For as long as cardiovascular physiologists have been able to measure pressure and flow in the ascending aorta, they have puzzled over the obvious differences in shape between the two waveforms. One way of explaining the measured differences in pressure and flow waveforms in the ascending aorta would be the existence of reflected waves because, although the changes in pressure and flow in the reflected waves are also proportional to each other, the sign of the constant of proportionality is reversed. Thus a reflected wave that reinforces the pressure change caused by the forward wave will have a canceling effect on the flow. However, despite many attempts, it has never been demonstrated that the effects of wave reflection are sufficient to explain the large, qualitative differences in the aortic pressure \((P_{Ao})\) and flow waveforms (24), particularly during diastole when \(P_{Ao}\) declines incrementally from a high level and aortic flow remains essentially zero. Indeed, Milnor (20) remarked that the properties of the aortic tree in the normal young animal are those of an almost perfect diffuser \((i.e., \text{it generates far fewer reflections than the best man-made distribution network})\). Many other suggestions for the resolution of this paradox have been advanced, but none have found general acceptance. \([\text{See both Milnor (20) and Nichols and O'Rourke (22) for exhaustive but inconclusive discussions of this problem.}]\)

In contrast to the prevailing frequency-domain concepts, according to which \(P_{Ao}\) and flow waveforms are separated into mean and oscillatory components \((2, 12, 20, 24, 28)\), we propose a new time-domain approach based on the two major functional properties of the arterial system, properties that describe the reservoir function and those that describe the wave-transmitting function. The reservoir properties of the system are those by which blood and potential energy are stored, to be subsequently expended in peripheral perfusion \((41, 42)\). In this regard, the aorta can be considered to be a zero-dimensional system \((33)\), in which pressure and volume changes are functions of time only. During systole, the ejected stroke volume increases the reservoir volume and thus the reservoir pressure. During diastole, outflow exceeds inflow so reservoir volume decreases, and this is the simple explanation for the decrease in pressure. This reservoir mechanism has been simulated using the hydraulic-integrator windkessel model. The wave-transmitting properties of the system are those by which forward- and backward-traveling pressure-velocity waves are supported \((23, 25, 43)\). In this regard, the aorta can be considered to be a one-dimensional system \((25)\) in which pressure and velocity are functions of distance as well as time. The aorta may be analogous to a canal lock, upon whose surface waves can propagate \([\text{somewhat similar to the classical soliton (30–32, 49)}]\) and whose hydrostatic pressure is a simple function of the water level.

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Following Frank (33), we consider the windkessel to be a hydraulic integrator whose change in pressure \( \Delta P_{\text{wk}} \) is directly related to its change in volume \( \Delta V_{\text{wk}} \) and, inversely, to its compliance. [The rate of change of volume \( V_{\text{wk}} \) is simply the difference between the inflow from the left ventricle \( Q_{\text{in}} \) and the outflow to the periphery \( Q_{\text{out}} \).] In considering wave propagation on a reservoir, Lighthill (15) proposed that the measured pressure was the sum of the reservoir pressure and the reservoir due to wave motion, which he called “excess pressure” \( P_{\text{ex}} \). Assuming that the windkessel can be considered as a quasisteady reservoir with resistance of the peripheral systemic circulation. 

Our findings suggest that the hemodynamics of aortic ejection must be reinterpreted, and, with this understanding of the windkessel as a hydraulic integrator, some of the discrepancies between theoretical models and experimental observations might be resolved.

**METHODS**

**Theory**

We propose that \( P_{\text{ao}} \) be represented as the sum of a time-varying reservoir pressure (independent of distance) and a \( P_{\text{ex}} \) that varies in time \( t \) and with distance along the arteries \( x \): \( P_{\text{ao}}(x,t) = P_{\text{wk}}(t) + P_{\text{ex}}(x,t) \).

Windkessel theory. The variation of aortic \( P_{\text{wk}} \) is determined by the difference between inflow and outflow and the change in volume (33)

\[
\frac{dP_{\text{wk}}(t)}{dt} = \frac{dP_{\text{wk}}}{dt} \frac{dV_{\text{wk}}}{dt} = \frac{Q_{\text{in}}(t) - Q_{\text{out}}(t)}{C} \tag{1}
\]

where \( C = \frac{dV_{\text{wk}}}{dP_{\text{wk}}} \) and is the compliance of the whole arterial tree (37) and assumed to be constant. If the outflow can be described by a simple resistive relationship, \( Q_{\text{out}}(t) = \left[ P_{\text{wk}}(t) - P_{\text{ex}} \right]/R \) (where the outflow is assumed to be driven by the difference between \( P_{\text{wk}} \) and the asymptotic pressure of the diastolic exponential decay \( P_{\text{ex}} \) (34)), at which flow from the arteries to the veins ceases. So defined, \( R \) is the effective resistance of the peripheral systemic circulation.

Substituting \( Q_{\text{out}} \) in terms of \( P_{\text{wk}} \) and \( P_{\text{ex}} \), Eq. 1 can be rewritten in terms of \( P_{\text{wk}} \)

\[
\frac{dP_{\text{wk}}(t)}{dt} + \frac{P_{\text{wk}}(t) - P_{\text{ex}}}{RC} = \frac{Q_{\text{in}}(t)}{C} \tag{2}
\]

and the general solution is

\[
P_{\text{wk}}(t) - P_{\text{ex}} = (P_0 - P_{\text{ex}}) e^{-t/\tau} + e^{-t/\tau} \int_{t_0}^{t} \frac{Q_{\text{in}}(t')}{C} e^{\sigma t'} dt' \tag{3}
\]

in which \( t_0 \) and \( P_0 \) are the time and pressure at the onset of ejection. During diastole, when \( Q_{\text{in}} = 0 \), the solution is a simple exponential, falling with the time constant \( \tau = RC \).

To solve Eq. 3, \( R \), \( C \), and \( P_0 \) have to be determined using experimental data. It is believed that waves are minimal during approximately the last two-thirds of diastole (38, 48). At this time, \( P_0 \) and \( \tau \) can then be determined by fitting the late-diastolic \( P_{\text{ao}} \) data by Eq. 3.

Several methods have been proposed for the fitting of a windkessel to measured arterial pressure (38, 48). We adopted an alternative approach where the parameters \( R \), \( C \), and \( P_0 \), which determine \( P_{\text{wk}} \), are calculated iteratively using a nonlinear search algorithm to minimize the mean square error over the last two-thirds of diastole. The minimization is done using the Matlab routine “fminsearch,” which uses the Nelder-Mead simplex (direct search) method. As initial estimates, we used \( C = 0.5 \text{ml/mmHg} \), \( P_0 = 30 \text{mmHg} \), and \( R \) calculated from the mean of the measured pressure and flow rate over the cardiac cycle. Although up to 20 iterations may be required for convergence, the method is robust.

Wave theory. Arterial wave theory is based on one-dimensional flow in an elastic tube (25–27). If frictional loss is neglected, wave equations can be derived from the conservation of mass and momentum in terms of the cross-sectional area \( A \) and velocity \( U \) (15, 46)

\[
A_1 + (UA)_x = 0 \tag{4}
\]

\[
U_t + AU_x + \frac{P_0}{\rho} = 0 \tag{5}
\]

where \( \rho \) is the density of blood.

Lighthill (15) states that a wave is a propagated disturbance and that it is driven by \( P_{\text{ex}} \), which he defined as the difference between the measured pressure and the undisturbed reservoir pressure. Because the aortic windkessel functions as a reservoir and its rate of pressure change is low relative to the pressure changes associated with the propagation of waves, we appropriated and modified Lighthill’s concept and here define \( P_{\text{ex}} \) as the difference between \( P_{\text{ao}} \) and \( P_{\text{wk}} \), i.e., \( P_{\text{ex}} = P_{\text{ao}} - P_{\text{wk}} \). The cross-sectional area \( A \) for the wave equations is described as a function of \( P_{\text{ex}} \), i.e., \( A = f(P_{\text{ex}}) \). The mass and momentum equations can be written in terms of \( P_{\text{ex}} \) and \( U \). Equation 4 can then be described as

\[
(P_{\text{ex}})_t + U(P_{\text{ex}})_x + \rho c^2 U_x = -\frac{dP_{\text{wk}}}{dt} \tag{6}
\]

where \( c \) is the wave speed \( |c| = v/\sqrt{\rho D} \), where \( D \) is the distensibility of the artery \( (D = 1/A \times dA/dP_{\text{ex}}) \). These equations are hyperbolic, and a general solution can be obtained using the method of characteristics. The arguments leading to the solution are subtle and somewhat difficult, but the solution is surprisingly simple. Any disturbance introduced into the artery will produce wavefronts that travel forward and backward with speeds of \( c \pm U \). The changes in pressure and velocity across these wavefronts are related by

\[
\frac{dP_{\text{ex}}}{dt} \pm c \rho dU_x = 0 \tag{6}
\]

where + refers to forward-traveling waves and − refers to backward-traveling waves. They can also be described in terms of the volume flow rate, \( dP_{\text{ex}}/dt = \pm c \rho A \times dQ_x \), which is the water hammer equation. \( c \rho A \) is the characteristic impedance, which was defined as the pressure-to-flow ratio of a forward-traveling wave (20).

**Experimental Preparation and Protocol**

Studies were performed on 15 healthy mongrel dogs weighing between 18 and 29 kg. Dogs were anesthetized by
thiopental sodium (20 mg/kg), followed by fentanyl citrate (30 μg·kg⁻¹·h⁻¹) and ventilated with a 1:1 nitrous oxide-oxygen mixture. The rate of a constant-volume respirator (tidal volume = 15 ml/kg, model 607, Harvard Apparatus; Natick, MA) was adjusted to maintain normal blood gas tensions and pH. Body temperature was maintained at 37°C using a circulating water warming blanket and a heating lamp. Lactated Ringer solution was infused through the jugular vein to maintain mean aortic blood pressure >80 mmHg. To enable us to equate thoracic aortic outflows to the aortic inflow, all the intercostal arteries were occluded using surgical clips. The spontaneous heart rates varied; the hearts were paced as slowly as possible (model S88, Grass Instruments; Quincy, MA).

We measured pressures in the left ventricle and at the aortic root using high-fidelity catheter-tip manometers (Millar Instruments; Houston, TX) and flows at the aortic root at the sources of the brachiocephalic artery, left subclavian artery, and aorta at the diaphragm using ultrasonic flow probes (Transonic Systems; Ithaca, NY). The left ventricular pressure catheter was introduced through the apex. Aortic root pressure was measured by introducing a micromanometer (2-Fr) inserted from the right subclavian artery retrograde through the brachiocephalic artery into the root of the aorta, ~1.5 cm from the valve. The aortic root manometer was positioned within 1 cm of the aortic root flow probe. Diameters were measured at the aortic root and diaphragm, located within 1 cm of the respective flow probes, using pairs of ultrasonic crystals (Sonometrics; London, Ontario, Canada). (From these diameters, the volume of the thoracic aorta was calculated, assuming that it was shaped as a truncated cone.) After control recordings were taken, in 12 dogs, a counterpulsation balloon was introduced into the abdominal aorta just proximal to the aortoiliac bifurcation, which could be rapidly inflated and deflated to produce backward-traveling compression and expansion waves at any time during the cardiac cycle. With the use of a two-channel laboratory stimulator with variable and independent delay controls (model S88, Grass Instruments), we both stimulated the heart and triggered the counterpulsation pump such that the backward-traveling waves were made to arrive at the left ventricle at any chosen time during systolic ejection. The backward waves could be identified by superimposing the pressure and flow waveforms of the balloon-inflated beat over those of the immediately preceding control beat.

In four paced dogs, we measured left ventricular pressure (through the apex) and $P_{Ao}$ (through the right subclavian with a micromanometer (2-Fr)), and a third manometer was inserted from the femoral artery and advanced retrogradely to the aortic root. Flow at the aortic root, $Q_{Ao}$, was also recorded. The third manometer was pulled back by 2-cm increments to the femoral artery, pressures being recorded at each position with the ventilator turned off at end-expiration. Three-dimensional plots of $P_{Ao}$ versus time and distance from the aortic valve were constructed, taking the aortic root pressure as a temporal reference.

**RESULTS**

Figure 1A shows typical measured left ventricular pressure and $P_{Ao}$, calculated $P_{Wk}$, and $P_e$. $P_{Wk}$ begins to increase 30–50 ms after $P_{Ao}$, when aortic inflow exceeds outflow, and it continues to increase until inflow decreases to equal outflow (note the vertical dashed lines). During the latter part of diastole, $P_{Wk}$ approximates $P_{Ao}$ very closely because we curve fit this segment in our determination of the windkessel parameters. The difference between $P_{Ao}$ and $P_{Wk}$ is $P_e$, which is plotted with the measured aortic inflow $Q_{in}$ in Fig. 1B. The P and Q scales were adjusted to show that the two waveforms are almost identical in shape, which indicates that the effects of backward-traveling waves are minimal under these conditions. Figure 1C shows the intensity (i.e., normalized power) of forward- and backward-traveling waves (40). The forward-traveling compression wave, which defines the power required to accelerate the stroke volume, is the prototypical example of a “wave” as defined in this paper.

Figure 2 is a plot of $P_e$ versus $Q_{in}$, with the solid line suggesting that the left ventricular stroke volume is injected into the windkessel by a mechanism that is effectively resistive. The slight deviations from the straight line are compatible with an inertial mechanism. Close analysis of the differences shows that $P_e$ is slightly above the line during periods when $Q_{in}$ is increasing (accelerating) and below it when $Q_{in}$ is decreasing (decelerating). The slope of the line is not different from characteristic impedance (data not shown).

Figure 3A shows the pressure waveforms measured every 2 cm from the aortic root to the femoral artery. The propagation of the pressure pulse and the modification of its shape as it propagates distally are clear. During late diastole, pressure decreases almost uniformly from the aortic root to the femoral artery, and there is no evidence of waves. This is shown even more clearly in Fig. 3B, which is an isobar contour plot of the same data. During systole and early diastole, the slope of the contours indicate the wave speed. During late diastole, when wave motion is negligible, there are no measurable differences in pressure throughout the length of the aorta, indicating that pressure is a function of time only, not of distance.

In Fig. 4, we compared $P_{Wk}$ and $\Delta V_{Wk}$ to two independent estimates of proximal aortic volume. Figure 4A shows the flow rate measured at the aortic root (i.e., the inflow) and that measured in the aorta at the level of the diaphragm, in the brachiocephalic artery, and in the left subclavian artery (i.e., the outflows). Figure 4B shows the volumes obtained by integrating the measured inflow ($V_{in}$) and outflows ($V_{out}$). The hatched area, $V_{in} - V_{out}$, represents the instantaneous thoracic aortic volume. To determine whether the change in $P_{Wk}$ was proportional to the change in thoracic aortic volume (Eq. 1), we plotted $P_{Wk}$ (arbitrarily scaled) and our two estimates of thoracic aortic volume [i.e., the difference in integrated inflow and outflow and the calculated volume of the truncated cone (method detailed later)] during one cardiac cycle (Fig. 4C). Finally, to determine how much of the total $V_{Wk}$ was contained within the thoracic aorta, we compared $\Delta V_{Wk}$ ($\Delta V_{Wk} = \Delta P_{Wk} \times C$) to the difference between inflow and outflow (Fig. 4D); we found that 45.1 ± 2.0% of the total $V_{Wk}$ was contained in the aorta above the diaphragm (Table 1).

These results suggest that, under normal experimental conditions, the waveform of $P_e$ is very similar in shape to the waveform of flow at the root of the
This suggests that reflected waves do not have a significant effect on the left ventricle. To explore the effects of reflected waves, we produced backward-traveling waves using a counterpulsation balloon inserted in the abdominal aorta. The inflation and deflation of the balloon was timed so that their effects would be manifest at the aortic valve during ejection. Figure 5A shows \( P_{\text{ex}} \) and \( Q_{\text{in}} \) in a normal beat scaled to show the similarity of the two waveforms. Figure 5B shows \( P_{\text{ex}} \) and \( Q_{\text{in}} \) measured during the succeeding beat, when the balloon was inflated and deflated. The arrival of the backward compression wave (due to balloon inflation) is evident in early systole, when the pressure and flow waveforms suddenly deviate from each other (a backward compression wave increases pressure and decelerates flow). The arrival of the backward expansion wave (due to balloon deflation) is evident in late systole, when the pressure and flow cross over (a backward expansion wave decreases pressure and accelerates flow).

**DISCUSSION**

We assumed that the aortic windkessel is fundamentally and essentially a hydraulic integrator and demonstrated that \( P_{Wk} \) is proportional to independent estimates of aortic volume. When \( P_{Wk} \) was subtracted from central \( P_{Ao} \) to define the pressure difference driving flow into the windkessel, that difference (here called \( P_{ex} \)) is directly and quite precisely proportional to the aortic inflow \( Q_{in} \). This observation could resolve the long-standing paradox arising from differences between \( P_{Ao} \) and flow waveforms. The ratio of \( P_{ex} \) to \( Q_{in} \) defines a proximal resistance, \( R_{prox} = P_{ex}/Q_{in} \), that is...
not quantitatively different from the characteristic impedance determined by traditional methods (45). We are proposing a way of subdividing PAo (into windkessel and wave components) that could illuminate the complex interaction between the left ventricle and the arterial system.

Frank’s windkessel model, however controversial, is still the most popular model of the arterial system (3, 13). It successfully explains diastole as the discharging of a volume integrator in which pressure falls exponentially and uniformly, as we demonstrated in Fig. 3. It has been widely utilized to estimate arterial compliance and stroke volume but, despite its popularity, Frank’s windkessel has been frequently criticized for its poor prediction of aortic waveforms, its failure to include wave reflection, and its seemingly inherent implication that wave speed is infinite (20, 28). However, the first criticisms are met by our identification of Pex, which, when added to PWk, predicts systolic aortic waveforms precisely and also accounts for wave reflection. The last criticism is meaningful only if one is attempting to explain the decline in aortic diastolic pressure using wave theory. The essence of this paper is the proposition that the decline in aortic diastolic pressure can be explained completely by the decreasing volume of the aorta (as outflow continues in the absence of inflow); we do not attempt to explain it by waves. The explanation is as straightforward and, perhaps, as compelling as the conclusion that the pressure in the bottom of a bathtub decreases because the water drains out of it. Thus the wave speed criticism of the windkessel would appear to be moot.

Other arterial models based on wave theory, the elastic tube model by Womersley (19, 47) and the lumped electrical circuit models based on transmission line theory (1, 44), may explain the phasic differences in the aortic waveforms, but they introduce other problems of interpretation.

For example, is wave length a simple inverse function of heart rate in diving mammals, which experience profound bradycardia, decreasing their heart rates from ~70 to ~10 beats/min? Does the wave length really increase sevenfold? Furthermore, the frequency-domain approach postulates that during diastole, there are identically declining forward and backward pressures, which add to yield the measured pressure (see Fig. 6). In Fig. 6, beat 2 is rather ineffective and produced a very small stroke volume, capable of increasing PAo by only a few millimeters of mercury. Application of our windkessel algorithm shows an excellent fit (Fig. 6A): the monotonic decrease in PWk is interrupted by the small ejection, during which aortic inflow briefly exceeds aortic outflow. Figure 6B shows that calculated Pex corresponds to aortic inflow for each beat. Figure 6, C and D, illustrates the results of the frequency-domain approach. Forward and backward pressure (Fig. 6C) and velocity (Fig. 6D) waves are calculated. It seems difficult to explain why the backward pressure wave after beat 2 is so small when the preceding beat was large. Similarly, after beat 3, why is the backward wave so large? What is implied about the persistence of resonant waves of different frequencies by this sequence of beats? As well as the problem of analyzing individual beats, these difficulties may stem from the fundamental assumptions in the established approach.

Since the 1960s, Frank’s windkessel model has been simulated using a two-element R-C circuit, a capacitor, and a resistor in parallel (23) and has been analyzed almost exclusively in the frequency domain (2, 7, 21, 23, 28, 39). The later addition of a proximal resistor led to improved prediction of systolic pressure (45). However, in our view, these approaches have not been a wholly satisfactory explanation of the differences between PAo and flow waveforms. Because Pex was not represented in the time domain, it was not clear how well PWk explains the variation in diastolic PAo. Because Pex was not subtracted from PAo, it was not clear that PWk was proportional, thus resolving the differences between PAo and flow waveforms.

The Windkessel as a Reservoir

To determine how precisely changes in PWk were proportional to changes in aortic volume, we scaled PWk arbitrarily and compared it with two independent estimates of the change in thoracic aortic volume. For the first estimate, we isolated the segment of the aorta between the aortic root and the diaphragm and then estimated its volume change by comparing the inflow (aortic root flow) to the outflows (flows in the brachiocephalic and left subclavian arteries and in the aorta at the diaphragm). For the second estimate, we measured aortic diameter at the root and at the diaphragm and, by assuming that the intervening segment was a truncated cone, we calculated the change in total volume. Both these estimates of blood volume change correlated very closely with the variation of ΔVWk, but there were small differences (Fig. 4C). The inflow-outflow
Fig. 3. A: three-dimensional plot of $P_{Ao}$ versus time and distance (by 2-cm increments, from the aortic root to the femoral artery). Data are from a single dog. B: isobar contour plot of the same data (each line indicates an increment of 2 mmHg). Note that, during late diastole, pressure is dependent on time but is independent of distance.
difference and the calculated volume both led $P_{Wk}$ during early ejection, which may be due to the fact that the ejected stroke volume was not completely distributed through the windkessel at that time. Also, after closure of the aortic valve, the inflow-outflow difference was greater than $P_{Wk}$ and the calculated volume. This may be due to the effect of forward diastolic flow at the diaphragm, which momentarily decreased the integral of diaphragmatic flow (see Fig. 4A), thus increasing the inflow-outflow difference.

To determine how the magnitude of the calculated change in total $V_{Wk}$ compared with our estimates of thoracic aortic volume, we converted $\Delta P_{Wk}$ to $\Delta V_{Wk}$ (by multiplying by $C$) and compared it with the inflow-outflow difference (see Fig. 4D). The results indicate that $45.1 \pm 2.0\%$ of the total $V_{Wk}$ is contained in the aorta within the thorax. To our knowledge, this is the first such measurement of $V_{Wk}$. On the basis of Westerhof’s data (44) from his electrical model for the arterial system, Stergiopolus (37) suggested that up to 65% of compliance is contained in the aortic trunk (ascending, descending, and thoracic aorta), an estimate that is not substantially different from ours, given that we did not consider the volume of the large thoracic branches of the aorta.

**Implications of “Waves”**

The wave nature of arterial flow is well established and universally accepted (2). It is also well established, but apparently not widely recognized, that pressure and flow are so inextricably linked in arterial waves that waves should be thought of as pressure/flow waves rather than separate pressure and flow waves. All waves involve an interchange between different forms of energy (15); in arterial waves, this interchange is between the elastic energy of the wall (mediated by the pressure) and the kinetic energy of the flow. This means that the change in pressure caused by an arterial wave is proportional to the change in flow that it causes (9). The relationship between the instantaneous change in pressure and the instantaneous change in flow is given by the water hammer equation (20, 23).

Different usages of “wave” demand that we reconsider its definition. As we have written elsewhere (11, 25, 26, 40, 43), using wave intensity analysis, we deal

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**Fig. 4.** A: thoracic aortic inflow ($Q_{in}$) and outflows ($Q_{dia}$, flow of the aorta at the diaphragm; $Q_{br}$, flow of the brachiocephalic artery; $Q_{sub}$, flow of the subclavian artery). The small branches of arteries were occluded. B: integrals of thoracic inflow and outflows (i.e., the volume coming into and flowing out of the thoracic aorta during one complete cycle). Note that the hatched area, the difference between inflow and summed outflows, represents the instantaneous change in windkessel volume ($V_{Wk}$). $V_{in}$, inflow volume; $V_{out}$, outflow volume; $V_{dia}$, volume of the aorta at the diaphragm; $V_{br}$, volume of the brachiocephalic artery; $V_{sub}$, volume of the subclavian artery. C: volume change in the thoracic aorta calculated by crystal measurements (black line) and by differences of inflow and outflows (blue line) plotted with the calculated $P_{Wk}$ (red line). They are similar in shape. D: total, absolute volume change of the whole arterial system during a cycle ($\Delta V_{Wk}$; red line) compared with the volume change in the thoracic section (inflow minus outflow; black line). Approximately 45% of the total $V_{Wk}$ is contained in the thoracic aorta.
with the propagation of infinitesimal wavefronts, the summation of which we define as waves [e.g., the forward-traveling compression wave that increases PAo and accelerates the stroke volume (25); see Fig. 1C]. This is consistent with the classical definition—a propagated disturbance (15). In our view, it is this forward-traveling compression wave, in particular, that is propagated toward the periphery and reflected to arrive back at the aortic valve before the end of left ventricular ejection in older people and others with stiffened aortas and increased wave speeds [i.e., Murgo’s type A pressure waveform (21)]. The incremental wavefronts are defined by the changes in pressure and velocity during each sampling interval, and it is the temporal summation of these successive wavefronts that give rise to pressure and velocity “waveforms” (this should be contrasted with Fourier transform-based impedance analyses, in which the measured pressure and velocity waveforms are treated as the superposition of sinusoidal wavetrains with different frequencies).

The one-dimensional theory of waves in elastic tubes implies that the differences between the pressure and flow waveforms measured in the root of the aorta can only be the result of backward waves arising from reflection sites (2, 23, 28) (see Fig. 6). Despite the consensus that there are few waves during late diastole (37, 38), using the impedance approach, the diastolic contour has to be accounted for as the sum of forward- and backward-traveling waves (2, 21, 28, 39). Furthermore, this mathematical solution requires that the amplitudes of the two waves are equal and exactly one-half the amplitude of the variation in aortic pressure during this interval (2, 23, 28).

On the other hand, according to our interpretation, the similarity of Pex and Qin and the results of wave intensity analysis imply that the Pex and Qin waveforms can be almost completely explained by forward-traveling waves (i.e., the forward compression and ex-

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pansion waves generated by the left ventricle). To better assess the possible contribution of backward-traveling waves, we introduced a counterpulsation balloon in the abdominal aorta and triggered it so that a backward compression wave (quickly followed by a backward expansion wave) would arrive at the aortic valve during ejection (see Fig. 5). The backward compression wave, caused by the inflation of the balloon, increased $P_{\text{ex}}$ but decreased $Q_{\text{in}}$ (Fig. 5B). The immediately following backward expansion wave, caused by the deflation of the balloon, decreased $P_{\text{ex}}$ but increased $Q_{\text{in}}$. These results support the conclusion that, after isolating $P_{\text{wk}}$ from $P_{\text{Ao}}$, the waveform of $P_{\text{ex}}$ is identical to that of $Q_{\text{in}}$ if there are no backward waves but different in contour if there are.

Furthermore, we note that the success of lumped-parameter electrical analog models of the arteries that implicitly assume a linear relationship between $P$ and $Q$ implies that there are no significant reflected waves in the root of the aorta. One of the most successful electrical circuit models is the three-element windkessel (45), which, following Broemser and Ranke (4), was proposed by Westerhof and is deferentially called the westkessel by some authors. The westkessel added a characteristic impedance, $\rho c/\rho c$, in series and proximal to Frank’s windkessel model, a parallel $R-C$ circuit. Its input is a current source, $Q_{\text{in}}$, and the outputs are the pressure across the characteristic impedance ($P_{c} = \rho c/\rho c \times Q_{\text{in}}$) and the windkessel pressure ($P = P_{c} + P_{\text{wk}}$). The pressure across the characteristic impedance is defined by the water hammer equation, $P_{c} = \rho c/\rho c \times Q_{\text{in}}$, which shows the pressure-flow relation for forward waves. In the time domain, the westkessel model describes the $P_{\text{Ao}}$ waveform as just the summation of a forward-traveling wave and the windkessel, which is the time-varying reservoir pressure. There is no backward wave in this model. That the westkessel could be used to successfully simulate the aortic waveforms of numerous species suggests that reflected waves are negligible in most of those species.

Fig. 6. Comparative analysis of a series of irregular beats using the windkessel-wave method (A and B) and the impedance method (C and D). A: calculated $P_{\text{wk}}$ (red line) fits the measured $P_{\text{Ao}}$ (black line) for each beat. B: calculated $P_{\text{ex}}$ (pink line) has similar contours as $Q_{\text{in}}$ (black line) for each beat. C and D: forward ($P_{\text{forward}}$, blue line) and backward ($P_{\text{backward}}$, red line) pressures (C) and forward ($U_{\text{forward}}$, blue line) and backward ($U_{\text{backward}}$, red line) velocities (D) calculated using the impedance method. See text for details.
Comparison to Impedance Analysis

It is not surprising that there should be similarities between our time-domain analysis and the established frequency-domain analysis. First, our definition of $P_{\text{rel}}$ arises from the assumption that the windkessel is a hydraulic integrator that corresponds to a two-element windkessel (i.e., a capacitance $C$ and a distal resistance $R$ connected to an outflow compartment whose pressure, $P_\text{ex}$, is not necessarily venous pressure); studies of the impulse response approximate our description of the windkessel, differing only because left ventricular ejection is somewhat sustained and not a pure impulse (5, 8, 14, 35). Second, our $P_{\text{ex}}$-$Q_{\text{in}}$ ratio also seems to correspond exactly to characteristic impedance, which, in the electrical analog, separates the flow source from the integrator in the three-element windkessel (45, 571). As Westerhof (45) originally pointed out and as Quick et al. (28) noticed, the operation of characteristic impedance on aortic inflow yields a pressure that is proportional to flow, thus equivalent to our $P_{\text{ex}}$. They termed this pressure “reflectionless,” which is consistent with our conclusion that $P_{\text{ex}}$ can be almost entirely explained by forward-traveling waves.

Thus the reader may be satisfied that our results are consistent with established work but may not believe that our approach has any important advantages. We believe there are three. First and perhaps foremost, the time-domain identification of $P_{\text{rel}}$ is eminently intuitive and easy to explain. The concept of a hydraulic integrator (based on the classical theory) is obvious to every student of physiology who has seen the classical diagrams, and it seems unfortunate that the workings of this simple device should have been so obscured by the frequency-domain analysis. Second, on a beat-to-beat basis, the time-domain representation of $P_{\text{rel}}$ and $V_{\text{rel}}$ can be related directly to primary hemodynamic measurements—pressures, dimensions, and flows—so interpretation is more straightforward, and new insights may be expected. Third, wave intensity analysis (10, 11, 16, 36, 40) provides for wave motion a similarly direct representation, identifying forward- and backward-traveling compression and expansion waves in the time domain, whereas variations of impedance spectra that are subtle and perhaps ambiguous are the only evidence of alterations in wave motion in the frequency domain. Having isolated $P_{\text{rel}}$ and continuing to employ wave-intensity analysis, we believe we have an alternative paradigm that will increase our understanding of arterial hemodynamics.

In conclusion, we feel that our proposed interpretation of aortic hemodynamics has some fundamental advantages. Our division of the pressure into $P_{\text{rel}}$ and $P_{\text{ex}}$ is based on recognized mechanistic properties of the arteries, the capacitive nature of the elastic arteries, and the wavelike nature of arterial flow. To the contrary, the description of the pressure waveform as the superposition of sinusoidal waves has no mechanical basis but is based on the mathematical observation that any periodic waveform can be represented by a Fourier series (or, in fact, by any orthogonal basis functions). Just because a waveform can be represented by sinusoidal waves, it does not follow that it was generated by sinusoidal waves. By ascribing the bulk of the pressure variation during diastole to the windkessel process, we find that the $P_{\text{ex}}$ waveform is virtually identical to the measured flow waveform. This implies that reflected waves are not very significant in the ascending aorta under normal conditions, which is consistent with previous observations. Finally, because $P_{\text{rel}}$ and $P_{\text{ex}}$ have a mechanistic basis, their separation could have implications about the mechanical linkage between the left ventricle and the arteries and therefore to a better understanding of the energetics of ventricular contraction. These implications require further study.

We acknowledge the excellent technical support provided by Cheryl Meek, Gerald Groves, and Rozsa Sas.

This study was supported by a grant-in-aid from the Heart and Stroke Foundation of Alberta (Calgary, Alberta, Canada) and by Canadian Institutes for Health Research (Ottawa, Ontario, Canada) Grant-In-Aid MT-15418 (to J. V. Tyberg). A. B. O’Brien was supported by the Dale and Rushton Fund of The Physiological Society, N. G. Shrive holds a Killam Professorship, and J. V. Tyberg is a Heritage Scientist of the Alberta Heritage Foundation for Medical Research (Edmonton, Alberta, Canada).

REFERENCES


