Coupling of left ventricular and aortic volume elasticity in the rabbit

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Wronski, T., P. B. Persson, E. Seeliger, A. Harnath, and B. Flemming. Coupling of left ventricular and aortic volume elasticity in the rabbit. Am J Physiol Regulatory Integrative Comp Physiol 279: R539–R547, 2000.—Changes in volume elasticity (VE) of the left ventricle and aorta could be important for blood flow. A procedure is presented to rapidly assess VE of the left ventricle and aorta by analyzing changes in the eigenfrequency. Six control rabbits and 11 rabbits with atheromatosis (12 wk of high-cholesterol feeding) were studied. In control rabbits, during the first half of the systole, left ventricular VE continuously increased to +43% (P < 0.05). Then VE gradually declined to an end-diastolic minimum (20% of the average systolic levels, P < 0.05). Aortic VE changes were in the opposite direction to the ventricle. Aortic VE continuously decreased throughout the systole; the last value was 20% lower than at the beginning of the systole (P < 0.05). Conversely, diastolic VE of the aorta took on greater values. This inverse time course between ventricle and aorta may reduce energy requirements for conveying blood. High cholesterol-fed rabbits did not reveal the inverse behavior of ventricular and aortic VE, e.g., aortic VE increased during the systole (119%, P < 0.05).

heart; aorta; compliance; left ventricular-arterial coupling; eigenfrequency; cholesterol; atherosclerosis

THE LEFT VENTRICLE CAN BE DESCRIBED as an elastic chamber, the mechanical properties of which change during the heart cycle. Similarly, compliance of the arterial system is not constant, although often treated as such, but varies because it is a function of distending pressure (1, 32). The coupling between the mechanical properties of the left ventricle and aorta is a decisive element in reducing energy requirements (5, 14, 21). From this energetic point of view, the cardiovascular system may aim at optimizing the relationship between flow and pressure along these compartments.

Volume-pressure relationships are commonly described in terms of compliance, elasticity, volume elasticity (VE), stiffness, or impedance, and several recent studies have assessed such volume-pressure relationships with regard to the cardiovascular system (4, 16, 17, 19, 20, 27, 32). These previous studies have significantly enhanced our understanding in this field; however, they have not yet fully resolved the dynamic coupling between the heart and vasculature. This is due to the technically demanding requirements that have led to a rigid spatial and temporal focus. For instance, some investigators analyze the left ventricle or the arterial vessels (6, 13, 25), whereas others study certain episodes, such as the telesystole (29) or the isovolumic contraction period (6, 13).

In this study, we present a technique that allows determination of several values of VE, which equal change in pressure over volume (mmHg/ml). Measurements were made simultaneously for both compartments, i.e., the left ventricle and aorta. The steep pressure changes in left ventricular and aortic pressure induced oscillations of a modified catheter-transducer system (CTS). The frequency and dampening of these oscillations depend, on the one hand, on the eigenfrequency, i.e., on the intrinsic dynamic characteristics of the system and dampening of the measuring system. On the other hand, they also rely on the properties of the heart and aorta. The shift in oscillation frequency and dampening were used for calculation of VE. By subdividing the dampened oscillation process into smaller segments, it was possible to determine a corresponding amount of VE estimates. The experiments were performed under closed-chest conditions in the anesthetized rabbit.

MATERIAL AND METHODS

Experimental groups. Rabbits (4 mo old) of both genders were used. They had access to food and water ad libitum. The control group (n = 6) was fed a standard pellet diet. The experimental group (n = 11) received a daily 120-g ration consisting of a standard pellet mixture with added 20 ml vegetable oil and 2 g of cholesterol. Cholesterol feeding was maintained for 12 wk and was terminated 2 wk before the experiment.

Surgical procedures. Rabbits were anesthetized with intravenous α-chloralose (60 mg/kg) and urethan (200 mg/kg). Rectal temperature was maintained between 37°C and 39°C by a thermostat table. The trachea was cannulated via tracheotomy; each animal breathed spontaneously throughout the experiments. After surgery, the animal was placed in the supine position. A constant-flow infusion pump was connected to a cannula placed into the right brachial vein. An infusion of a mixture of 180 mmol/l of glucose and 180 mmol/l mannitol at 0.12 ml/min was used to maintain constant

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plasma volume and osmolality. A catheter with a length of 15 cm and an outer diameter of 2.5 mm was inserted into the left ventricle via the carotid artery. An aortic catheter with similar dimensions was positioned via the brachial artery.

**Transducer.** Left ventricular and aortic pressures were measured with pressure transducers (Siemens Elema, Sweden), as depicted in Fig. 1, A and B. Analog-to-digital conversion was performed at a rate of 1,000 Hz and an accuracy of 12 bits.

A chamber for decreasing the VE of the transducer was attached to this setup (Fig. 1, C and D). This attached compartment consisted of a short tube of a transparent material, to allow bubbleless filling. Both sides of the tube were sealed with steel membranes, which, together with the wall of the tube, determine the elastic properties of the transducer chamber (Fig. 1C). The resulting VE coefficient of the measuring device of ventricular pressure with the attached chamber was 27,950 mmHg/ml. The corresponding VE for the measuring device of aortic pressure with the attached chamber was \( E_{T9} \) 29,400 mmHg/ml. VE of the transducer system was determined statically by applying pressure and measuring the volume change. Moreover, a dynamic VE measurement of the setup was made by applying external pressure oscillations. The relative standard error for repeated controls of measuring system VE was 3%.

Figure 2 shows a typical left ventricular pressure curve with the oscillating component. The pressure induced by the heart is superimposed by damped oscillations during ejection and filling (Fig. 2A). The heart itself creates these oscillations.

**Data analysis.** For a detailed description of the theoretical background see the Appendix. Left ventricular pressure was separated from the superimposed eigenoscillations as depicted in Fig. 2. To this end, the transducer chamber was repeatedly disconnected from the measuring setup, thus providing “pure” blood pressure traces without superimposed oscillations. A reliable reconstruction of the real blood pressure time course was achieved by a combination of the pure recordings and the original time series after smoothing. A second-order Butterworth filter with a corner frequency of 18 Hz eliminated the superimposed oscillations, however, at the cost of blunting the steep pressure changes that occur during the isovolumic phases of the heart cycle. The intersection points of the filtered and unfiltered pressure recordings during ejection and filling agree well with the pure pressure traces. Pressure at these intersections stems from left ventricular pressure only. The time course of left ventricular pressure was obtained by third-order spline interpolation of the intersection values and two additional values before and thereafter. Subtraction of left ventricular pressure from the original pressure signal provides the damped pressure oscil-

Fig. 1. A schematic of the experimental setup. **A:** catheter-transducer system (CTS) coupled to a hollow body (\( M \) is the effective mass of the catheter-filling liquid, \( E_T \) is the volume elasticity of the transducer, \( Z \) is the friction resistance of the catheter filling liquid, \( E \) is the volume elasticity of the hollow body, \( q \) is the oscillatory flow in the catheter). **B:** electrical analog model of A. **C:** CTS connected to the additional parallel chamber for decreasing volume elasticity, \( E_C \) is the volume elasticity of the chamber. **D:** electrical analog model of the latter setup is specified. **E:** an electrical analog model of a CTS coupled to a hollow body with cylindrical opening. This setup was used for modeling the ventricle during the ejection phase and filling phase. Thus there is an additional mass component \( m \) (the blood column in the aorta) and a friction resistance \( r \). \( R \) refers to the viscous resistance of the ventricular wall. **F:** the analog model for \( E \), after summarizing \( r \) and \( R \) to a resulting \( r' \).
The experimentally determined flow (damped oscillation pressure is divided by the VE value of the experimentally determined velocity of the transducer (see Eqs. A4a and A4b). The starting values were $E' = 300$ mmHg/ml, $r' = 1$ mmHg s/ml, initial value $q_1 = f_1$ (first value of $f$), initial value $dq/dt = \text{numeric derivative at point } f_1$, initial value $q_2 = \text{initial value } q_1$, and initial value $dq/dt = \text{initial value } dq/dt$. Fitting was performed by the Levenberg-Marquardt algorithm (23). When this procedure converged and the relative changes in all fit parameters became $<10^{-4}$, calculation was terminated and the determined parameters were stored.

A time interval of 40 ms was chosen for fitting in which averaged values for $E'$ and $r'$ were obtained. The step rate was 10 ms.

The effective mass ($M$ coefficient) cannot be fitted, because various combinations of $E'$ and $M$ will yield the same angular frequency. Thus the differential Eqs. A4a and A4b cannot be resolved unequivocally. Therefore, $M$ was determined by the aortic and atrial geometry. It must be considered, however, that during the ejection phase, axial and radial distension of the aorta occurs. The axial component tapers off along the aorta. Thus only a short aortic segment can be taken for estimating the mass. Aortic diameter was around 3 mm in the animals; 2 mm were assumed for the cylinder length. According to Eq. A1, the resulting mass is $M = 0.0025$ mmHg s$^2$/ml $= 18.75 \times 10^3$ kg/m$^4$.

**Calibration of the method.** There is no “gold standard” for measuring elastic properties of objects that geometry vary time dependently. The acquisition device, mathematical modeling, and the software were therefore tested in vitro. Various artificial hollow bodies with known VE were analyzed. An air bubble of a defined volume was injected. VE of an air bubble can be determined statically or it can be calculated by the laws of thermodynamics. An air bubble of 1 ml has a VE coefficient $E' = 760$ mmHg/ml, provided atmospheric pressure is 760 mmHg. This value is valid only for isothermal compression or decompression with small amplitudes. Because of the rapid oscillations of our measuring system, this condition is not fulfilled. In fact, the value for $E'$ approaches 1.064 mmHg/ml, as under adiabatic conditions. This factor of 1.4 results from the quotient $c_p/c_v$ (specific heat at constant pressure/specific heat at constant volume) for a two atomic gas mixture.

The calibration with external hollow bodies required a piezoelectric stimulation of the system. Thus a piezoelectric disk was fixed onto one of the membranes of the attached transducer chambers ($E_2$), as depicted in Fig. 1C. The calibration chamber with the air bubble was then sealed. The calculation of $E'$ was based on Eq. A2.

To model a branched system, such as the heart attached to the aorta, a calibration chamber was used containing a cylindrical borehole. One wall of the chamber consisted of an elastic rubber membrane, whereas the other wall was stiff. The calculations were based on Eqs. A4a and A4b.

**Statistical analysis.** The heart beats from each animal were analyzed during at least one complete respiratory cycle. All data of a heart cycle were normalized to yield a standard-ized heart beat with the relative duration of one. These normalized heart beats of each animal during the complete respiratory cycle were summarized to one resulting representative heart beat for each group (see Figs. 5 and 6). The tick interval of these figures is 0.05. For an average heart rate of 300 beats/min, this value corresponds to 10 ms. The fitting procedure provided moving average values every 10 ms; the window size was 40 ms.

All values are given as means ± SE. Significance was tested using the Wilcoxon test for paired or unpaired observations. Differences in values exceeding $P < 0.05$ were considered significant.

**RESULTS**

**Effects of cholesterol feeding.** Table 1 shows control data for the anesthetized rabbits under a normal (control) or cholesterol-enriched diet (cholesterol). Heart rate and mean arterial pressure are not statistically different between the normal and the atherosclerotic rabbits.

None of the control animals shows signs of atherosclerosis. Conversely, the aortas and carotid arteries from the cholesterol-fed rabbits consistently reveal atherosclerotic lesions (data not shown). Moreover, cholesterol plasma concentrations of these animals are drastically elevated (Table 1).

**Validation procedure.** Figure 3 shows the effect of air bubbles of 0.5, 1, and 2 ml on the angular frequency and the resulting VE. For this validation experiment, the electrical stimulation of the piezoelectric disk generated the damped oscillation (Fig. 3A). Reducing the volume of air bubbles from 2 to 0.5 ml causes an increase of angular frequency from 158 to 164.2 Hz (Fig. 3B). The precision of the calculation diminishes with decreasing oscillation amplitude, i.e., the variances become larger over time. Figure 3C shows the three different time courses of the VE coefficient $E'$ as obtained by Eq. A2. The time courses and absolute levels of angular frequency and $E'$ are practically identical. In these feasibility experiments, the frequency alone contains all information of VE, because an air bubble has no viscous properties and the damping coefficient does not change.

Figure 3D depicts the calculated values of $E'$ vs. the theoretical values for air bubbles. Also indicated is the result of calculating a calibration chamber with rubber membrane and parallel connection of a mass. The statistically measured value of the rubber membrane is 307 mmHg/ml; the calculated value from Eq. A4 is 300 ±

| Table 1. HR, MAP, body weight, plasma cholesterol, sodium, potassium, and calcium in the control and cholesterol-fed rabbits |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|
| **Control**                     | **Cholesterol** |
| Body weight, kg                 | 4.2 ± 0.4       | 4.3 ± 0.5       |
| HR, beats/min                   | 292 ± 15        | 306 ± 7         |
| MAP, mmHg                       | 80 ± 7          | 77 ± 8          |
| Plasma cholesterol, mmol/l      | 1.9 ± 0.017     | 13.7 ± 4.1     |
| Plasma sodium, mmol/l           | 138 ± 1.1       | 132 ± 0.7      |
| Plasma potassium, mmol/l        | 3.78 ± 0.2      | 3.79 ± 0.24    |
| Plasma calcium, mmol/l          | 3.57 ± 0.1      | 3.47 ± 0.07    |

Values are means ± SE; n = 6 control and 11 cholesterol-fed rabbits. HR, heart rate; MAP, mean arterial pressure. *P < 0.05 (cholesterol vs. control).
The correlation coefficient of all values in Fig. 3D is 0.9997 and the gain is 0.91.

Time course of VE and viscous resistance. Figure 4A depicts a time course of left ventricular pressure with eigenfrequency oscillations for 80 consecutive heart beats. The corresponding values for VE are shown in Fig. 4B. Synchronous changes to the respiratory cycle are apparent in both panels. Arrhythmic beats occur at beat numbers 34 and 48, which also influences VE.

Figure 5A shows the time series of a normalized beat, averaged over one respiratory cycle, of left ventricular and aortic pressures. In Fig. 5B, the left ventricular VE is depicted. In the control group, there is a marked increase in VE during the first half of the systole, which later tapers off. The diastolic VE values for both groups are significantly lower than those during the systole. Remarkably, VE continuously decreases during the diastole and finally reaches a baseline level that amounts to <20% of the average systolic levels.

The cholesterol-fed animals reach maximum systolic VE earlier than the controls. Furthermore, the changes in VE are not pronounced as in the atherosclerotic group.

The changes in aortic VE are less than those of the left ventricle. Furthermore, the changes seen during the first half of the systole are in the opposite direction as for left ventricular VE. As depicted in Fig. 5C, the control animals exhibit steadily declining aortic VE values during the systole. Greater VE is observed during the later one-half of the diastole. This dynamic response is perturbed in the cholesterol-fed group. In fact, these animals reveal a slight increase in VE during the systole. Diastolic changes are in the same direction in both groups; however, the magnitude of changes is somewhat less in the atherosclerotic animals.

The time course of left ventricular viscous resistance is similar to the changes in VE (data not shown). In control animals at the onset of the systole, viscous resistance is 0.16 mmHg s/ml. A maximum of 0.23 mmHg s/ml is reached at 0.35 of the normalized beat. There are no major changes in visceral viscous resistance for the cholesterol-fed animals. During the diastole, viscous resistance reaches lower values for both groups, in resemblance to the VE time course. No consistent changes of aortic viscous resistance are observed during the systole. Within the diastole there was an increase to twofold levels; however, the values had a greater stray than for VE.

In Fig. 6, all values are normalized by considering the earliest systolic value as 1. After this procedure, the pressure time courses of both groups overlap. Remarkably, on the other hand, the VE data maintain their typical pattern. Basically, the same differences are observed for the absolute data, i.e., the animals kept on an atherogenic diet have a blunted response in left ventricular VE. The differences in the aortic VE time course is also apparent. As shown in Fig. 6C, the control animals decrease aortic VE significantly. In contrast, increase aortic VE significantly.

DISCUSSION

In the present study, analysis of eigenfrequency detuning proved to be a feasible method to determine VE. The procedure was validated ex vivo in an artificial setup. As depicted in Fig. 3D, the estimations correlated very well with the theoretical values (r = 0.9997, gain 0.91). The reason for having a gain slightly less than the line of identity is due to the fact that strict adiabatic conditions cannot be achieved. In other words, there is a temperature exchange between the tested hollow body and the surrounding environment. Thus the experimental estimates are somewhat below the theoretical values. The relative error of the exper-
imental method was around 3% for time invariant systems.

Considerable efforts have been made to model and determine mechanical properties of the heart and aorta (1, 6, 13, 14, 18, 20, 24, 25, 27, 32). The VE measure relies on several factors, such as wall stiffness, wall thickness, heart chamber volume, and finally on the complex geometry of the heart. VE is particularly important for hemodynamics because it constitutes an immediate link between changes in pressure and volume. The energy requirements for the propulsion of blood can in theory be minimized if VE is adjusted appropriately.

The advantages of the current procedure to determine VE are that the measurements are obtained in situ, that several determinations can be made within one cardiac cycle, and that the values are assessed simultaneously for the left ventricle and aorta.

As demonstrated by Fig. 3, the procedure accurately determines VE for time invariant systems. Moreover, the procedure is very reproducible, as shown in Fig. 4. Hemodynamic changes, such as the respiratory rhythm or arrhythmic episodes, are mirrored in changes of VE. The VE time courses for the heart and aorta seen in Figs. 4–6 are smoothed by a moving average procedure (window size 40 ms). Thus any sudden changes are underestimated. The scope of the present analysis is therefore confined to changes observed among these 40-ms averages. No conclusions can be made regarding potential instantaneous alterations in VE. However, changes in the mean values cannot occur without corresponding alterations in the instantaneous values. Increasing time resolution to <10 ms is possible; however, in practice, this proved to be less reliable due to erratic variations and the relatively coarse digitization. Nonetheless, using this setup, we could demonstrate a specific change of mean values of ventricular and vascular VE even within the systole and diastole (Figs. 4–6). Absolute values for VE vary among previous studies (33). The absolute values in Figs. 4 and 5 depend on the mass, as indicated schematically in Fig. 1F. This mass was estimated and thus the absolute values of VE rely on the accuracy of this approximation. Aortic VE in control animals was found to be around 45 mmHg/ml, which is slightly above the estimates of previous studies (33). Comparisons of relative changes have the advantage that they are independent of mass. The time course of these values, as shown in Fig. 6, underscores the typical pattern of the absolute data (Fig. 5). Peak ventricular VE is reached during the middle of the systole, and concomitantly aortic VE decreases. This behavior of VE for both compartments should facilitate volume transport during the systole. In the diastole, left ventricular VE decreases to <20% of the systolic levels, which eases the filling of the left ventricle. Conversely, diastolic aortic VE takes on greater values, thereby enhancing blood flow to the periphery.

These findings support a concept of Vrettos and Gross (32). They suggest that a decrease in aortic VE during the systole reduces the energy demands on the heart. It is more economical to convey larger volumes...
without changing the pressure difference. This effect is enhanced if VE increases at the source of volume, while VE of the connected conduit vessels decrease, as observed in the present study. We cannot, however, make any conclusions as to whether these changes in cardiovascular VE are brought about actively or whether they occur passively in consequence of contraction and ejection.

Our data, however, do not totally agree with a study of Berger and Li (1). In that study, arterial elasticity reaches a minimum near the start of ventricular ejection. In our hands, there is a continuous decline in aortic VE throughout the entire systole. This may seem in contrast to the general notion that vessel walls become stiffer as transmural pressure increases (15). However, an inverse relationship between VE and pressure may indeed be explainable by the particular vessel wall architecture of large conduit arteries. Joanides et al. (15) propose that changes in midwall stress directly affect wall VE by unloading stiffer wall components. Specifically, during the systole, a decrease in aortic VE may be accomplished by the vascular smooth muscle cells, which unload the stiff collagen fibers in favor of the more elastic elastin fibers. In line with this interpretation, i.e., that elasticity can decrease during vessel contraction, is the finding that arterial elasticity declines during sympathetic stimulation in isolated vessels (15). Remarkably, one only finds a strong relationship between aortic pressure and diameter after chronic sympathetic blockade (10). Other factors, such as nitric oxide formation, seem unlikely to account for adaptations below the range of seconds. Although the formation of nitric oxide is comparatively rapid, it is still too slow to mediate changes on an intrabeat scale (22).

To determine possible changes in the cardiovascular coupling of VE in atherosclerosis, cholesterol feeding was performed in a similar manner as in other inves-
tigations (2, 3). This diet produces well-defined atherosclerotic lesions, as described in previous studies by other groups (8, 9, 26) and ourselves (11). Although this feeding regimen causes widespread lesions in the aorta, blood pressure and heart rate do not change (Table 1). Nonetheless, cholesterol feeding significantly blunted alterations in left ventricular VE.

Moreover, the pattern of aortic VE was perturbed (Fig. 6). We no longer observed an inverse behavior between ventricular and aortic VE. Thus the energy requirements for blood flow should be higher in these animals. Several mechanisms can be put forward to explain the altered response of aortic VE. These include structural and functional changes, e.g., the aortas of hyperlipidemic rabbits typically exhibit less lamellar elastin units (7). Functional alterations also have been reported, such as the medial smooth muscle layer located beneath the atheroma, which is hyperactive to vasoconstrictor agents (12, 30). Furthermore, the endothelium reveals an impaired response to nitric oxide (12, 31).

Perspectives

This study employs a novel technique for assessing changes in left ventricular and aortic VE throughout the systole and diastole. It is suggested, that VE constitutes a dynamic element of great importance within the cardiovascular control network. Changes in VE will have immediate effects on volume propulsion. During the systole, an increase in VE at the origin of blood flow (in this case the left ventricle) together with a decrease in VE at its destination (the aorta) occurs. This increases the pressure difference and thus augments flow. Hence, vessel and chamber elasticity play an important role in optimizing hemodynamic demands. In the diastole, the aortic compartment becomes the source of blood flow. Aortic VE increases during this phase, which again facilitates blood flow to the peripheral segments. Left ventricular VE, on the other hand, decreases in the diastole, which supports the filling phase. If ventricular filling were to occur only passively, then VE should increase exponentially! After high-cholesterol feeding, the interplay between the left ventricular and the aortic VE is perturbed, suggesting that the energy requirements for maintaining blood flow is higher.

APPENDIX

Theoretical background of measurements. As mentioned in MATERIAL AND METHODS, a liquid-filled oscillating system was coupled to the heart and aorta. This was done to continuously monitor VE of these compartments. Changes in cardiac and aortic elasticity also affect the dynamic properties of this oscillating system, i.e., after connecting the oscillating system (with known eigenfrequency and damping characteristics) to the heart chamber, small volume oscillations were induced by the heart. In other words, by adding the cardiac elastic components to the system, the oscillating frequency and the dampening of the measuring device change.

What are the characteristics of the liquid-filled system used? As for all mechanical oscillating systems, there is an elastic component and a mass. The elastic element of the measuring device consisted of a chamber with an elastic wall. The VE coefficient (E) is a characteristic of the chamber. In liquid-filled systems, the so-called effective mass replaces the physical mass. In contrast to physical mass, the effective mass relies on the fluid compartment. The effective mass (M) in a cylinder or a tube is determined by

\[ M = \rho l A \]  

(A1)

The effective mass has the unit kg/m\(^3\) equals \(1.33 \times 10^{-9}\) mmHg s\(^2\)/ml, where \(\rho\) is the density of the liquid, \(l\) is the length of the fluid cylinder, and \(A\) is the cross-section area of the cylinder.

In addition to mass, fluid friction plays a role and can be determined by Poiseuille's law. The frequency of the volume oscillations should on the one hand be high enough to induce several cycles during the heart period, and on the other hand it must not be too high for initiating oscillations in the system. The wavelength decreases with increasing frequency. If the wavelength is greater than the dimensions of the hollow body, then all wall components oscillate with the same phase and each of the spatially distributed elastic, mass, and friction components can be considered as concentrated elements. Thus in this case, the hollow body is represented by only one value for VE, friction resistance, and mass.

Every catheter-transducer system (CTS) is an oscillating system in the above-described sense. Figure 1A shows a schematic CTS coupled to a hollow body. The liquid in the catheter oscillates between the transducer and the hollow body. Because of incompressibility of liquid, the small-volume perturbations induce volume oscillations of the heart. In the thin catheter, the oscillating fluid reaches greater values of velocity and acceleration, and therefore the mass becomes effective only here. Figure 1B shows the electrical analog model of the CTS. The differential Eq. A2 is evident in this model. The sum of all partial pressure components in a mesh must be zero, provided the system is allowed to oscillate freely

\[ Zq + Mdq/dt + E_T \int qdt + E' \int qdt = 0 \]  

(A2)

with the solution

\[ q = q_0 e^{-\delta t} \sin \omega t \]

\[ \delta = \frac{Z}{2M} \]

\[ \omega = \sqrt{\left(E_T + E'\right)/M - \delta^2} \]

where \(E_T\) is the VE of the transducer, \(E'\) is the VE of the hollow body, \(M\) is the effective mass of the catheter, \(Z\) is the friction resistance of the catheter, and \(q\) is the oscillatory flow in the catheter.

The solution of this differential equation is a damped oscillation with two key parameters: 1) the damping \(\delta\) and 2) the angular frequency \(\omega\). The angular frequency contains the unknown parameter \(E'\), beside the three known constants: \(E_T\), \(M\), and \(Z\). If \(E_T > E'\), which is normally the case for conventional pressure measurements, then \(E'\) has no influence on \(\omega\). For this study, we wished to determine the sensitivity of \(\omega\). Thus it was necessary to decrease \(E_T\) of the transducer. Figure 1C depicts this arrangement schematically. A liquid-filled chamber with a VE coefficient \(E_C\) lays parallel to the transducer and decreases the resulting VE

\[ E = E_T E_C / (E_T + E_C) \]  

(A3)
In the presented experiments, the transducer chambers took on $E_c$ values near 30,000 mmHg/ml. This is much less than for any modern pressure transducer. Thus under these conditions the $E'$ of the heart chamber is able to influence $\omega_0$.

This simple model allows the calculation of VE, provided that the hollow body is always sealed. In situ measurement of ventricular VE, however, is more complicated. The heart chamber opens during the ejection and filling phases. In consequence, the oscillating volume branches off, thereby eliciting radial oscillations of the hollow body and axial volume shifting in the adjoining blood vessel. Thus an additional mass ($m$) and friction component ($r$) lay parallel to the heart (under the premise that the aorta is considered as a tube) (28). Figure 1E depicts this arrangement schematically. This model contains four unknown elements: $m$, $r$, $E$, $R$. $E$, $R$ represents the viscous properties of the left ventricular wall. Both resistances $r$ and $R$ can be summarized to a resulting $r'$. Figure 1F depicts this model with its remaining three unknown components. As can be derived from Fig. 1F, two differential equations for the two meshes can be formulated

$$mdq_1/dt + r'q_1 + E'\int q_1 dt - E'\int q_1 dt = 0 \quad (A4a)$$

$$E'\int q_1 dt + r'q_1 + Mdq_1/dt + Zq_1 + E\int q_1 dt - r'q_2 = 0 \quad (A4b)$$

where $q_1$ is the oscillatory flow mesh 1 and $q_2$ is the oscillatory flow mesh 2.

The differential Eqs. A2, A4a, and A4b have solutions that are damped oscillations symmetrical to zero.

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