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Arterial Wave Propagation Phenomena, Ventricular Work, and Power Dissipation

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Abstract—The effects of wave propagation phenomena, namely global reflection coefficient ($\Gamma_G[\omega]$) and pulse wave velocity (c_{ph}), are studied in a model of the coupled left ventricle/arterial system. The left ventricle consists of a time-varying elastance, while the arterial system is modeled as a single, uniform, elastic tube terminating in a complex load. Manipulation of model parameters allowed for the precise control of $\Gamma_G(\omega)$ and c_{ph} independent of each other, peripheral resistance, and characteristic impedance. Reduction of $\Gamma_G(\omega)$ and c_{ph} were achieved through increases in load compliance and tube compliance, respectively. The equations describing the system were solved for left ventricular and aortic pressures and aortic flow. From these, stroke volume (*SV*), left ventricular stroke work (*SW*), and steady ($\dot{W}_s$), oscillatory ($\dot{W}_o$), and total power dissipation ($\dot{W}_t$) in the arterial system were calculated. An index of arterial system efficiency was the ratio $\dot{W}_o/\dot{W}_t$ ($\%\dot{W}_o$), with lower values indicating higher efficiency. Reduction of $\Gamma_G(\omega)$ yielded initial increases in $\dot{W}_s$, while $\dot{W}_o$ increased for the entire range of $\Gamma_G(\omega)$, resulting in increased $\%\dot{W}_o$. This reduced efficiency is imposed on the ventricle, resulting in increased *SW* without increased *SV*. On the other hand, decreased c_{ph} yielded in a steady increase in $\dot{W}_s$ and a biphasic response in $\dot{W}_o$, resulting in reduced $\%\dot{W}_o$ for most of the range of reduced c_{ph}. These results suggest that differential effects on arterial system efficiency can result from reductions of $\Gamma_G(\omega)$ and c_{ph}. In terms of compliance, changes in arterial compliance can have different effects on efficiency, depending on where the compliance change takes place. Reasons for these results are suggested, and the role of distributed compliances is raised as a new problem.

Keywords—Wave reflections, Pulse wave velocity, Stroke work, Arterial system power dissipation model experiments.

INTRODUCTION AND BACKGROUND

The energy associated with the movement of the stroke volume (*SV*) from the ventricle into the arterial system is the stroke work (*SW*). Most of this energy is eventually dissipated in the systemic circulation. The dissipation of external ventricular work in the systemic circulation may be viewed as the sum of two components, one steady and one oscillatory. For what appear to be historic reasons, energy delivery by the ventricle is often expressed in terms of average power or work rate. Because multiplying power by time gives energy, and given that the period of the cardiac cycle is known, no information is lost. Expressing the energy quantities as power also allows one to use flow in the relevant calculations of energy dissipation in the arterial system.

Steady power is associated with mean flow and pressure (arterial) and therefore describes the energy needed to overcome the peripheral resistance. Oscillatory power, often called pulsatile power, results from the pulsations of pressure and flow. Regarding the movement of blood into the periphery, this oscillatory power is wasted because it does not contribute to the net movement of blood to the peripheral organs. As such, the ratio of oscillatory power, $\dot{W}_o$, to total power, $\dot{W}_t$, has been used as an index of system inefficiency (13,15). That is, the greater the portion of $\dot{W}_t$ devoted to $\dot{W}_o$, at the expense of steady work, $\dot{W}_s$, the less efficient the system. The pulsatile nature of the heart guarantees that there will be at least some oscillatory power dissipation every beat if ejection occurs. Although wasted in terms of net blood movement, the information contained in pulsations is utilized (*e.g.*, by the baroreceptor reflex system to function properly). The oscillatory contribution to total power in the normal systemic circulation is typically 20% in the dog (11) and somewhat less in humans (14), whereas in the pulmonary circulation it is considerably greater in both humans and dog (12,13).

Berger *et al.* (1) showed with model experiments that the global reflection coefficient, $\Gamma_G(\omega)$, (*i.e.*, reflection coefficient at the system entrance [20]), and the pulse wave velocity, c_{ph}, each affect pressure and flow developed by the left ventricle, and hence its *SW*. This would be expected since the coupling of the left ventricle to the arterial system allows the latter to exert influence on the pumping performance of the former. What was surprising,

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however, was the continuous increase in *SW* with decreasing reflection while *SV* remained unchanged. Thus, it appeared that, all else being equal, reduced reflection leads to reduced pumping efficiency of the left ventricle, since more work is performed with no gain in *SV* (1). Such direct effects of wave propagation phenomena, specifically wave velocity and reflection coefficient, on *SW* and its subsequent dissipation in the arterial system have not yet been studied. In this report, the influence of these wave propagation phenomena on power dissipation and pumping efficiency is investigated in detail with the use of a coupled heart/arterial system model. For a wide range of reflection coefficients and pulse wave velocities, varied independently of each other and other arterial system properties, the generation of *SW* and its dissipation in the arterial system are examined.

THEORY

Pulsatile and Steady Power

The external work of the left ventricle, *SW*, is given by:

$$SW = -\int_{V_{ed}}^{V_{ee}} P_v(t)dV_v = \int_{t_{BE}}^{t_{EE}} P_v(t)Q_{ao}(t)dt, \quad (1)$$

where P_v and V_v are left ventricular pressure and volume, respectively; Q_{ao} is aortic flow; V_{ed} and V_{ee} are left ventricular end-diastolic and end-ejection volume, respectively; t is time; and the subscripts *BE* and *EE* indicate the beginning and end of ejection. The formal definition of *SW* requires that integration be performed over the entire cardiac cycle, but since Q_{ao} equals zero outside the ejection period, the integration limits may be restricted to the ejection period as in Eq. 1. The steady power dissipation is obtained from mean root aortic pressure and flow:

$$\dot{W}_s = \overline{P}_{ao}\overline{Q}_{ao}, \quad (2)$$

where the over-bars indicate mean values. The total oscillatory power is determined from the harmonics of P_{ao} and Q_{ao} as follows:

$$\dot{W}_o = \frac{1}{2}\sum_{i=1}^{N} P_{ao}(\omega_i)Q_{ao}(\omega_i), \quad (3a)$$

where i is the harmonic number and N is the number of harmonics used. Equation 3a can also be written in terms of the input impedance of the arterial system, $Z_{in}(\omega)$, the harmonic ratio of aortic pressure to flow,

$$\dot{W}_o = \frac{1}{2}\sum_{i=1}^{N} Q_{ao_i}^2 Z_{in_i}\cos\phi_i, \quad (3b)$$

where Q_{ao_i} and Z_{in_i} are the magnitudes of the ith harmonic of Q_{ao} and Z_{in}, respectively, and ϕ_i is the phase of input impedance. The total power is the sum of steady and oscillatory powers,

$$\dot{W}_t = \dot{W}_s + \dot{W}_o. \quad (4)$$

Finally, the percentage of oscillatory power, $\%\dot{W}_o$, is given by the ratio,

$$\%\dot{W}_o = \frac{\dot{W}_o}{\dot{W}_t}(100). \quad (5)$$

In addition to the steady and oscillatory powers already described, there is also a kinetic energy component associated with the movement of blood in into the arterial system. This kinetic energy, which is a function of blood flow velocity and lumen diameter (11), also has mean and oscillatory components, with the oscillatory component being larger than the mean. It has been shown in humans and dogs that the kinetic energy is a small percentage of the total energy (6,11,13). Therefore, kinetic energy was not included in the analysis.

Combined Left Ventricular/Arterial System Model

The models used in this study are identical to those described in Berger *et al.* (1). In brief, the arterial system model comprised a single uniform tube of length L terminating in a complex load, as shown in Fig. 1. This model was used because of its ability to reproduce the major features of $Z_{in}(\omega)$ while allowing its properties to be controlled independently (2,4). Pressure and flow in the tube were described by linear transmission equations with tube properties given by L', R', and C'; inductance, resistance, and compliance per unit length, respectively. The terminal load was a three-element Windkessel with characteristic impedance, Z_o, peripheral resistance, R_s, and compliance, C. The left ventricle was described as a time-varying compliance chamber with a given compliance function, $C_v(t)$ (Fig. 1). Solutions for P_{ao}, Q_{ao}, and P_v were approximated numerically (1).

This model affords the freedom to adjust independently various properties of the system, permitting the study of how changes in these properties directly affect *SW* and power dissipation. Specifically, because the tube is very nearly lossless ($R' \approx 0$), c_{ph} can be adjusted without affecting Z_o, through proportional changes in L' and C' (1). Changing c_{ph} in this manner also will not affect the magnitude of $\Gamma_G(\omega)$ ($|\Gamma_G|$) since for a lossless tube there is no attenuation and only the phase of $\Gamma_G(\omega)$ (ϕ_Γ) will be affected. In other words, alterations in c_{ph} will yield changes in $Z_{in}(\omega)$ such that Z_o and $|\Gamma_G|$ are unaffected, while changes in ϕ_Γ will simply reflect the change in arrival time of reflected waves. Finally, $\Gamma_G(\omega)$ can be adjusted without altering R_s, Z_o, or c_{ph}; $|\Gamma_G|$ is reduced by increasing C (*i.e.*, decreasing the magnitude of the reflection coefficient at the tube-load interface), and the system can be made reflection free by using a sufficiently large C (1).

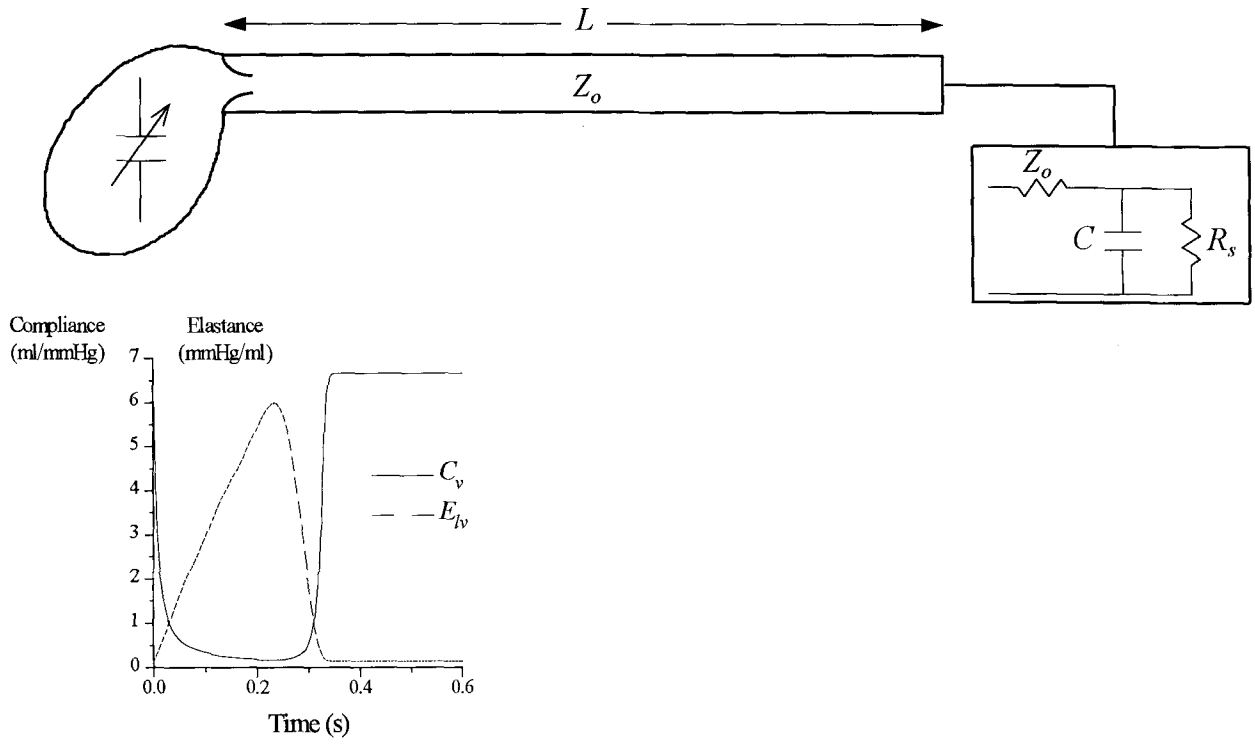

FIGURE 1. Coupled left ventricular/arterial system model. The left ventricle is a time-varying compliance (C_v) and the terminal load is modeled as a modified Windkessel. Connecting the two is a linear transmission tube model. The plots show C_v and its reciprocal, the time-varying elastance (E_{lv}).

METHODS AND RESULTS OF MODEL EXPERIMENTS

The results from computer experiments are analyzed in the present study. In all experiments, heart rate was 100 min^{-1} and model parameters represented those of the dog. C was adjusted from 0.1 through 10.0 ml/mm Hg, in various steps, to alter $\Gamma_G(\omega)$. To alter c_{ph}, L' and C' were altered proportionally over a wide range to maintain the same value for Z_o. The experiment with C = 0.3 ml/mm Hg and c_{ph} = 420 cm/sec was taken as "control." The reader is referred to Tables 1 and 2 in Berger *et al.* (1) for a complete listing of model parameter values. Note that the ranges of C and c_{ph} used (0.1–10.0 ml/mm Hg and 168–2100 cm/sec, respectively) were quite large, containing values outside the normal physiological range. These ranges were chosen so that the entire ranges of physiological and potential pathophysiological values of $\Gamma_G(\omega)$ and c_{ph} were included. We also wanted to investigate the reflectionless condition as a limiting case.

The effects of changes in C on the magnitudes and phases of $Z_{in}(\omega)$ ($|Z_{in}|$ and ϕ_{Zin}, respectively) and on $|\Gamma_G|$ can be found in (1). These effects include diminished $|\Gamma_G|$ for all harmonics, reduced oscillations of $|Z_{in}|$, and a flattened ϕ_{Zin} as C increases. Figure 2 contains $|Z_{in}|$ and ϕ_{Zin} for a wide range of c_{ph} showing reduced oscillation of both magnitude and phase with increasing c_{ph} (*i.e.*, becoming more Windkessel-like). In addition, the zero-crossing of ϕ_{Zin} is moved to higher frequencies with increasing c_{ph}, a classic indicator of increased wave velocity. Figure 3 shows $|\Gamma_G|$ for different c_{ph}, indicating that changes in wave velocity alone do not affect the reflection coefficient magnitude. In Berger *et al.* (1), *SW* and *SV* were determined for the full ranges of c_{ph} and $\Gamma_G(\omega)$. In the interest of facilitating comparison, those results are reproduced here together in Fig. 4. Notice in the upper panel that *SV* initially increases with increasing C (reduced $\Gamma_G[\omega]$) up to C = 0.5 ml/mm Hg, while *SW* increases over the entire range.

Figure 5 shows $\dot{W}_s$ and $\dot{W}_t$ in the top panel with $\dot{W}_o$ in the bottom panel, plotted against C. The behavior of $\dot{W}_t$ is the same as for *SW*, which is mandatory, increasing rapidly from C = 0.1 ml/mm Hg up to C = 0.5 ml/mm Hg, followed by a slowly increasing portion through C = 10.0 ml/mm Hg. On the other hand, $\dot{W}_s$ increases up to C = 1.0 ml/mm Hg and remains constant thereafter. Comparing the course of $\dot{W}_s$ to that of *SV* in Fig. 4, it is seen that their behaviors are similar. The plot in Fig. 6 shows $\%\dot{W}_o$ for the range of C The number next to each data point is the magnitude of the first (most significant) harmonic of $\Gamma_G(\omega)$, $\Gamma_{G,1}$. This graph demonstrates the clear effect of reduced reflection on $\%\dot{W}_o$: an increase in the oscillatory portion of $\dot{W}_t$. In Fig. 7, the magnitude of the flow harmonics, Q_{ao_t}, for the range of C tested are displayed, showing little effect at low frequencies. This will become im-

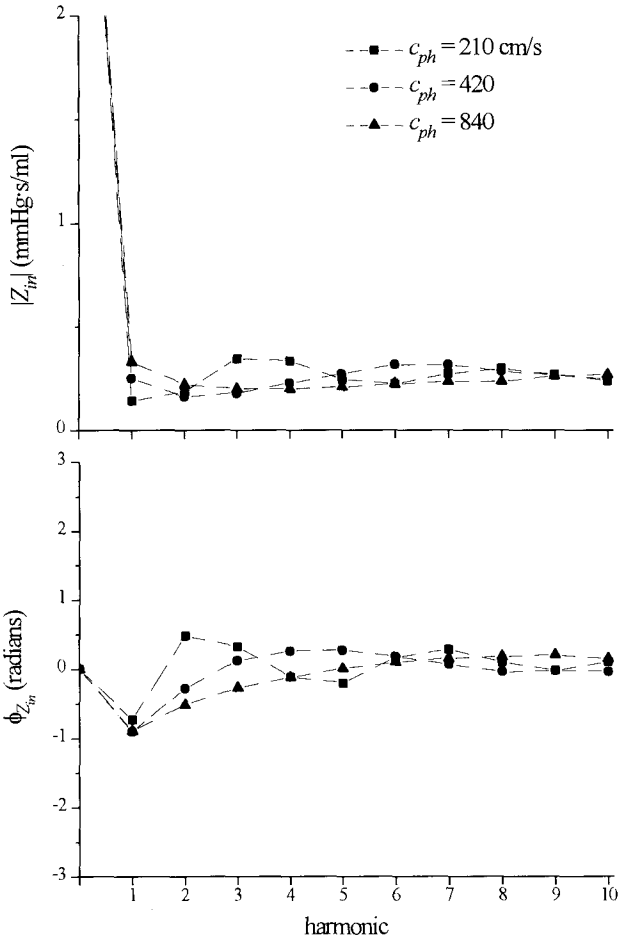

FIGURE 2. Magnitude [$|Z_{in}|$] and phase (ϕ_{Zin}) of input impedance for a wide range of c_{ph}.

portant when discussing the reasons for the changes in oscillatory power.

The effect of changes in c_{ph} on $\dot{W}_s$ and $\dot{W}_t$ are shown in the top panel of Fig. 8 $\dot{W}_s$ shows a steady decrease with increasing c_{ph}, reaching a plateau in the high velocity range. Because $\dot{W}_s$ is the major contributor to total power, it is no surprise that $\dot{W}_t$ shows a similar trend. In fact, the changes in $\dot{W}_s$ and $\dot{W}_t$ seem the same, indicating that changes in $\dot{W}_o$ are not large. This is confirmed in the bottom panel of Fig. 8. Notice that the scale for $\dot{W}_o$ is smaller than its counterpart in Fig. 5, meaning that $\dot{W}_o$ is less sensitive to changes in c_{ph} than to changes in $\Gamma_G(\omega)$. The plot of $\dot{W}_o$ against c_{ph} shows a biphasic response similar to that found in peak systolic pressure (1), showing minimum $\dot{W}_o$ near control c_{ph}. Figure 9 shows $\%\dot{W}_o$ as a function of c_{ph}. Again, its scale is smaller than that in Fig. 6 so that, like $\dot{W}_o$, $\%\dot{W}_o$ is less sensitive to c_{ph} than to $\Gamma_G(\omega)$. Not surprisingly, $\%\dot{W}_o$ also has a biphasic shape.

DISCUSSION

A relatively small percentage of the total energy used by the left ventricle is converted into mechanical work (7) (*i.e.*, energy associated with moving blood from the ventricle into the arterial system). This external mechanical energy is the stroke work. The notion that the left ventricle itself generates separate steady and oscillatory work, corresponding to $\dot{W}_s$ and $\dot{W}_o$ as defined earlier, is misleading for two reasons. First, direct coupling between the ventricle and the arterial system load only occurs during ejection, when the aortic valve is open. Because power dissipation takes place over the entire cardiac cycle, the ventricle is uncoupled from most of the power dissipation process. Second, the presence of the aortic valve, itself a large nonlinearity, means that energy, in the form of moving blood, must be delivered to the arterial system in an intermittent fashion.

It is perhaps more appropriate then to consider left ventricular external work simply as time-varying and to bear in mind that the physical process of power dissipation in the arterial system is not the same as generation of ventricular external work. In the dynamically coupled system, P_{ao} and Q_{ao} are fully determined by $C_v(t)$, V_{ed}, and $Z_{in}(\omega)$. Thus, the ventricle and arterial system together determine SW (and $\dot{W}_t$). One way to analyze arterial power dissipation is to separate $\dot{W}_t$ into $\dot{W}_s$ and $\dot{W}_o$, each having physiological relevance. Specifically, $\dot{W}_s$ and $\dot{W}_o$ correspond to energy dissipated in maintaining net forward flow and energy wasted in pulsations not contributing to net forward flow, respectively. A similar separation of external work generated by the ventricle, although mathematically feasible, does not have a physiological basis.

Berger *et al.* (1) argued that changes in $\Gamma_G(\omega)$ and/or c_{ph} appear to the left ventricle as equivalent changes in the arterial system input impedance. In other words, if one evokes a change in $\Gamma_G(\omega)$ and/or c_{ph}, a change in $Z_{in}(\omega)$ must also occur. As such, altering $\Gamma_G(\omega)$, for example, will yield changes in aortic pressure and flow through changes in ejection (altered arterial system hydraulic load)

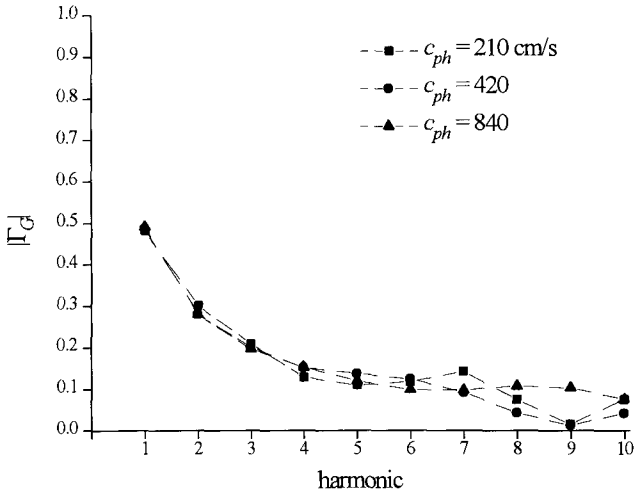

FIGURE 3. Magnitude of the global reflection coefficient [$|\Gamma_G|$] for a wide range of c_{ph}.

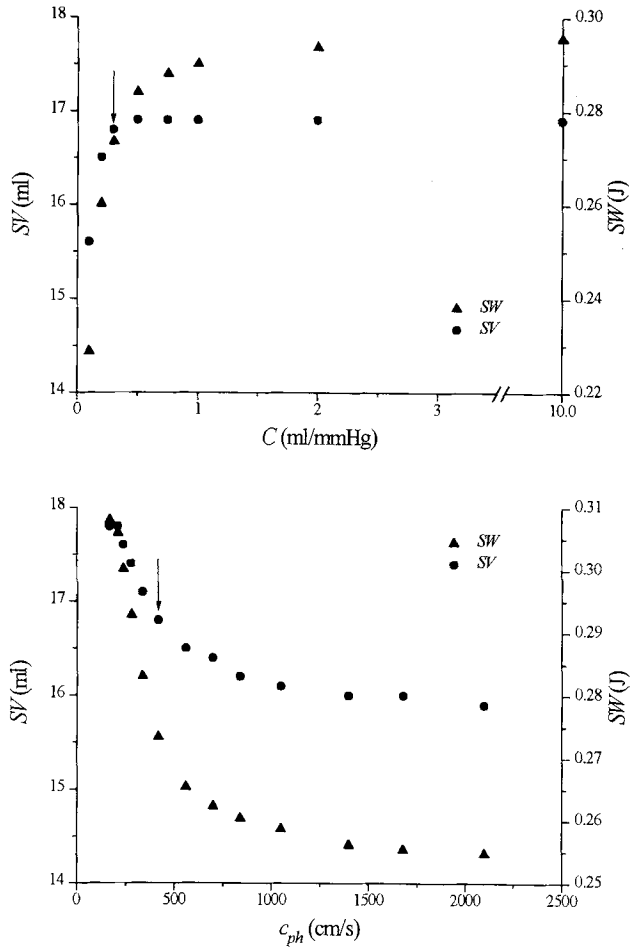

FIGURE 4. Left ventricular *SV* and *SW* for the range of *C* (top) and for the range of c_{ph} (bottom). The arrows indicate the control condition.

along with changes in reflected waves. Similarly, effects of changes in $\Gamma_G(\omega)$ and/or c_{ph} on *SW* and power dissipation also must be viewed as resulting from changes in ejection as well as changes in propagation and reflection.

Power Dissipation and Reflection Coefficient

The increase in $\dot{W}_t$ with reduced $\Gamma_G(\omega)$ (Fig. 5) is expected because reduced $\Gamma_G(\omega)$ also yields increased *SW* (Fig. 4). The shapes of the $\dot{W}_t$ and *SW* curves are also the same, rising rapidly at high Γ_G (low C) values, with a more gradual increase for $C > 0.5$ ml/mm Hg. When $\dot{W}_t$ is separated in to $\dot{W}_s$ and $\dot{W}_o$, differences appear. $\dot{W}_s$ shows the same rapid increase at high $\Gamma_G(\omega)$ values but remains constant for $C \geq 0.5$ ml/mm Hg. $\dot{W}_o$, on the other hand, shows a continued increase for the entire range of decreasing $\Gamma_G(\omega)$. This means that as $\Gamma_G(\omega)$ decreases, all increases in *SW* are eventually dissipated as $\dot{W}_o$; all increases in *SW* for $C \geq 0.5$ ml/mm Hg are wasted. The values for $\dot{W}_s$, $\dot{W}_o$, $\dot{W}_t$, and $\%\dot{W}_o$ in the control condition are well within the normal ranges found in dogs (6,11).

That $\dot{W}_s$ eventually reached a constant value with decreasing $\Gamma_G(\omega)$ is consistent with both *SV* (Fig. 4) and $\bar{P}_{ao}$ eventually reaching constant values (1). As a result, increased *SW* with no increase in *SV*, with decreasing $\Gamma_G(\omega)$, manifests itself as increased $\dot{W}_o$. Inspection of Eq. 3b provides a clue as to why $\dot{W}_o$ increases with decreasing $\Gamma_G(\omega)$. The three terms in the summation may or may not change depending on the changes made to the arterial system model. For altered $\Gamma_G(\omega)$, only *C* was altered so Z_o remained the same. As the system becomes more reflection free, its input impedance will approach that of a tube of infinite length while pressure and flow become more in phase (*i.e.*, they become more similar in shape). This results in $\phi_i \rightarrow 0$ and $Z_{in}(\omega_i) \rightarrow Z_o$ for all *i* as $|\Gamma_G| \rightarrow 0$. These phenomena are clearly demonstrated in Berger *et al.* (1). Therefore, the $\cos\phi_i$ term becomes larger (approaches 1) for all ϕ_i as $|\Gamma_G| \rightarrow 0$. This is especially important at lower harmonics where Q_{ao_i} are larger and phase is normally negative. The plots in Fig. 7 reveal that Q_{ao_i} are little affected by changes in *C*, especially at lower harmonics where most of the energy is located. Because the differences in $Z_{in}(\omega_i)$ are small and bidirectional, most of the increase in $\dot{W}_o$ is due to the reduction of phase between P_{ao} and Q_{ao}. Thus the interpretation is clear: in

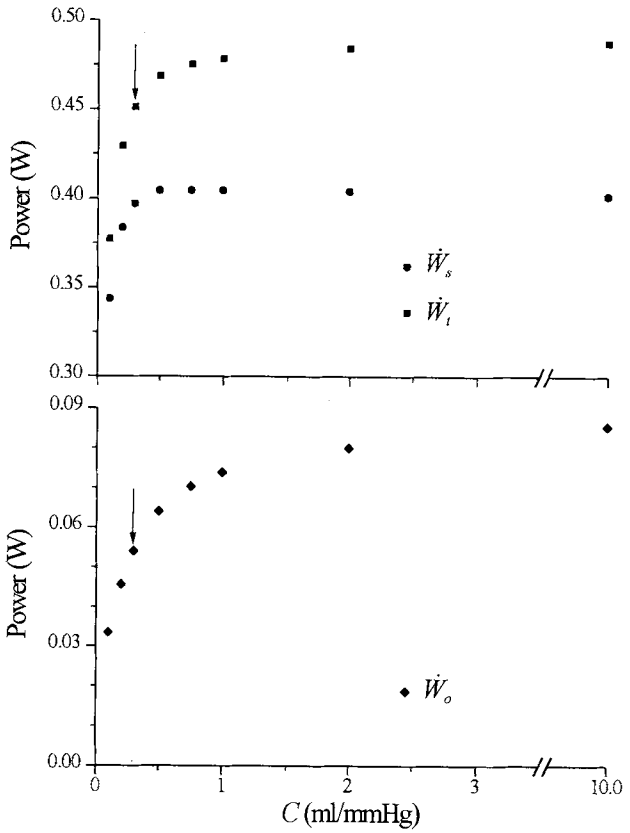

FIGURE 5. $\dot{W}_s$ and $\dot{W}_t$ in the top graph and $\dot{W}_o$ in the bottom for the range of *C*. The arrows indicate the control condition. Note the different vertical scales.

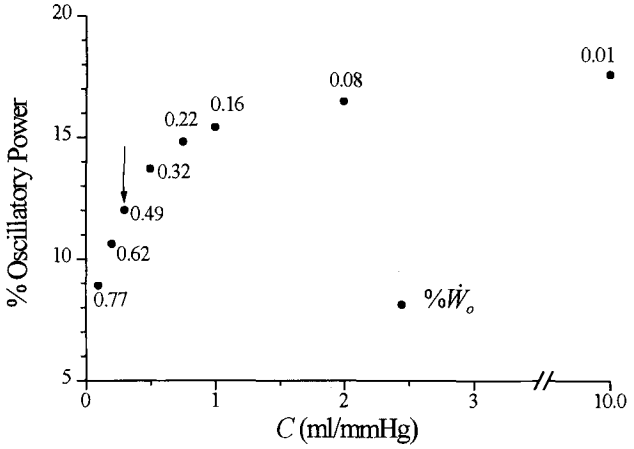

FIGURE 6. $\%\dot{W}_o$ for the range of C showing an increase in the oscillatory portion with reduced reflections. The numbers next to the data points are magnitudes of the first harmonic of $\Gamma_G(\omega)$. The arrow indicates the control condition.

the arterial system with finite pulse wave velocity, the presence of wave reflections leads directly to a reduction of oscillatory power and an increase in the efficiency of power dissipation. These changes are brought about primarily through the introduction of phase differences between pressure and flow with little change in mean values.

Comparisons with *in vivo* experimental studies cannot be made at this time because changes in $\Gamma_G(\omega)$ independent of all else have yet to be accomplished in the intact circulation. Investigations of power dissipation with altered arterial system load have been done, but none have focused on the reflection coefficient *per se*. O'Rourke (15) studied power dissipation in the presence of increased aortic stiffness; however, as discussed in the next section, that study is best compared to altered c_{ph}.

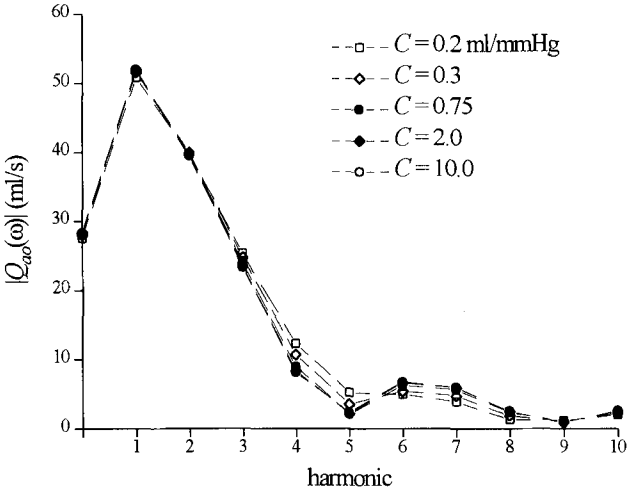

FIGURE 7. Magnitude of flow harmonics, $Q_{ao,i}$, for the range of C.

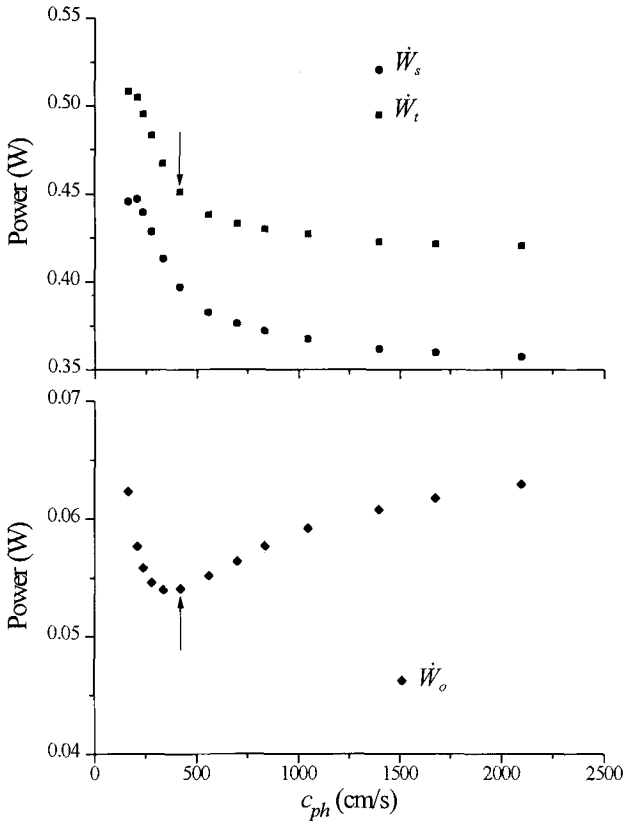

FIGURE 8. $\dot{W}_s$ and $\dot{W}_t$ in the top graph and $\dot{W}_o$ in the bottom for the range of c_{ph}. The arrows indicate the control condition.

Power Dissipation and Pulse Wave Velocity

$\dot{W}_t$ and $\dot{W}_s$ reductions with increasing c_{ph} (Fig. 8) parallel the changes in *SW* and *SV*, respectively, (bottom panel in Fig. 4). The response of $\dot{W}_o$ to changes in c_{ph} is biphasic, similar to the effect of c_{ph} on peak systolic pres-

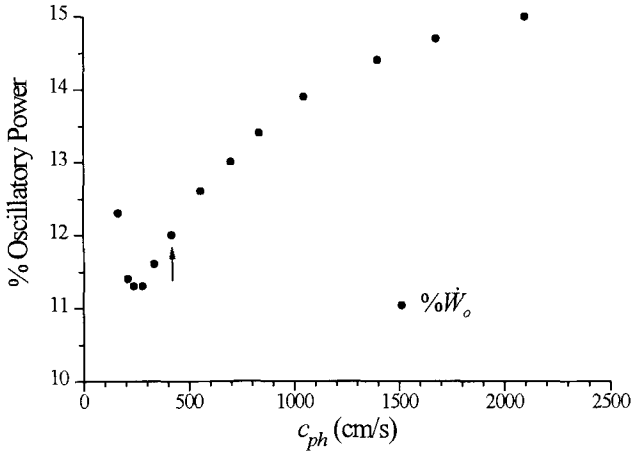

FIGURE 9. $\%\dot{W}_o$ for the range of c_{ph} showing the biphasic response in the oscillatory portion with increased propagation velocity. The arrow indicates the control condition.

sure (1), which can be explained as the system appearing more reflectionless as c_{ph} becomes very small. Two important differences reveal themselves when comparing Fig. 5 with Fig. 7. First, the sensitivity of $\dot{W}_o$ to changes in c_{ph} is much less than its sensitivity to changes in $\Gamma_G(\omega)$. The same is true for $\dot{W}_s$. Second, for $c_{ph} > 336$ cm/sec, changes in $\dot{W}_o$ and $\dot{W}_s$ are opposite in direction. The increase in $\dot{W}_o$ is small compared with the decrease in $\dot{W}_s$ as c_{ph} increases, therefore, $\dot{W}_t$ follows $\dot{W}_s$.

Given the behavior of $\dot{W}_s$ and $\dot{W}_o$ as c_{ph} increases, the increase in $\%\dot{W}_o$ seen in Fig. 9 is expected. Note that while $\%\dot{W}_o$ is biphasic, the inflection point is shifted to the left compared with $\dot{W}_o$. Therefore, $\%\dot{W}_o$ does not begin to increase with decreasing c_{ph} until very low velocities. This leads to the prediction that, for the range of c_{ph} likely to be encountered, reduction of c_{ph} will yield increased arterial system efficiency. Because changes in c_{ph} directly affect only the delay time of the reflected waves (Figs. 2 and 3), $|\Gamma_G|$ was not a factor in changes of $\dot{W}_o$ and $\%\dot{W}_o$ due to changes in c_{ph}. Keeping in mind the discussion in Berger *et al.* (1) regarding extrapolating these results to the intact animal, one can compare the model experiments of altered c_{ph} with animal experiments in which large artery distensibility is impaired. Several investigators have succeeded, using different approaches, in altering "aortic" stiffness in the intact animal. O'Rourke (15) studied simulated large artery disease, in a dog model, by stiffening the aorta and brachiocephalic arteries through application of plaster and/or Lucite ferrules. Urschel *et al.* (19) bypassed the descending aorta of the dog with a glass tube while Randall *et al.* (16) placed a stiff tube within the aorta. Recently, Kelly *et al.* (8) successfully bypassed the entire aorta with a stiff Tygon tube in an *in situ* dog preparation. While all of these preparations yielded significant reduction in "aortic" compliance, only O'Rourke (15) included analysis of arterial system power dissipation.

Because O'Rourke's treatment was applied only to large arteries, peripheral resistance was little affected, while aortic characteristic impedance was increased and reflected waves returned earlier. After the application, $\%\dot{W}_o$ showed a marked increase over control. The model experiments for altered c_{ph} are comparable to those of O'Rourke (15) because the increase c_{ph} was achieved by reducing C', similar to reducing compliance of the large proximal arteries. The primary arterial system difference is that Z_o was constant in the present study while in O'Rourke (15) it was not. Nevertheless, $\%\dot{W}_o$ as a function of c_{ph} (Fig. 9) compare well qualitatively with O'Rourke (15), although the magnitudes of the changes were generally not as great. We showed $\%\dot{W}_o$ increasing from 11.5% to 15% while O'Rourke (15) reported $\%\dot{W}_o$ increases typically two to three times as large. This indicates that some of the diminished arterial system efficiency demonstrated by O'Rourke (15), in terms of wave propagation phenomena, was due directly to increased c_{ph}. Other factors possibly contributing to $\%\dot{W}_o$ changes in the dog experiments include alterations in characteristic impedance, preload, heart rate, and ejection time (6,14,15).

Arterial System Efficiency and LV Performance

Quantifying arterial system efficiency with $\%\dot{W}_o$ makes intuitive sense given that $\dot{W}_s$ is related to the net movement of blood into the periphery while $\dot{W}_o$ is not. Berger *et al.* (1) make a passing reference to the ratio *SV/SW* and argue that changes in this ratio can be used in the model experiments as an index of efficiency. This idea comes directly from the *SV* and *SW* results in Fig. 4 (top panel) which shows increased *SW* with no increase in *SV* as $\Gamma_G(\omega)$ decreases. Therefore, beyond the point of increasing *SV*, none of the additional external work was devoted to maintaining the steady flow and hence was wasted. Two caveats must be observed when analyzing the *SV/SW* ratio. One is that this ratio does not really represent an efficiency, since it is not dimensionless. That is, it is not the ratio of some output quantity to input quantity of the same dimension. Because of this, the quantity *SV/SW*, standing alone, does not have clear meaning (it is actually the reciprocal of mean P_v). Of equal importance is the nature of the model experiments; model experimental conditions were under extreme control. This precise control can be used to advantage by looking not at a single *SV/SW*, but tracking its changes as the load changes in the special case of constant *SV* with changing *SW*. Doing so reveals the relationship between the steady-state power dissipation in the arterial system and the steady-state performance of the ventricle.

The consequences of $\dot{W}_o$ and changes in $\%\dot{W}_o$ on the ventricle arise from interaction with the arterial system. From the vantage point of the ventricle, as stated earlier, a given amount of external work is generated and delivered to the arterial system. From the vantage point of the arterial system, this work is dissipated in maintaining steady flow and in pulsations, while the (in)efficiency of power dissipation is largely a description of the arterial system. However, when viewed as a complete arterioventricular system, increases in $\%\dot{W}_o$ are always imposed on the ventricle which, in the case of decreased reflection, manifests itself clearly as increased *SW* with no increase in *SV*. So, while $\%\dot{W}_o$ and changes in *SV/SW* do not represent actual mechanical efficiency of the left ventricle alone (*i.e.*, they do not consider myocardial oxygen consumption), they can be viewed as an indicator of coupled system efficiency.

Distributed Compliance

Thus far, the discussion of the arterial system load has been in terms of pulse wave velocity and reflections coefficient. Alternatively, one can view the changes in c_{ph} and $\Gamma_G(\omega)$ as changes in compliance (1). For changes in

c_{ph}, tube or proximal compliance is altered, whereas for changes in $\Gamma_G(\omega)$, peripheral of distal compliance is altered. If all compliances were added, resulting in a total compliance of the system, C_a, a given change in C_a might result in different changes in power dissipation and efficiency depending on where the change in compliance occurred. For example, an increase in C or C' will result in increased C_a although they will have opposite effects on $\%\dot{W}_o$. Therefore, an increase in C_a might not necessarily indicate a more efficient system. Since the effects of blood pressure reducing medication on the changes in distributed compliance are not known, the results of the model experiments may explain, in part, the inconsistent effects of antihypertensive treatment on arterial efficiency (5,10, 14,18).

The statements made above apparently contradict one of the classical ideas of hemodynamics, that being the general goodness of arterial compliance. In particular, in terms of arterial system efficiency, we find that while increasing the tube compliance (reduced c_{ph}) is beneficial, increasing the terminal compliance (reducing $|\Gamma_G|$) of the model is not. To the extent that tube models and their parameters adequately represent arterial system properties and input impedance, and there is mounting evidence that this is the case (2–4,17), one might infer from these results that an increase in total arterial compliance may not be desirable. While giving full attention to this problem is outside the current scope, one must remember that total arterial compliance is normally evaluated as a lumped quantity, especially in the clinic, and its value is typical determined from P_{ao} and Q_{ao} using diastolic pressure decay for example. Such a viewpoint is consistent with the Windkessel, a fully reduced model wherein wave velocity is infinite, rendering spatial properties immaterial. However, the compliance of the arterial system *is* spatially distributed and the effects of this distribution on total compliance and arterial load have yet to be studied (9). When thought of in terms of compliance, as opposed to $\Gamma_G(\omega)$ and c_{ph}, the results of this study simply suggest that the way compliance is distributed may affect P_{ao}, Q_{ao}, SW, and power dissipation differently, depending on c_{ph} and the distances involved. It is also possible that the value of lumped compliance determined from P_{ao} and Q_{ao} waveforms may not accurately reflect the sum of all compliances in the distributed system. This aspect of arterial hemodynamics requires further attention.

REFERENCES

1. Berger, D. S., J. K-J. Li, and A. Noordergraaf. Differential effects of wave reflections and peripheral resistance on aortic blood pressure: a model based study. *Am. J. Physiol.* 266(*Heart Circ. Physiol.* 35):H1626–H1642, 1994.
2. Berger, D. S., J. K-J. Li, W. K. Laskey, and A. Noordergraaf. Repeated reflection of waves in the systemic arterial system. *Am. J. Physiol.* 264(*Heart Circ. Physiol.* 33): H269–H281, 1993.
3. Burattini, R., and K. B. Campbell. Effective distributed compliance of the canine descending aorta estimated by modified T-tube model. *Am. J. Physiol.* 264(*Heart Circ. Physiol.* 33):H1977–H1987, 1993.
4. Campbell, K. B., L. C. Lee, H. F. Frasch, and A. Noordergraaf. Pulse reflection sites and effective length of the arterial system. *Am. J. Physiol.* 256(*Heart Circ. Physiol.* 25):H1684–H1689, 1989.
5. Cholley, B. P., S. G. Shroff, J. Sandelski, C. Korcarz, B. A. Balasia, S. Jain, D. S. Berger, M. B. Murphy, R. H. Marcus, and R. M. Lang. Differential effects of chronic oral antihypertensive therapies on systemic arterial circulation and ventricular energetics in African American patients. *Circulation* 91:1052–1062, 1995.
6. Cox, R. H. Determination of systemic hydraulic power in unanesthetized dogs. *Am. J. Physiol.* 226:579–587, 1974.
7. Gibbs, C. L. Cardiac energetics. *Physiol. Rev.* 58:175–254, 1978.
8. Kelly, R. P., R. Tunin, and D. A. Kass. Effect of reduced aortic compliance on cardiac efficiency and contractile function of *in situ* canine left ventricle. *Circ. Res.* 71:490–502, 1992.
9. Laskey, W. K., H. G. Parker, V. A. Ferrari, W. G. Kussmaul, and A. Noordergraaf. Estimation of total arterial compliance in man. *J. Appl. Physiol.* 69:112–119, 1990.
10. Merillon, J. P., G. J. Fontenier, J. F. Lerallut, M. Y. Jaffrin, G. A. Motte, C. P. Genain, and R. R. Gourgon. Aortic input impedance in normal man and arterial hypertension: its modification during changes in aortic pressure. *Cardiovasc. Res.* 16:646–656, 1982.
11. Milnor, W. R. Cardiac Dynamics. In: *Hemodynamics*. Baltimore: Williams & Wilkins, pp. 260–293, 1989.
12. Milnor, W. R., C. R. Conti, K. B. Lewis, and M. F. O'Rourke. Pulmonary arterial pulse wave velocity and impedance in man. *Circ. Res.* 25:637–649, 1969.
13. Milnor, W. R., D. H. Bergel, and J. D. Bargainer. Hydraulic power associated with pulmonary blood flow and its relation to heart rate. *Circ. Res.* 19:467–480, 1966.
14. Nichols, W. W., C. R. Conti, W. E. Walker, and W. R. Milnor. Input impedance of the systemic circulation in man. *Circ. Res.* 40:451–458, 1977.
15. O'Rourke, M. F. Steady and pulsatile energy losses in the systemic circulation under normal conditions and in simulated arterial disease. *Cardiovasc. Res.* 1:313–326, 1967.
16. Randall, O. S., G. C. Van den Bos, and N. Westerhof. Systemic compliance: does it play a role in the genesis of essential hypertension? *Cardiovasc. Res.* 18:455–462, 1984.
17. Shroff, S. G., D. S. Berger, R. M. Lang, R. H. Marcus, C. Korarcz, and D. E. Miller. Physiological relevance of T-tube model parameters: emphasis on arterial system compliance. *Am. J. Physiol.* 269(*Heart Circ. Physiol.* 38): H365–H374, 1995.
18. Ting, C. T., K. P. Brin, S. J. Lin, S. P. Wang, M. S. Chang, B. N. Chiang, and F. C. P. Yin. Arterial hemodynamics in human hypertension. *J. Clin. Invest.* 78:1462–1471, 1986.
19. Urschel, C. W., J. W. Covell, E. H. Sonnenblick, J. Ross, Jr., and E. Braunwald. Effects of decreased aortic compliance on performance of the left ventricle. *Am. J. Physiol.* 214:298–304, 1968.
20. Westerhof, N., P. Sipkema, G. C. Van den Bos, and G. Elzinga. Forward and backward waves in the arterial system. *Cardiovasc. Res.* 6:648–656, 1972.