressure source (U_f) and emptied into a load of three components: small central resistor (R_c), compliance (C), and main load resistance (R_l). B: Calculated stroke work vs R_l and C . Note that peak stroke work shifts to a low value of R_l when C is reduced to a low value (curve $C = 0.01$ ml/mm Hg).

a time-varying elastance (analogous to a time-varying capacitor) filled by a constant pressure source (analogous to an electrical battery) over a small load resistance and unidirectional valve. The "ventricle" ejects into a modified Windkessel consisting of a central resistance R_c , a compliance C , and a peripheral resistance R_l . By the introduction of the resistance R_c , the input impedance of the load will approach the value of R_c at high frequencies, and R_c may therefore be taken to represent the characteristic impedance of the load. The experimental work of Suga and co-workers (12,13), have demonstrated the applicability of this ventricular model and have shown that the ventricular pressure may be expressed by $P_v = E(t) \cdot (V - V_d)$, where $E(t)$ is the time-varying elasticity, V the instantaneous volume, and V_d the unstressed ventricular volume at zero pressure. For the present model calculations, one may, e.g., assume a (hypothetical left) ventricle of approximately 15 ml diastolic volume and a V_d of 5 ml. Assuming a diastolic pressure of approximately 10 mm Hg, the diastolic (minimum) E should be 1 mm Hg/ml. The filling pressure should then be 10 mm Hg, and the filling resistor may be set to 0.1 mm Hg sec/ml. The isovolumic pressure of the ventricle may be assumed to be 300 mm Hg at 15 ml volume, and maximum E must then be 30 mm Hg/ml. Hence, E should cycle between 1 (diastole) and 30 (E_{\max}) mm Hg/ml. One may write $E(t) = 1 + 29 \cdot f(t)$, where $f(t)$ may be a squared half sine wave: $f(t) = \sin^2(6.28 t/T)$ for $t > T/2$ and $f(t) = 0$ for $t \leq T/2$. The following equations may now be solved by a time-stepping procedure in a computer:

$$\text{Ventricular pressure: } P_v = E(t) \cdot (V - V_d)$$

$$\text{Diastolic flow: } I_d = (U_f - P_v)/R_f$$

$$\text{Systolic flow: } I_s = (P_v - V_c/C)/R_c$$

(only positive flow allowed; V_c is the volume in C)

$$\text{Load } R_l \text{ flow: } I_r = V_c/C/R_l$$

$$V = V' + (I_d - I_s) \cdot dt$$

$$V_c = V'_c + (I_s - I_r) \cdot dt$$

(dt = time step = 0.005 sec; cycle period $T = 0.5$ sec; ' denotes the preceding value at $t - dt$).

By running through a number of cycles, and each time using the end-cycle conditions as the initial conditions of the next beat, stable configurations of the flow and pressure pulses are reached. These represent the solution of the above set of equations. Stroke work, W , is calculated from ventricular pressure and flow:

$$W = \int_0^T P_v \cdot (I_s - I_d) dt .$$

Results of W vs R_l and C are shown in Fig. 3B. W increases abruptly at low R and, at high values of C , has a maximum around 10 to 15 mm Hg sec/ml. High W is maintained over a wide range of R (curves of $C = 1.0$ to

$C = 0.05$ ml/mm Hg of Fig. 3B). Reduction of C has little effect unless C falls below a lower critical level (curve $C = 0.01$ of Fig. 3C). At low C (below 0.05 mm Hg/ml) the W vs R curve shows a sharp peak at a low R level (R around 2 mm Hg sec/ml).

These data are similar to the results obtained from the Thevenin model and indicate that compliance of the arterial load should be above a certain critical level to achieve efficient matching between ventricle and load. Above this level, the energy output of the ventricle is little influenced by the load compliance.

Assuming high arterial compliance, the arterial pressure pulsations will be small and, accordingly, end-systolic pressure will be close to the mean arterial pressure. This enables one to perform a simple calculation of the matching conditions when the variable elastance model is assumed: End-systolic volume (ESV) is given by E_{\max} , V_d , and load pressure (P_l):

$$\text{ESV} = V_d + P_l/E_{\max} .$$

Stroke volume (SV) and stroke work (SW) are then given by

$$\text{SV} = \text{EDV} - V_d - P_l/E_{\max}; \text{SW} = \text{SV} \cdot P_l .$$

Using peak SW as matching criterion, matching is achieved when

$$\text{EDV} = 2 \text{SV} + V_d$$

and

$$P_l = E_{\max} \cdot \text{SV} .$$

P_l and SV are related by $P_l = R_p \cdot \text{SV} \cdot \text{heart rate}$ (R_p = peripheral resistance), accordingly:

$$R_p = E_{\max}/\text{heart rate} . \quad (3)$$

In the calculation result shown in Fig. 3, optimum R was found around 10 to 15 mm Hg sec/ml, E_{\max} was 30 mm Hg/ml, and heart rate was 2 Hz. These data fit the prediction of Eq. 3.

EXPERIMENTAL CORRELATES

Wilcken *et al.* (17) measured left ventricular stroke work in conscious dogs and induced acute perturbations in afterload by sudden aortic pressure elevation or reduction. They found that stroke work and ventricular power of the heart beat succeeding the load perturbation fell compared with the immediately preceding control beat, both when the aortic pressure was increased and reduced.

Elzinga *et al.* (3) measured ventricular power of both left and right ventricles of isolated cat hearts. Both ventricles were filled to normal end-diastolic pressures (8 and 2 mm Hg, respectively) and pumped into hydraulic models of their respective arterial systems (16). In these loads, the input impedances were adjusted to be similar to those encountered in the systemic and pulmonary vessel beds of the cat. In the control situation, the arterial pressures were 80 and 16 mm Hg, respectively. The arterial pressures on both sides of the heart were altered within single heart beats, and ventricular power and mean flow of both ventricles were calculated for these beats (Fig. 4). For both ventricles well defined maxima of the power vs flow curves were found. The maxima fell closely at the mean flow measured during the control situation (marks on the flow axes of Fig. 4).

These experiments with dog and cat hearts thus indicate that the ventricles normally work at peak output power and that the ventricle-load systems appear, accordingly, to be matched.

In isolated cat hearts, Piene and Sund (7) measured how power output of the right ventricle depends on load resistance and compliance. These hearts pumped into a hydraulic model of the pulmonary arterial system in which the load resistance, R , and compliance, C , could be altered separately. It was observed that the power output rose with R to a maximum at certain R and then declined with further R increase (Fig. 5). With high C ($60 \cdot 10^{-3}$ ml/cm H₂O), R at maximum power (nonoscillatory part) was 9.4 cm H₂O sec/ml, a value that is in the high normal range of cat lung vessel bed resistance. At low C ($6 \cdot 10^{-3}$ ml/cm H₂O) the power maximum shifted toward

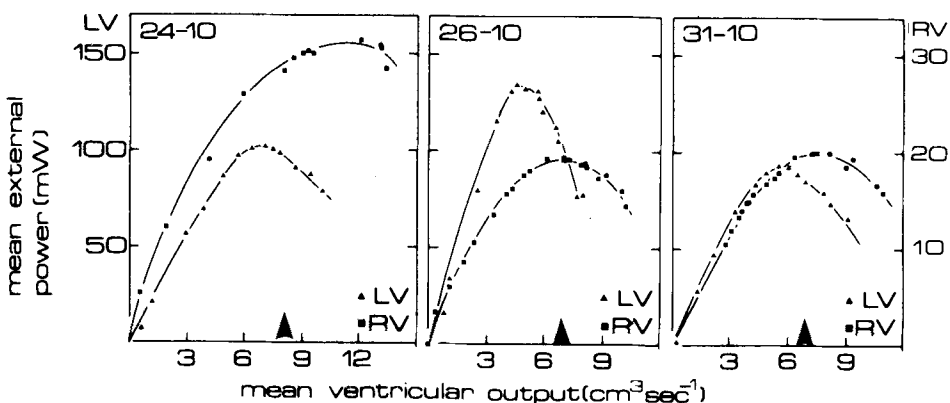


FIGURE 4. Mean external power from left and right ventricles (LV, RV) of isolated cat heart preparation with both ventricles working. RV and LV arterial load pressures were varied independently for the two sides of the heart. Maximum power output for both sides occurred approximately at the ventricular flow output, which corresponded to a physiologic load (arrows on abscissas). Mean external power was calculated as integral of the product of instantaneous ventricular pressure and flow (3).

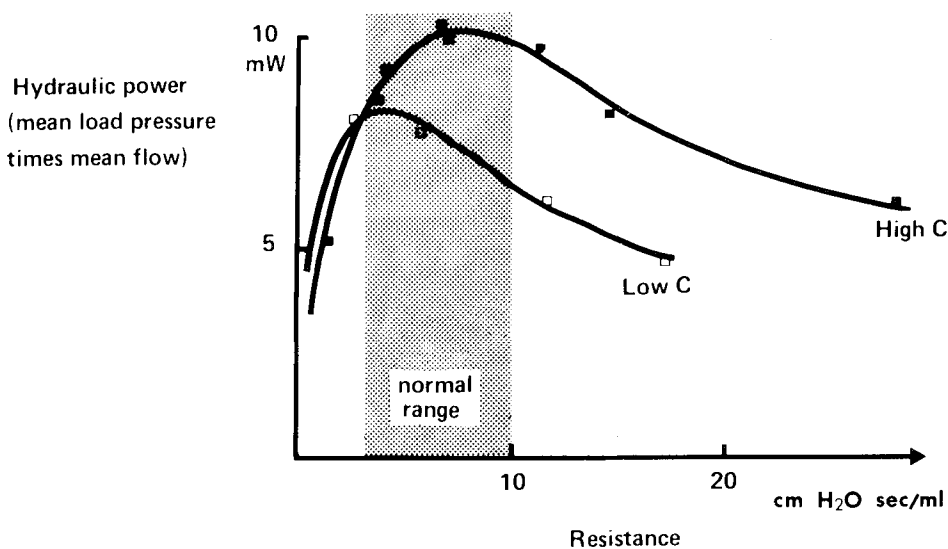


FIGURE 5. Power output of the RV of isolated cat hearts pumping into a load of variable compliance (C) and resistance. Reduction of C caused lowering of power maximum and shift of power-resistance relationship toward lower values of load resistance (7). Note similarity to predictions shown in Fig. 3.

lower R , and maximum (nonoscillatory) power was reduced. Peak total power (oscillatory plus nonoscillatory) was, however, little influenced by C , although the position of the peak still shifted toward lower values of R when C was reduced (7).

These results are completely analogous to the predictions of the Thevenin equivalent discussed above. To further examine this analogy, the (apparent) source resistance of the right ventricle was calculated by relating mean ventricular pressure to mean flow (Fig. 6). The source resistance was found to be 3.4 cm H₂O sec/ml, i.e., 2.7 times the value of R at peak output power at high C . The predicted ratio of approximately 2.5 (estimate of T/t) between optimum load resistance and source resistance was thereby also verified experimentally. As also predicted, the ratio between optimum load resistance and source resistance fell when C was lowered.

In these experiments we did not observe any sudden transition of the power curves as predicted by the calculations based on the variable elastance ventricular model above, possibly for the reason that load C was only altered in large steps. The value of "high" C in these experiments was, however, possibly lower than the normal compliance of the cat lung vessel bed (6). We observed that increase of C above the assumed "high" level had minimal influence on power output, which is in line with the predictions of the variable elastance model.

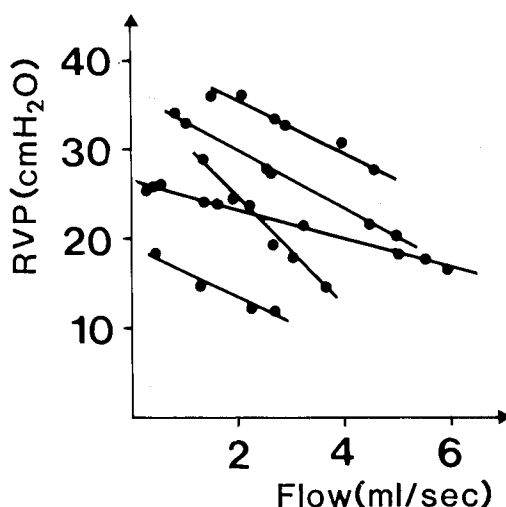


FIGURE 6. Relationship between mean RV flow and mean RV pressure (RVP) of isolated cat hearts. The lines were used to estimate the "apparent" source resistance (R_s) of the RV, calculated as the negative value of line slopes. Results indicated that R_s is approximately 2.5 times smaller than the optimal pulmonary vessel resistance. Adopted from Piene and Sund (7).

PREDICTION OF AN OPTIMUM LOAD IMPEDANCE

According to experimental results of Suga, Sagawa and collaborators (12,13), and of Piene and Sund (8), a ventricle can be characterized by the relationship between instantaneous ventricular pressure and volume. According to Suga and Sagawa (12), this can be written as $P = E(t) \cdot (V - V_d)$, where $E(t)$ is interpreted as a time variable elastance. Piene and Sund (8) preferred a more general expression, $P = \text{function}(V, t)$ to allow for possible nonlinear relations between P , V , and t . In both cases the functional relationship may be expressed as a curved surface in the three dimensions P , V , and t (Fig. 7) (8,9,10,14). A cardiac contraction can be characterized as a path over this P - V - t surface, e.g., the broken curve over the surface shown in Fig. 7. One may now ask whether there will exist a specific path that, in some way, is optimal for the ventricle. As ventricular pressure and flow are both specified by a path, ventricular stroke work may be obtained by integration along a path. If one chooses not to specify the end-diastolic volume that defines the starting point on the surface, it is obvious that maximum stroke work is not an appropriate optimization criterion because the higher the end-diastolic volume, the higher would be the ventricular stroke work. Also, identical stroke work might be found for an infinite number of possible paths. One therefore has to consider for what purpose the ventricle produces energy to the bloodstream. This purpose is twofold: the ventricle should deliver a certain stroke volume according to the oxygen demand of the body, and it should produce a sufficient pressure head in the

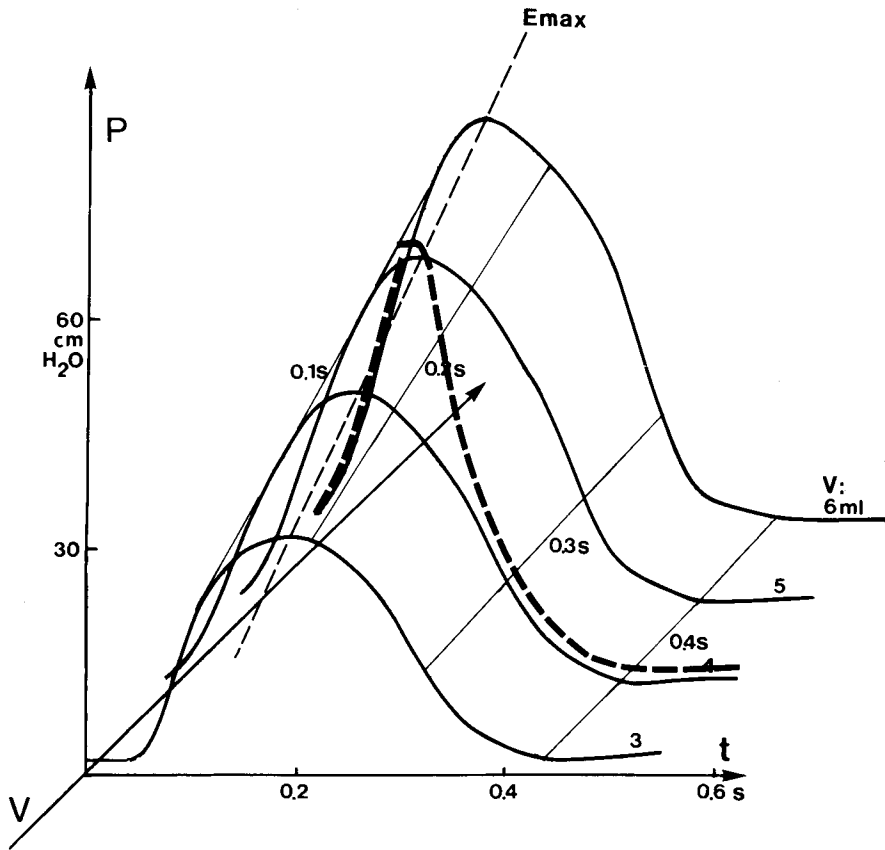


FIGURE 7. Experimentally obtained relationship between RV pressure (P), volume (V), and time (t) after onset of systole of isolated cat hearts. The relationship is displayed as a three-dimensional surface in the P - V - t coordinate system. The "ridge" of the surface is equivalent to the "end-systolic" (E_{max}) line of P - V loops. A contraction can be described by a path on the surface (broken curve), as such path defines $P(t)$ and $V(t)$ and any derived variable (flow, pressure-volume product, etc.). The path is uniquely defined by end-diastolic V and load impedance. From Piene and Sund (9).

arterial system for efficient perfusion of the organ or organs supplied by the ventricle. It is therefore reasonable to use the following three constraints on an optimum path over the P - V - t surface: [1] The stroke volume and the mean load pressure should both be specified. Still, however, a multitude of possible paths may fulfill this constraint. As a further optimization criterion it may be assumed that: [2] the ratio of external ventricular work to total potential, static energy developed within the cavity should be maximum. This means that the ventricle should work with as little relative waste of energy as possible. It was recently demonstrated (5,11) that the total static energy developed in the ventricle can be measured as the area A - B - C - V_d of Fig. 8 [the "pressure-volume area" in the notation of Suga *et al.* (11)]. It has been shown that this area bears a high correlation to the ventricular oxygen

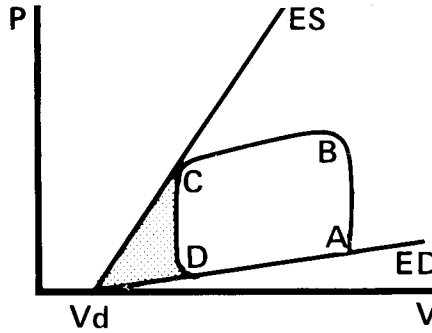


FIGURE 8. Hypothetical P - V loop demonstrating relationship between external ventricular work (area A, B, C, D) and the total static energy developed in the ventricular cavity (area A, B, C, D , plus the shaded area C - V_d - D). Energy efficiency of a ventricular contraction was defined as the ratio between these areas.

consumption (11). Ventricular “efficiency,” Q , was therefore expressed as the ratio of external work (W_{ext}) to total static potential energy (W_{stat}), equal to the ratio of the two areas A - B - C - D and A - B - C - V_d of Fig. 8. With these constraints one might still obtain totally unrealistic paths over the P - V - t surface. For example, as the pressure-volume loop of Fig. 8 is a projection into the P - V plane of a P - V - t surface path, one could imagine a path that, projected into the P - V plane, would fall along A - B - C - V_d of Fig. 8. Such a path could fulfill all the above criteria and would give $Q = 1$. Therefore, one has to assume: [3] a realistic load of some form. We assumed a load consisting of a central resistor, R_c , a central inductance, L , a lumped compliance, C , and a peripheral resistor, R_p , connected to the ventricle over a unidirectional valve as shown in Fig. 9A. The combination of R_c and L assures that the input impedance is approximately flat in the high-frequency range and that the phase angle turns toward zero or becomes positive at high frequencies. By this particular combination of the load components some possible “wild” results are eliminated, but it should be noted that except for R_p , which is defined when stroke volume and arterial pressure are set, the component values are not defined and, consequently, the input impedance consistent with an optimal path over the P - V - t surface is unknown.

To describe the ventricular P - V - t relationship, we measured ventricular pressure (P) of the right ventricle of isolated perfused cat hearts and calculated volume changes by integration of the flow signal (9). A multitude of registrations at different load conditions were combined to express $P = \text{function}(V, t)$. The observed data were primarily fitted to the equation

$$P' = C \cdot \exp[-(t - t_0/t_l)^4] \cdot (V - V_d) + P_d .$$

The constants C , t_0 , t_l , V_d , and P_d were obtained by multiple regression analysis. Pressure residuals, ΔP , between P calculated from this equation

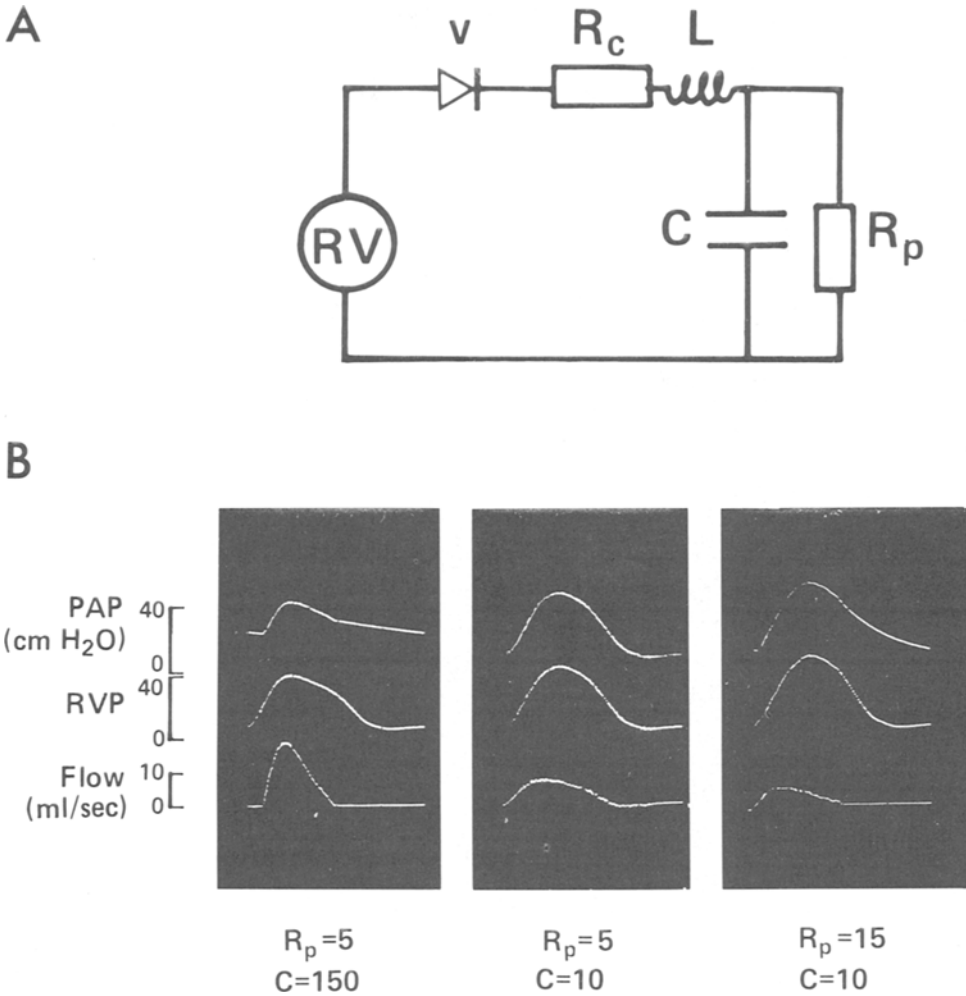


FIGURE 9. Theoretical pulmonary artery pressure (PAP), RV pressure (RVP), and flow as obtained when the RV P - V - t relationship shown in Fig. 7 was combined with a 4-element Windkessel representing the pulmonary arterial system (panel A). R_c , characteristic impedance; L , blood inertia; C , lumped arterial compliance; R_p , lumped peripheral vessel resistance. Lower panel (B) shows calculated PAP, RVP, and flow at three different combinations of C and R_p (9).

and observed P were then used to calculate the constants of the equation

$$\Delta P = a_1 \cdot Vt + a_2 \cdot V + a_3 \cdot t + a_4$$

over small regions of the P - V - t surface.

P at a specific V and t was consequently given by

$$P = P'(V, t) + \Delta P(V, t) .$$

Good fit between observed and calculated pressures was obtained by this procedure (14). The following equations may now be established in order to calculate flow and ventricular and load pressures at specific R_c , L , C , and R_p :

At closed valves ($P < P_l$):

$$\begin{aligned} P &= \text{function (EDV, } t) \\ P_c &= P_c(t = 0) \cdot \exp(-t/R_p C) \\ I &= 0 \\ P_l &= P_c \end{aligned}$$

At open valves ($P > P_l$):

$$\begin{aligned} P &= \text{function (EDV} - \int_0^t I dt, t) \\ I &= (P - P_c')/R_c \\ V_c &= (V_c' + I dt)(1 + t/R_p C) \\ P_c &= V_c/C \\ P_l &= R_c I + (I - I')L/dt + P_c \end{aligned}$$

where EDV = end-diastolic volume,
 P_l = load inlet pressure,
 P_c = pressure over C ,
 I = flow,
 V_c = volume in C ,

and superscript ' denotes value at $t - dt$.

To start computation, initial values were assigned to EDV and to P_c . By running through a number of cycles, each time letting the initial conditions be equal to the end conditions of the preceding beat, all variables were finally repeated from one beat to the next. This represented the stable solution of the above equations. Examples of calculated flow and pressure for the same EDV (5 ml) and three different combinations of R_p and C are shown in Fig. 9B. With selected stroke volume (2.5 ml) and mean load pressure of 20 cm H₂O, R_p was in the following computations fixed at 4.0 cm H₂O sec/ml. For each solution at the selected combination of R_c , L , and C , EDV was changed to find that giving the correct preset stroke volume. C , L , and R_c were varied successively, and the variation of the ventricular energy variables (W_{stat} , W_{ext}), the "efficiency" Q , and EDV were found. Figure 10 shows in the left panel W_{ext} , W_{stat} , and EDV when C was varied at constant L ($=1$ cm H₂O/ml/sec²), R_c ($=0.75$ cm H₂O sec/ml), stroke volume (2.5 ml), and mean arterial pressure (20 cm H₂O) (9).

Increase of C from a low value reduced W_{ext} , W_{stat} , and EDV (left panel), and Q increased (middle panel). C above 100 to $200 \cdot 10^{-3}$ ml/cm H₂O did not, however, improve Q much. When assuming $C = 150 \cdot 10^{-3}$ ml/cm H₂O and varying R_c , a maximum in Q was found for R_c around 1 cm H₂O sec/ml. Variation of L had little effect, a tenfold increase (from

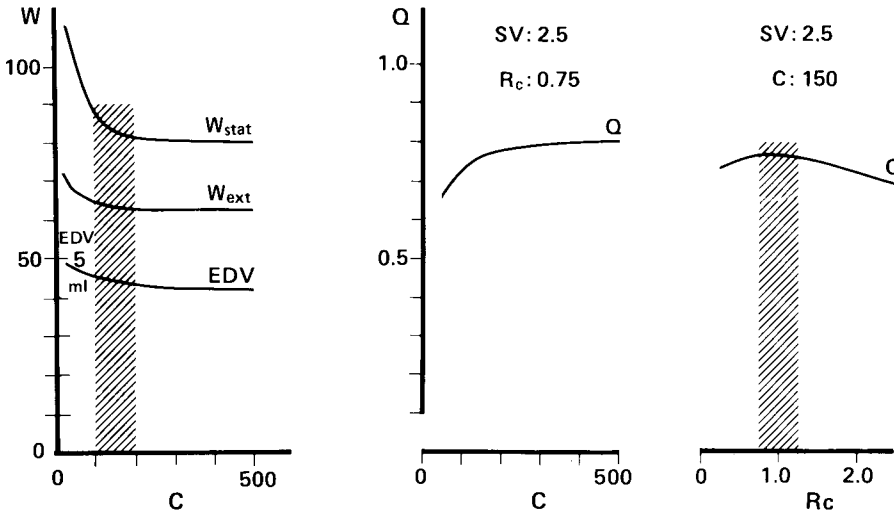


FIGURE 10. Left and middle panels: Calculated developed static ventricular energy (W_{stat}), external work (W_{ext}), energy efficiency ($Q = W_{ext}/W_{stat}$), and EDV consistent with 2.5 ml stroke volume (SV) when load compliance (C) was increased. An optimum value of C was assumed at $C = 100 - 200 \cdot 10^{-3}$ ml/cm H₂O (shaded column of left panel). Right panel: At fixed C ($150 \cdot 10^{-3}$ ml/cm H₂O) variation R_c yielded an optimum Q at $R_c = 1.0$ cm H₂O sec/ml (shaded column of right panel).

1 to 10 cm H₂O/ml/sec²) caused slight upward shift of all curves in the left panel and slight downward shift (about 5%) of the curves in the right panels. Reduction of L below 1 had negligible effect. Choosing other values of C and L did not much affect the position of the Q optimum, which always fell around $R_c = 1.0$ cm H₂O sec/ml. Selecting $C = 150 \cdot 10^{-3}$ ml/cm H₂O and $L = 1$ cm H₂O/ml/sec² [which are close to the values that one may calculate for the cat lung vessel bed (6)] and using $R_c = 1$ cm H₂O sec/ml, one obtains the input impedance shown in Fig. 11. Experimentally determined input impedance of the cat lung is shown for comparison. As can be seen, the optimum impedance derived by this procedure was very close to the actual, experimentally derived impedance of the cat lung vessel bed.

DISCUSSION

The above model calculations, experimental results, and theoretical prediction of the optimal load impedance all put emphasis on the Windkessel properties of the arterial load. High C raises the load resistance that matches the source, minimizes power lost in oscillatory blood movement, and increases ventricular “efficiency,” Q . High C , and the consequently low characteristic input impedance of the arterial load, serves, according to Taylor (15), to “uncouple” the ventricle from the high peripheral resistance. The

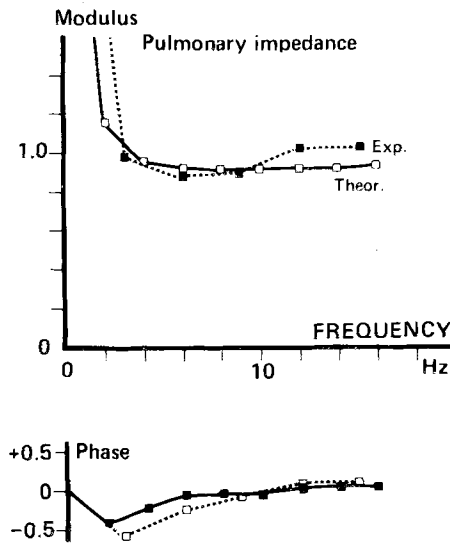


FIGURE 11. Load input impedance when assuming optimum values of load components (see text). Experimentally obtained input impedance at the pulmonary artery of the cat is shown for comparison (*broken curves*). Modulus is given in $\text{cm H}_2\text{O sec/ml}$ and phase in radians (9).

present analyses show the effect of this uncoupling in terms of impedance matching between ventricle and load.

The hypothesis that a ventricle and the arterial bed constitute a matched energy source-load system borrows its rationale from the field of electrotechnical or mechanical engineering. As the purpose of an energy source—be it an electrical generator, a hydraulic pump, or any other device—is to transfer energy to the external load, the load should be so constructed that the extractable energy from the source is maximum. Some problems arise when this concept is adapted on the cardiovascular system. For example, it is well known that the power output of a ventricle increases with the diastolic filling of the ventricle (Starling's law of the heart), with increased inotropy and with heart rate. According to the premise that the ventricle optimally works at maximum output power, the conclusion might be drawn that diastolic filling, inotropy, and heart rate should all be maximum. This is clearly contradictory to what one observes. The clue to this contradiction is that the ventricle, the energy source, changes properties with the diastolic filling, inotropy, or heart rate. Therefore, the same external load will not match the ventricle at any diastolic filling or inotropic or chronotropic state.

This raises a number of questions for future research: First, does the concept of ventricle-load matching hold both during rest and during bodily exercise? And in that case, how are the necessary adjustments of the arterial bed accomplished? Second, how is the matching influenced by cardiac or

arterial diseases, and do reactive changes in the cardiac muscle, e.g., during hypertension or after infarctions, bear any relationship to ventricle-load matching? Studies of ventricle-load matching under such circumstances may be warranted.

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