Optimal Power Generation by the Left Ventricle
A Study in the Anesthetized Open Thorax Cat

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SUMMARY. We studied the interaction of the left ventricle and the systemic arterial bed in the open thorax cat. In the steady state, the ventricle can be characterized by the pump function graph (i.e., the relationship between mean left ventricular pressure and mean outflow). From this pump function graph, the apparent source resistance of the heart is found. Apparent source resistance is defined as the ratio of the difference between maximal and actual mean left ventricular pressure, and mean outflow. The arterial system can be characterized by the ratio of mean aortic pressure and mean flow (peripheral resistance). The pressure and flow at which the heart operates is defined as the working point. We have investigated whether the ventricle in the intact cat is working optimally, i.e., that it cannot increase work output further at the end-diastolic volume, contractile state, and prevailing heart rate. This condition is considered as "matching" of ventricle and load. It could be shown that optimal power is transferred when the ratio of peripheral and apparent source resistance equals twice the ratio of mean aortic and mean left ventricular pressure (the matching principle). In four cats, we observed that mean aortic and mean left ventricular pressures are proportionally related. Mean external power (the time integral of the product of pressure and flow divided by cycle length) and steady power (the product of mean pressure and mean flow) were found to be proportional as well. These proportionalities allow for the calculation of peripheral resistance and mean external power from the pump function graph. Pump function graphs were determined in three groups: control (n = 9), atrial pacing (n = 8), and halothane (n = 5). We compared the ratio of peripheral and source resistance at the working point and at the point of optimal work output (expressed in steady ventricular power). It could be shown that, in all investigated groups, the power optimum and the working point coincide. It was concluded that circulatory control in the intact anesthetized cat keeps the ventricle at optimal work output under the conditions studied. (Circ Res 56: 252-261, 1985)

IN this study, we investigated the interaction between the left heart and the systemic arterial bed in the anesthetized open thorax cat. In our approach, the heart is regarded as a source (pump), loaded by the arterial system. The pumping ability of the heart can be described quantitatively by the pump function graph (Elzinga and Westerhof, 1973, 1978, 1979). This graph relates mean left ventricular pressure and mean flow and allows for determination of the (apparent) source resistance. The arterial system is described by its peripheral resistance, i.e., the ratio of mean aortic pressure and mean flow. We attempt to answer the following questions: (1) Does the heart in the anesthetized open thorax cat pump at optimal external power under control conditions? (2) Do changes in heart rate and inotropic state move the working point (i.e., the mean pressure and mean flow value at which the heart operates) closer to or further away from the point where external power is optimal? (3) Can we derive quantitative relations between (apparent) source resistance of the heart and peripheral resistance as a basis for a "matching" principle?

Methods

Surgical Procedure

Male cats (2.8-4.2 kg) anesthetized with sodium pentobarbital (Nembutal), 40 mg/kg, were artificially ventilated with a mixture of room air and oxygen (2:1). During surgery, halothane (1.5 vol%) was added to the ventilatory gas mixture. Oxygen and carbon dioxide content of the expired air were continuously monitored by a capnograph (Datex Normocap), and kept constant at 23-27 and 3.5-3.8 vol%, respectively, by adjusting the room air-oxygen ratio and the tidal volume. In this way, it was possible to maintain blood gas values stable within the normal ranges for the anesthetized cat, i.e., pH 7.32-7.42, arterial Pco2 28-32 mm Hg, and arterial Po2 > 100 mm Hg (Fink and Schoolman, 1963). Body temperature was maintained at 37°C with a heating pad. A midline sternotomy was performed. All wound edges were cauterized carefully to minimize blood loss. The pericardium was opened longitudinally in the midline, and the edges sutured to the thoracic wall. The ascending aorta was then dissected free from surrounding structures, up to the root of the right brachiocephalic trunk. Two concentric purse-string sutures were put in the ventral wall of the ascending aorta, 1-1.5 cm cranial from the aortic valves. A stiff polyeth-
ylene catheter was introduced into the left ventricular cavity through the apex to obtain left ventricular pressure. The animals were heparinized with 1000 IU/kg. From this point onward, two different procedures were followed.

**Pressure and Power Experiments**

In four cats, a stiff polyethylene catheter was introduced through the purse-string sutures into the proximal aorta. The catheter tip was positioned approximately 0.5 cm distal to the aortic valves. This catheter was used to measure aortic pressure. A perivascular flow probe put around the root of the ascending aorta completed the instrumentation. These experiments were used to relate mean left ventricular pressure to mean aortic pressure, and to investigate how mean external power and steady power compare.

**Experiments Relating Heart and Arterial System**

In 22 cats, the ascending aorta was partially clamped with an atraumatic side clamp (Cooley). A hole was punched in the ventral aortic wall in the center of the purse-string sutures. Then the clamp was put more dorsally, obstructing the blood flow toward the periphery completely. During this procedure, femoral artery pressure was measured. The cannula of the artificial load (see below) was introduced through the hole in the aortic wall. Both purse-string sutures were immediately tightened, and the Cooley clamp removed. The duration of complete flow obstruction was less than 10 seconds. The aortic occluder (Fig. 1) was then put in place, just distal to the cannula. A femoral vein was cannulated for reinfusion of blood ejected into the reservoir of the artificial load. Finally, a perivascular electromagnetic flow probe was put around the root of the ascending aorta.

**The Artificial Load**

To obtain left ventricular pump function graphs in the open thorax cat, we measured left ventricular pressure and flow while the left ventricle was made to eject against different artificial loads, imposed on a beat-to-beat basis. This beat-to-beat approach was used to avoid changes in left ventricular filling and inotropic state. To provide different loads to the left heart on a beat-to-beat basis, a combination of two devices was used, as shown in Figure 1. Normally, device A was closed, and device B open; the heart then was loaded by the animal's arterial system. When ejection against an artificial load was desired, device B occluded the aorta just proximal to the right brachiocephalic trunk, while, at the same time, the valve in device A was opened. In this situation, the heart ejected against an adjustable resistance, into a reservoir from which the blood was returned to the animal. Changes from normal to artificial load were performed in selected diastoles by a trigger pulse, derived from the cardiotachometer (see below). The devices were reset after the beat studied, so that the normal periphery loaded the heart except during the selected beats with artificial load. This procedure enabled us to obtain heartbeats under different loading conditions for a constant inotropic state and filling.

**Measurements and Recordings**

During all experiments, left ventricular, ascending aortic, and femoral artery pressures were measured with Statham P23Db transducers. The pressure transducers were calibrated before and after each experiment against a Wallace and Tiernan precision manometer. In the experiments relating the heart and the arterial system, it was verified that mean aortic and mean femoral artery pressures were comparable, which means that the devices caused no obstruction of the ascending aorta. Heart rate was obtained from left ventricular pressure via a cardiotachometer. Trigger pulses derived from the cardiotachometer were used to activate the artificial load (if present). These pulses were also recorded to control sampling during off-line A to D conversion of the pressure and flow signals. Flow in the ascending aorta was measured by a perivascular cuff probe (Skalar Transflow 601 system). Zero drift was corrected for by automatic zero control during diastole (Dijkema and Elzinga, 1973). Flow through the artificial load was measured by a cannulating electromagnetic flow probe made in our workshop. Flow probes were connected to Skalar flowmeters (Transflow 601). Both flow-measuring systems were calibrated in vitro with saline. All signals were recorded on an eight-channel ink writer (Elena Schoenander, type EMT 81), and on analog tape (SE 7000).

**Experimental Protocol**

**Pressure and Power Experiments**

The following interventions were used to obtain a wide range of pressures and flows: (1) halothane (0.5–3.5 vol%), (2) norepinephrine (0.2–0.8 mg/kg per min), (3) atrial

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**Figure 1.** Devices used to apply different artificial loads to the left ventricle on a beat-to-beat basis. Device A consists of a cannula, which is normally closed by a pneumatic valve. An adjustable outflow resistor mounted at the distal end of the cannula constitutes the actual artificial load. By opening the valve and closing the aortic occluder (device B) in a selected diastole, the left ventricle faces this load during the next systole. A cannulated flow probe permits measurement of blood flow. Via a side opening in the cannula, proximal aortic pressure can be measured continuously. The bore of the cannula is 6 mm, except at the very proximal end, where it is 3 mm. Device B is a pneumatic occluder, used to obstruct all flow to the arterial system when an artificial load was applied. Both devices could be controlled separately. At the right hand side, both devices are shown in situ.
In the type B experiments, the signal driving the aortic conversion was carried out over exactly one heart period. Ascending aortic flow signals were converted from analog to digital form. In the type A experiments, A to D artificial load (in the experiments of type B only), and ular pressure, ascending aortic pressure, flow through the end of the run differed less than 10%.

**Experiments Relating Heart and Arterial System**

Each experimental run consisted of 12-24 beats, during which the left ventricle faced a different artificial load. The time between two such beats amounted to 2 or 3 minutes. This time was sufficient to reach the stable control condition again. At the start and end of an experimental run, an isometric beat was imposed by closing the outflow resistor in device A completely. After each ejection into the artificial load, the blood was returned from the reservoir to the animal. Normal heart beats, i.e., the heart ejecting into the animal's own arterial system, were recorded in the steady state. These normal beats were used to calculate peripheral resistance.

We studied the interaction between the heart and the arterial system during control (n = 9) and for two interventions: (1) changes in heart rate (n = 8), and (2) reduction of contractility and decrease of peripheral resistance (n = 5). These interventions were brought about by increasing the pace frequency, and by administration of halothane to the animal. Except for the control series, the hearts were paced from the right atrium.

Experimental runs were accepted if mean left ventricular pressure of the isometric beats at the start and at the end of the run differed less than 10%.

**Data Analysis**

Data analysis was performed off-line, from analog tape, with a DEC PDP 11/34 computer. Heart rate, left ventricular pressure, ascending aortic pressure, flow through the artificial load (in the experiments of type B only), and ascending aortic flow signals were converted from analog into digital form. In the type A experiments, A to D conversion was carried out over exactly one heart period. In the type B experiments, the signal driving the aortic occluder was used to control the period of sampling by the computer. Sampling was performed at a rate of 500 Hz. The digitized tracings were visualized one by one by plotting every signal as a function of its sample number on a storage oscilloscope (Tektronix 611). An example of a graphic representation taken from an experiment of series B is shown in Figure 2B. The original recording is shown in Figure 2A.

At the start of the tracings in Figure 2B, the heart is in diastole; the aortic valves are closed, and aortic pressure exceeds left ventricular pressure. At the moment the tracings start, the pulse to close the aortic occluder has been given, but closure is effected after a mechanical delay of about 60 msec. The moment of actual closure can be seen from the pressure oscillations in the tracing of aortic pressure (moment T1). Until then, the valve in device A remained closed. Hence, flow is zero per definition. This initial flow level is used as the zero value during the analysis. Then, about 20 msec later, the valve in device A is opened, while the heart is still in diastole (moment T2). Since the blood in the proximal aorta is under pressure, blood is expelled through device A, a positive flow is measured, and aortic pressure falls (emptying of proximal aortic windkessel). When systole starts, left ventricular pressure rises, the aortic valves open, and the heart ejects into the artificial load. Shortly after the onset of the next systole, the valve in device A is closed again, and the aortic occluder (B) is opened, reestablishing the natural load conditions to the heart (not shown in Fig. 2B).

**Calculations**

Mean pressures were obtained by integration of the respective signals over the heart period, and dividing by this period. However, in the experiments of type B, it is not correct to integrate the measured flow signal over the whole cardiac cycle due to (partial) changes in the blood volume stored in the proximal aorta. This amount of blood stored is assumed to depend only on pressure in the ascending aorta. Therefore, the integration period was taken between isobaric points, starting at the onset of pressure rise (i.e., between moments 11 and 12, Fig. 2B). Following this procedure, it is implicit that no ejection should occur after moment 12. When the decays of flow in the phases marked 1 and 2 are identical. The differences in the decays were small. For further explanation, see text.
of stroke volume in any experiment. All integrations were carried out numerically, using Simpson’s rule.

Left ventricular end-diastolic pressure (LVEDP) was determined at 20 msec before the onset of rise of the dP/dt curve. Left ventricular dP/dt was determined by differencing the digitized tracing of the left ventricular pressure curve.

Pump function graphs were fitted (Marquardt, 1963) with a parabolic function (Eq. A1). \( P'_{\text{sys}} \) and \( F'_{\text{sys}} \) are the intercepts of the graph with the pressure and flow axis. The optimum of mean left ventricular power (Eq. A9) is found when flow is equal to \( F'_{\text{sys}}/\sqrt{3} \).

Results

Pressure and Power Experiments

Figure 3A shows mean aortic pressure in four cats, plotted as a function of mean left ventricular pressure for a wide range of values (mean \( P_0 \) from 15 to 100 mm Hg). The data are fitted (Marquardt, 1963) with a straight line through the origin. Numbers of data points, coefficients of correlation, and regression coefficients are given in Table 1. The observed proportionality (constant \( a \)) makes it possible to couple the pump function graph and the systemic arterial pressure-flow relation. Peripheral resistance can now be calculated (Eq. A12) for any point on the pump function graph.

The relations between mean external power and steady ventricular power, and between mean external power and steady aortic power (for definitions see Appendix, Eqs. A4, A5, and A6) are shown in Figure 3, B and C. Again, proportional relationships are found between the steady powers and mean external power. From Table 1, it can be seen that a reliable description of the data in Figure 3 is given by a single coefficient characterizing each experiment. From the slopes of the relationship between mean external power and aortic steady power (Fig. 3C), it can be seen that aortic oscillatory power (i.e.,

![Figure 3. Relationships between mean aortic pressure and mean left ventricular pressure (panels A), between mean external power and left ventricular steady power (panels B), and between mean external power and aortic steady power (panels C) in four cats. All data were fitted with straight lines through the origin. The dashed lines in panels B and C are lines of identity, and the differences between the data and these lines are equal to oscillatory power. Ventricular oscillatory power is a large part of mean external power; aortic oscillatory power is a small fraction. See Table 1 for details.](image-url)

![Table 1. Proportionality Constants](table-url)
FIGURE 4. Pump function graphs from nine experiments on open thorax cats. These graphs were all determined with the hearts beating at their natural rate and filling. The working point in each graph is indicated by W; the mean flow value for which external power transfer by the heart would be optimal (W_{opt}; c.f. Fig. 5) is indicated by the dotted lines.

In summary, we now have the tools to determine, from the pump function graph, the point of optimum external power, the working point, and peripheral and apparent source resistances.

Experiments Relating Heart and Arterial System

Control

Figure 4 shows pump function graphs obtained from experiments in nine cats. The hearts of these cats were allowed to pump at their own rates. Apart from the anesthetic agent, no drugs or fluids were administered. The data are fitted with the parabola of equation A1.

Number of data points, correlation coefficients, and intercept values are shown in Table 2. Coefficients of variation of heart rate, left ventricular dP/dt, and LVEDP in these experiments were 0.65%, 2.52%, and 7.94%, respectively (average values), reflecting the stability of the preparation during the determination of the pump function graphs.

From the pump function graph, left ventricular steady power can be derived as a function of mean flow (Eq. A7). An example of the “fit” of this derived curve to the actual left ventricular steady power data is given in Figure 5. Optimal left ventricular steady power and the mean flow value for which it oc-
FIGURE 5. Left ventricular steady power as a function of mean flow. The curve was derived from the equation of the pump function graph, using the same values of $P_w$ and $F_w$. The mean flow value for which the optimum value of steady power is found ($F_w^{\text{max}}$) corresponds to the position of the dotted lines in Figure 4. (Data from experiment PFCL)

curred were calculated according to Equations A8 and A9. This flow value ($F_w^{\text{max}}$) is indicated in each pump function graph; the working point is indicated by $W$ (Fig. 4). In all nine experiments, the ratio between mean aortic pressure and mean left ventricular pressure was determined from beats recorded when the animal's own periphery loaded the heart. The Rp:Rs ratio in the point of optimal mean external power can be obtained (see Appendix), and is found to be equal to twice the proportionality constant ($a$) between mean aortic pressure and mean left ventricular pressure:

$$Rp:Rs = 2a$$  \hspace{5cm} (A14)

Rp:Rs ratios were calculated at the working point, and at the power optimum of each experiment. Table 2 shows the individual ratios and the mean and standard deviations at working points and power optima. Rp:Rs ratios at the working point could not be shown to differ significantly from those at the point of optimal external power (paired $t$-test, $P > 0.10$). When power at the working point is expressed as a percentage of optimal power, an average value of 97.8% is found. Individual percentages are shown in Table 2 as well.

Interventions

Changes in Heart Rate. In eight cats, experimental runs to determine the pump function graph were carried out at two different heart rates. Of these two, the one with the lower heart rate was regarded as control. Figure 6 shows an example of such an experiment.

In Table 3, the relative changes in heart rate, $P_{max}$, and $F_{max}$, are given for each experiment. It can be seen that the relative changes in $P_{max}$ and $F_{max}$ are about equal, indicating a parallel shift of the pump function graph. Also, the sign of the changes is negative, indicating an inward shift of the graph due to pacing at increased frequency. In experiments on the isolated cat heart, it was found that, for constant filling, an increase of heart rate causes the graph to shift outward (Elzinga and Westerhof, 1980); an outward shift was also found for increased filling at constant heart rate (Elzinga and Westerhof, 1978). In our experiments, an increase in heart rate resulted in a reduction of left ventricular end-diastolic pressure (LVEDP). (The small values of LVEDP led to large changes percentage-wise. The actual control values were 0.76, 0.94, 0.97, 0.92, 0.98, 1.05, and 0.56 kPa, respectively.) Apparently, heart rate changes and changes in LVEDP counteract in these experiments.

The effect of atrial pacing on the arterial system is also shown in Table 3. Except in experiment 4, aortic pressure and flow at the working point were

<table>
<thead>
<tr>
<th>Cat</th>
<th>$P_{max}$</th>
<th>$F_{max}$</th>
<th>$HR$</th>
<th>$LVEDP$</th>
<th>$P$</th>
<th>$F$</th>
<th>$Rp$</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR1</td>
<td>-17</td>
<td>-15</td>
<td>+14</td>
<td>-9</td>
<td>-4</td>
<td>-30</td>
<td>-17</td>
</tr>
<tr>
<td>HR2</td>
<td>-9</td>
<td>-2</td>
<td>+11</td>
<td>-17</td>
<td>-14</td>
<td>+1</td>
<td>+17</td>
</tr>
<tr>
<td>HR3</td>
<td>-15</td>
<td>-18</td>
<td>+11</td>
<td>-27</td>
<td>-23</td>
<td>-11</td>
<td>+15</td>
</tr>
<tr>
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<td>-0</td>
<td>+2</td>
<td>+13</td>
<td>-14</td>
<td>+4</td>
<td>+2</td>
<td>-2</td>
</tr>
<tr>
<td>HR5</td>
<td>-12</td>
<td>-13</td>
<td>+13</td>
<td>-30</td>
<td>-22</td>
<td>-2</td>
<td>+25</td>
</tr>
<tr>
<td>HR6</td>
<td>-18</td>
<td>-16</td>
<td>+20</td>
<td>-24</td>
<td>-17</td>
<td>-18</td>
<td>-1</td>
</tr>
<tr>
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<td>-17</td>
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<td>-20</td>
</tr>
<tr>
<td>HR8</td>
<td>+13</td>
<td>-12</td>
<td>+20</td>
<td>+170</td>
<td>-6</td>
<td>-8</td>
<td>-3</td>
</tr>
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</table>

Mean $-10$ $-11$ +14 $-12$ $-12$ $+2$

$P_{max}$ and $F_{max}$ = pressure and flow axis intercepts; HR = heart rate; LVEDP = left ventricular end-diastolic pressure; $P$ = aortic pressure at working point; $F$ = flow at working point; $Rp$ = peripheral resistance.
Heart rate was kept constant by right atrial pacing. Working halothane to the ventilatory gas mixture in the same cat (experiment FIGURE 7). Pump function graphs both before and after addition of halothane to the ventilatory gas mixture in the same cat (experiment HAL1). Heart rate was kept constant by right atrial pacing. Working points, etc., as in Figure 6. O: control; *: halothane.

<table>
<thead>
<tr>
<th>Lower heart rate</th>
<th>Increased heart rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cat</td>
<td>Rp:Rs at WP &amp; at PO</td>
</tr>
<tr>
<td>HR1</td>
<td>3.76 &amp; 3.44</td>
</tr>
<tr>
<td>HR2</td>
<td>4.16 &amp; 3.74</td>
</tr>
<tr>
<td>HR3</td>
<td>5.32 &amp; 3.48</td>
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<td>HR4</td>
<td>4.90 &amp; 3.48</td>
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<tr>
<td>HR5</td>
<td>2.96 &amp; 3.52</td>
</tr>
<tr>
<td>HR6</td>
<td>3.00 &amp; 3.52</td>
</tr>
<tr>
<td>HR7</td>
<td>2.88 &amp; 3.52</td>
</tr>
<tr>
<td>HR8</td>
<td>3.06 &amp; 3.48</td>
</tr>
<tr>
<td>Mean</td>
<td>3.76 &amp; 3.52</td>
</tr>
<tr>
<td>sd</td>
<td>0.95 &amp; 0.09</td>
</tr>
</tbody>
</table>

*Rp:Rs ratios are at the working points (WP) and at the points of optimal external power (PO). Left ventricular steady power (Wm) is indicated as a percentage of its maximum.

Reduced. Statistical analysis could not demonstrate a significant change in peripheral resistance (t-test, P > 0.20). These findings agree with the parallel inward shift of the pump function graph in all experiments; in experiment 4, where aortic pressure and flow remained practically unchanged, the pump function graph did not shift either.

Table 4 shows the individual ratios of peripheral resistance and apparent source resistance, both at the working point and at the power optimum. Neither under control conditions nor during increased heart rate could statistically significant differences be demonstrated between the ratios of peripheral and apparent source resistances at the working point and at the point of optimal external power (P > 0.20).

Halothane. In a series of five experiments, pump function graphs were determined both with and without halothane. This drug is known to depress blood pressure both by its negative inotropic and chronotropic effects on the heart and by reducing peripheral resistance. Heart rate was kept constant by pacing during both runs. After determination of the pump function graph during control, halothane (2 vol%) was added to the ventilatory gas mixture. The animals were allowed to stabilize for approximately 20 minutes before a new series of artificial loads was applied to the heart.

Figure 7 shows an example of an experiment from this series. Relative changes of $P_{\text{max}}$ and $F_{\text{max}}$ due to halothane are given in Table 5. It appears that the flow axis intercept, $F_{\text{max}}$, does not change after halothane (paired t-test, P > 0.20). However, the pressure axis intercept, $P_{\text{max}}$, is affected markedly (paired t-test, P < 0.001); on the average, $P_{\text{max}}$ is reduced by more than 30%. This effect on the pump function graph can be described as an anti-clockwise rotation around the flow axis intercept. LVEDP did not change after halothane intervention (paired t-test, P > 0.20).

The data describing the effect of halothane on the arterial system are also given in Table 5. Mean flow at the working point was relatively little affected (average reduction: 4%); whereas aortic pressure was reduced in all cases (average reduction: 31%). In all experiments, peripheral resistance was reduced as well (17–37%).

The Rp:Rs ratios at the working point and at the power optimum were determined both before and during halothane. These values and the percentage of optimal steady power at the working point are given in Table 6. Statistical analysis (paired t-test) shows that the working point and the power optimum coincide. This holds true both with (P > 0.10) and without (P > 0.20) halothane.

**Discussion**

We have demonstrated in this study that the left heart of the anesthetized open thorax cat pumps at optimal external power output. This holds both under control conditions and after changes in heart rate and inotropic state. We found that pumping at
optimal power output implies that peripheral and apparent source resistance are related, such that their ratio equals twice the ratio of mean aortic and mean left ventricular pressure (Eq. A14).

We were able to obtain these results from the pump function graph alone, due to the quantitative relationships we found to exist between mean aortic and mean left ventricular pressures and between mean external power and left ventricular steady power (Fig. 3). To a good approximation, both relationships appear to be proportional. The proportionality, however, simplifies the calculations considerably.

In two of the control animals (PFC3 and PFC4), the ratio of peripheral resistance and apparent source resistance at the working point is found to be large. This is due to the fact that the ratio Rp:Rs changes nonlinearly with flow (see Eq. A13). When the working point is located to the left of the power optimum, the ratio varies more than when it is located to the right. In Table 2, Rp:Rs ratios deviate more from 2a for smaller flows, than for larger flows. Although not statistically significant, the working point is more often found to the left of the optimum power than to the right. When the working point is located to the left of the power optimum, the heart behaves as a pressure source, i.e., pressure changes little for changes in flow. Such a source would have the possibility of "flow reserve."

Piene and Sund (1979) showed, for a simple analog model, that the ratio \( P_w / P_v \) would approximate the ratio of cardiac cycle time and ejection time if the compliance of the load approached infinity. Using an analog in which the load consists of a three-element windkessel, which is a more accurate representation of the arterial system (Westerhof et al., 1971), we were able to calculate the ratio \( P_{w0} / P_v \) as a function of peripheral resistance (Rp) and arterial compliance (C). In this model, we found a constant ratio \( P_{w0} / P_v \) equal to about 0.75 \( \times T/TE \) (T and Te are cycle time and ejection time, respectively), for a wide range of resistance values, provided that the RpC time of the windkessel amounted to more than 300 msec for heart rates from 2 to 4 Hz (the range in which the cat heart operates). In the pressure and power experiments (A), the RpC time of the arterial system was estimated from the diastolic decay of the aortic pressure curve and was found to be between 900 and 1200 msec. Moreover, reploting mean aortic pressure as a function of mean left ventricular pressure times the ratio \( T/TE \) yielded almost straight lines through the origins \( (r > 0.99) \), with slopes of 0.72, 0.74, 0.74, and 0.80, respectively.

With respect to the proportional relationship between mean aortic and mean left ventricular pressures, it is of interest to note that Sagawa (1967) suggested that one could characterize ventricular pump function by a graph relating mean aortic pressure and cardiac output. In experiments on isolated cat hearts, where compliance and resistance of the loading system were changed independently (Elzinga and Westerhof, 1973), proportional relationships between mean aortic and mean left ventricular pressures can be demonstrated as long as compliance does not fall below half of the control value. Therefore, for smaller compliance values, ventricular pump function can be described only by relating mean left ventricular pressure and output. Although, by itself, Sagawa's concept is limited, his approach is useful in intact animals in view of the results presented here.

Our finding that, under normal conditions, the heart operates at optimal external power is in agreement with that of Wilcken et al. (1964), who showed that acute changes of the load resulted in a diminution of external work in the dog.

A feedback system responsible for such an optimization should operate unambiguously. However, detection of external power is ambiguous, since the same value of external power is found for two different pressures and flows. Moreover, power receptors are not known to exist.

Several authors (O'Rourke, 1965; Kenner and Estelberger, 1976; Yamashiro et al., 1978; Milnor, 1979) proposed that minimization of oscillatory power is a criterion governing the interaction between heart and arterial system. We found oscillatory power in the systemic circulation to be only about 13% of mean external power, which is in agreement with the 10% mentioned by McDonald (1974). From the standpoint of overall cardiac energy turnover, considering that the heart is about 25% efficient (Elzinga and Westerhof 1980), minimization of oscillatory power (13% of 25% energy turnover) seems hardly important.

Wilcken et al. (1964) reported that, if the intra-aortic balloon they used to increase the load to the heart was kept inflated for 30 seconds and then
released, external power always increased. This indicates that, during those 30 seconds, the hearts were not working at their power optimum. This could mean that the mechanism responsible to achieve optimal power output needs more than half a minute. Therefore, mechanisms other than neural may be involved in this interaction. The fact that, in our experiments, the hearts could be demonstrated to operate at optimal external power transfer during halothane indicates that the depth of narcosis does not disturb the control mechanism. This may also point toward a nonneural mechanism.

What mechanisms may lead to a setting of the cardiovascular system so that external power is optimal? In most mammals, blood pressure is about the same. On the other hand, the tissues require a certain blood flow. Both requirements are satisfied at a finite external power value (Stein and Sabbah, 1976; Schultz et al., 1980; Elzinga, 1983). For a given setting of the arterial system, this can be achieved by an infinite number of settings of the heart, but there is only one setting such that external power is optimal for that heart. This is illustrated in Figure 8, where three pump function graphs are shown, all delivering equal pressure, equal flow, and equal external power. Without assuming other criteria, it is not clear why the heart chooses to pump at its power optimum. Such a criterion might be minimization of the rate of energy turnover of the heart.

Finally, one may raise the question whether the observation that ventricle and arterial systems work together to keep power output optimal, can be regarded as a matching principle. The word "matching" suggests here that certain properties of ventricle and arterial system are closely connected and change together in order to achieve a common goal. Within this context, optimization of power output may be regarded as the matching principle for the left ventricle and the systemic circulation.

Appendix

Pump function graphs were fitted (Marquardt, 1963) through the obtained pressure and flow data, using a parabolic function of the form:

\[ \bar{P}_{lv} = \bar{P}_{max} \cdot (1 - F^2/F_{max}^2). \]  
(A1)

where \( \bar{P}_{lv} \) is mean left ventricular pressure, \( \bar{F} \) is mean flow, and \( \bar{P}_{max} \) and \( F_{max} \) are the pressure axis and flow axis intercepts of the pump function graph.

Apparent source resistance (Rs), is a function of mean flow. We can derive Rs from the pump function graph:

\[ \text{Rs} = \frac{(\bar{P}_{max} - \bar{P}_{lv})/\bar{F}}{\bar{F} \cdot \bar{P}_{max}/F_{max}}. \]  
(A2)

Peripheral resistance (Rp) is calculated from mean aortic pressure (\( \bar{P}_{ao} \)) and mean flow data in the control beats as:

\[ \text{Rp} = \frac{\bar{P}_{ao}}{\bar{F}}. \]  
(A3)

Mean external power (\( W_{ext} \)) is defined (McDonald, 1974) as:

\[ W_{ext} = \frac{1}{T} \cdot \int_0^T P(t) \cdot F(t) \, dt, \]  
(A4)

and can be calculated, either from aortic or from left ventricular pressure, leading to essentially the same result. The heart period is \( T \). Steady power is calculated from mean values of pressure and flow.

Aortic steady power (\( W_{ao} \)) is defined as:

\[ W_{ao} = \frac{\bar{P}_{ao} \cdot \bar{F}}{T}. \]  
(A5)

Left ventricular steady power (\( W_{lv} \)) is:

\[ W_{lv} = \frac{\bar{P}_{lv} \cdot \bar{F}}{T}, \]  
(A6)

and can be obtained directly from the pump function graph as a function of mean flow (Eqs. A1 and A6):

\[ W_{lv} = \bar{P}_{max} \cdot (\bar{F} - F^2/F_{max}^2). \]  
(A7)

Maximal left ventricular steady power is found when the derivative of Equation A7 with respect to mean flow is zero:

\[ \frac{d[W_{lv}(\bar{F})]}{d\bar{F}} = \bar{P}_{max} \cdot (1 - 3F^2/F_{max}^2) = 0 \]  
(A8)

This optimum is (reached for flow \( F_{lv} \max \):

\[ \bar{P}_{lv_{max}} = \frac{\bar{F}_{max} \cdot \bar{P}_{max}}{\sqrt{3}}. \]  
(A9)
Finally, we define oscillatory powers ($W_{aos}$ and $W_{vos}$) as the differences between mean external power and steady powers:

$$ W = \bar{W} - W_{aost} \quad (A10) $$

and

$$ W_{vos} = \bar{W}_{ext} - W_{lvst}. \quad (A11) $$

It will be clear that aortic oscillatory power is much smaller than ventricular oscillatory power, whereas ventricular steady power is much smaller than aortic steady power.

When mean left ventricular and mean aortic pressures can be assumed to be proportional, as shown in the series of experiments called $A$, we can write:

$$ Rp = a \cdot \frac{P_{lv}}{F}. \quad (A12) $$

From Equations $A1$, $A2$, and $A12$, we can calculate the ratio of peripheral resistance and apparent source resistance:

$$ \frac{Rp}{Rs} = a \cdot \frac{F^{2}_{vos}/F^{2} - 1}{}. \quad (A13) $$

so that, at optimal external power (Eq. A9),

$$ \frac{Rp}{Rs} = 2a. \quad (A14) $$

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