Compliance of the main pulmonary artery during the ventilatory cycle

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GRANT, BRYDON J. B., AND BARUCH B. LIEBER. *Compliance of the main pulmonary artery during the ventilatory cycle.* J. Appl. Physiol. 72(2): 535–542, 1992.—Transmural pulmonary arterial pressure (Ppa), diameter (D), and length (L) of a segment of the main pulmonary artery (MPA) were measured simultaneously in anesthetized open-chest dogs. The instantaneous volume was calculated from D and L. Pulmonary arterial elasticity for diameter (Ep_D) was calculated as the ratio of the amplitude of Ppa to D oscillations normalized by the mean D. Similar indexes were calculated for L (Ep_L) and V (Ep_V). Compliance per unit length was calculated from the dimensions and elasticity of the MPA. Under control conditions with 5 cmH_2O positive end-expiratory pressure, Ep_D, Ep_L, and Ep_V at cardiac frequency were 175 ± 27, 147 ± 27, and 55 ± 7 cmH_2O, respectively. Ep_D increased with positive end-expiratory pressure, but Ep_L decreased and Ep_V was unaffected. Ep_D, Ep_L, Ep_V, and compliance per unit length were not significantly different between the start of inspiration and the start of expiration. In addition, there were no significant phase differences between the oscillations of Ppa and V at respiratory frequency. We conclude that the previously reported time variation of pulmonary arterial compliance during the ventilatory cycle is not due to time-varying properties of the MPA.

pulmonary arterial compliance; positive end-expiratory pressure; respiratory cycle; time-varying compliance; dogs

PREVIOUSLY, we calculated pulmonary arterial compliance (C) from the input impedance spectrum of the main pulmonary artery (MPA) with a lumped parameter model (5). We found that pulmonary arterial C at the start of inspiration was not altered by positive end-expiratory pressure (PEEP). At each level of PEEP, C was greater at the start of expiration (SE) than at the start of inspiration (SI). The mean increase in C ranged from 9% at 5 cmH_2O PEEP to 169% at 15 cmH_2O. We suggested two mechanisms that might account for the time-varying pulmonary arterial C. First, the time-varying pulmonary arterial C may be due to shifts of blood volume between the extra-alveolar and the alveolar vessels during the ventilatory cycle. Second, the wall of the pulmonary arteries may have time-varying properties.

To clarify further the mechanism of a time-varying pulmonary arterial C, we made direct measurements of the dimensions and elasticity of the MPA in the intact dog. The MPA was studied because most of the pulmonary arterial C is contained within the proximal pulmonary arteries. To calculate C per unit length of the MPA, transmural pressure (Ppa), diameter (D), and length (L) of a segment of MPA were measured simultaneously. The instantaneous volume (V) was calculated from D and L. C of the MPA was calculated at two frequencies: cardiac and respiratory. Cardiac frequency (f) was selected because this was the fundamental f of the impedance spectrum from which pulmonary arterial C was calculated. Respiratory f was selected because time-varying properties of the vascular wall may result in phase shifts between Ppa and V at this f.

METHODS

Animal preparation. Experiments were performed on six mongrel dogs that weighed between 17 and 21 kg. Anesthesia was induced with thiamylal 1.5 mg/kg, immediately followed by intravenous injection of 120 mg/kg body wt of α-chloralose solution. Anesthesia was maintained with a continuous infusion of α-chloralose at a rate of 43 $mg \cdot kg^{-1} \cdot h^{-1}$. Details are provided elsewhere (4).

Each dog was ventilated with 100% oxygen through an endotracheal tube with a volume cycled pump (Harvard Apparatus, Natick, MA, model 681). The pump was used at a respiratory rate of 12–15 min^{-1} with a constant tidal volume throughout each experiment. Inspiration was achieved by positive pressure; expiration was passive. The inspiratory and expiratory times were fixed and equal. Intravenous pancuronium bromide (0.1 mg/kg) was used for muscular paralysis. Additional doses were given if needed throughout the experiment. No pancuronium was given 10 min before or during the period of data collection. A catheter was placed in the right femoral artery to monitor systemic arterial pressure with a Statham P23 ID pressure transducer. The ventilator was adjusted to obtain an arterial P_{CO_2} of ~40 Torr.

A left thoracotomy was performed through the fifth intercostal space, and 5 cmH_2O of PEEP was applied to prevent atelectasis. A longitudinal incision was made in the pericardium, and the pulmonary artery was isolated by blunt dissection.

Dimensions of the MPA were measured by sonomicrometry (model 120, Triton Technology, San Diego, CA). The factory calibration of the sonomicrometer was confirmed by moving a pair of piezoelectric crystals through a known distance. The D of the MPA was measured with each crystal (3 mm diam) attached to a Dacron patch. The patches were sewn to the adventicia midway along the vessel in a plane transverse to the flow

to measure D of the MPA. A second pair of crystals was mounted orthogonally on Dacron patches with polystyrene acoustic support. These crystals were used to measure length of a segment of MPA. The patches were sewn in a longitudinal plane of the MPA to straddle the transverse plane of the D crystals.

Before insertion, a 3F micromanometer-tipped catheter (Millar, Houston, TX) was calibrated with zero set to atmospheric pressure. The catheter was placed through purse-string sutures on the right ventricle into the pulmonary artery. Its tip was placed close to the site of the ultrasonic crystals for measurement of Ppa. This pressure was considered to be the transmural pressure because the chest was open and the lungs were positioned to avoid impinging on the MPA.

Experimental protocol. The protocol was started when the preparation was judged to be stable, as manifested by constant mean systemic arterial pressure. The level of PEEP was increased from 5 to 10 cmH_2O and then from 10 to 15 cmH_2O at intervals of 2 min. This sequence was repeated. During the last minute of each level of PEEP, data were recorded at 250 Hz/channel. In all dogs, measurements were collected over a period of 10 breaths at 5, 10, and 15 cmH_2O PEEP.

Data collection and analysis. All electrical analog signals were converted to digital form (DT2811, Data Translation, Malborough, MA). The signals were displayed on the monitor of an AT compatible computer with a waveform scroller system (Dataq, Akron, OH). Data were collected at 250 Hz/channel and stored on disk for later analysis. The V of the segment of the MPA was calculated at each instant from the simultaneous measurements of D and L

$$V(t) = 0.25\pi D(t)^2/L(t) \quad (1)$$

where t is an instant in time. The ventilatory cycle was identified from the output of a microswitch that was positioned on the ventilator to deliver a 5-V pulse at end expiration. End inspiration was defined as the midpoint between two end-expiratory pulses because the inspiratory and expiratory times were equal.

The start and finish of individual heart cycles were identified from a pulse triggered by the QRS complex of the electrocardiogram (ECG/Biotach amplifier, Gould, Cleveland, OH). Heart cycles were specified at two phases of the ventilatory cycle for 10 successive breaths. Two cardiac cycles were selected in each breath. The first complete cardiac cycle that occurred just after end expiration (SI) was selected. The other cardiac cycle was the first complete cycle that occurred after end inspiration (SE). The same process was used in our previous study (4). SI and SE were chosen to ensure that the cardiac cycles were contained completely within the inspiratory and expiratory phases of the ventilatory cycle, respectively. These data were used to calculate heart rate (HR), mean Ppa, D, and L of the segment of the MPA by averaging over the cardiac cycle at SI and SE.

Direct Fourier analysis was used to calculate the amplitudes and phase relation between pressure and D, pressure and L, L and D, and between pressure and V at SI and SE. The Ppa-D, Ppa-L, L-D, and Ppa-V relations were calculated for each of the set of 10 cardiac cycles. The results were averaged at each f of the Fourier series. The analytical approach used was similar to that described for pressure and flow in a previous report from our laboratory (4). These data were used to calculate elasticity and C at cardiac f at SI and SE. Elasticity was calculated in terms of the pressure strain modulus from the following equation

$$Ep_D(f) = Ppa(f)[D(f)/\bar{D}]^{-1} \quad (2)$$

where $Ppa(f)$ is the amplitude of the pressure oscillations at a given f, $D(f)$ is the amplitude of the D oscillations, and $\bar{D}$ is mean diameter. Similar indexes of elasticity were developed for the segment L and for its V

$$Ep_L(f) = Ppa(f)[L(f)/\bar{L}]^{-1} \quad (3)$$

$$Ep_V(f) = Ppa(f)[V(f)/\bar{V}]^{-1} \quad (4)$$

C per unit L (C_V) of the MPA was calculated in one of two ways. First, it was calculated directly from Ep_V

$$C_V(f) = \bar{V}[\bar{L}Ep_V(f)]^{-1} \quad (5)$$

Second, it was calculated indirectly from Ep_D and Ep_L

$$C_{DL}(f) = 0.5\pi\bar{D}^2\{[Ep_D(f)]^{-1} + [2Ep_L(f)]^{-1}\} \quad (6)$$

The second method neglects the higher order terms of L and D (see APPENDIX). This method ignores any phase difference between the changes of D and L. In addition, Fourier analysis was performed on the entire data set over a single breath for 10 successive breaths. The elasticity and C were calculated similarly at respiratory and at cardiac f. The results for the set of 10 breaths at each level of PEEP were averaged at each f.

If the variation of pulmonary arterial C during the ventilatory cycle is associated with time-varying properties of the MPA, the following results would be expected. First, there would be differences in C_V at cardiac frequency between SI and SE. These differences may result due to changes in mean D, L, V, Ep_D, Ep_L, or Ep_V. Second, the phase relation between Ppa and V would differ from zero. A nonzero phase relation may result from a nonzero phase relation between Ppa and D and/or between Ppa and L.

Statistical analysis. We used analysis of variance for repeated measurements for statistical assessment (3). A Bonferroni correction was used for post hoc tests. Circular mean phase angles and SD[1] were calculated by directional statistical methods (14). The existence of a mean angle was verified by Raleigh's R test. The median test was used to determine whether a mean angle differed from zero. It was also used to determine the significance of differences between two mean angles. Statistical significance was accepted at the 5% level in all cases. Results are shown as the mean ± standard error unless otherwise indicated.

RESULTS

Figure 1 shows the temporal changes of Ppa, D, and L of a segment of main pulmonary artery during the venti-

[1] The circular mean of angles (a_i, $i = 1 \cdots n$) is calculated as arctan (y/x) where $x = (\Sigma \cos a_i)/n$ and $y = (\Sigma \sin a_i)/n$. The circular standard deviation is $\sqrt{(-2 \log_e r)}$ radians where $r = \sqrt{(x^2 + y^2)}$.

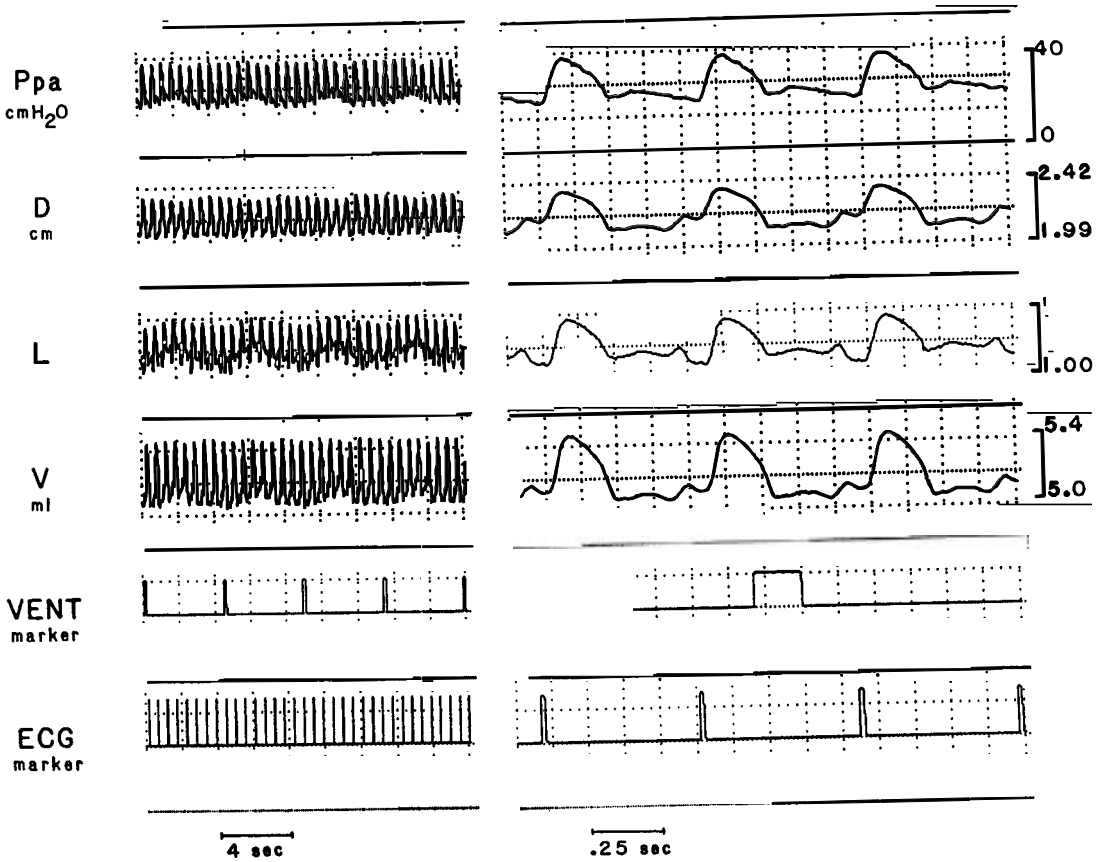

FIG. 1. Temporal changes of transmural pressure (Ppa), diameter (D), length (L), and volume (V) of segment of main pulmonary artery (MPA) during respiratory and cardiac cycles. V was calculated from instantaneous values of D and L.

latory and cardiac cycles. The instantaneous measurement of V was calculated from the instantaneous values of D and L.

The changes in mean Ppa, D, L, and V of a segment of the MPA during the ventilatory cycle are shown in Fig. 2. Mean Ppa increased with PEEP ($P < 0.005$), but there was no significant change between SI and SE. Although there was a significant increase in D between SI and SE, the magnitude of these changes was very small. There were small increases in both mean D and V with PEEP. There was no significant change in mean L.

HR was not significantly different at SI than at SE. At 5 cmH_2O of PEEP, HR was 2.22 ± 0.13 Hz at SI and 2.24 ± 0.12 Hz at SE. At 10 cmH_2O of PEEP, HR was 2.29 ± 0.13 Hz at SI and 2.27 ± 0.14 Hz at SE. At 15 cmH_2O of PEEP, HR was 2.28 ± 0.13 Hz at SI and 2.3 ± 0.14 Hz at SE.

Figure 3 shows Ep_D, Ep_L, Ep_V and C_V of a segment of MPA at cardiac f during the ventilatory cycle. There was no significant difference between SI and SE for Ep_D, Ep_L, or Ep_V. PEEP increased Ep_D ($P < 0.025$) but decreased Ep_L ($P < 0.05$), whereas Ep_V was unaffected. The overall effect of the changes of vascular dimensions and elasticity is expressed by C_V. C_V was unaffected by the phase of the ventilatory cycle or PEEP.

The effect of ventilation and PEEP on the phase relation between Ppa and D, between Ppa and L, and between Ppa and V is shown in Table 1. There were no significant effects of ventilation on the phase relations of Ppa and D or Ppa and V. The phase relation between Ppa and L at SI was a statistically significant difference from the phase at SE, but these differences were of small magnitude. Both the phase relation between Ppa and D and between Ppa and L were significantly different from zero at both SI and SE. The negative phase relation between Ppa and D indicates that changes of D precede changes of Ppa. In contrast, the phase relation between Ppa and L was positive, which indicates that changes of Ppa preceded L. The combined effect of these phase differences between Ppa, D, and L was that Ppa and V were in phase.

The percent difference of C_{DL} from C_V is plotted against the phase difference between L and D at cardiac f in Fig. 4. When there is no phase difference between the oscillations in L and D, C_{DL} was <3% smaller than C_V. With increasing phase differences between D and L, C_{DL} overestimates C_V.

Figure 5 compares Ep_D, Ep_L, Ep_V, and C_V at respiratory (0.22 Hz) and cardiac f (~2.2 Hz). Although there was a tendency for Ep_D, Ep_L, and Ep_V to vary with f, these differences did not attain statistical significance. Although Ep_D increased with PEEP ($P < 0.05$), it was greater at respiratory than at cardiac f.

The phase relations between Ppa and D, Ppa and L, and Ppa and V at both respiratory and cardiac f are shown in Table 2. There were no significant differences between respiratory or cardiac f at any level of PEEP. Changes of Ppa preceded D at respiratory f, but changes of D preceded Ppa at cardiac f ($P < 0.001$). Changes of

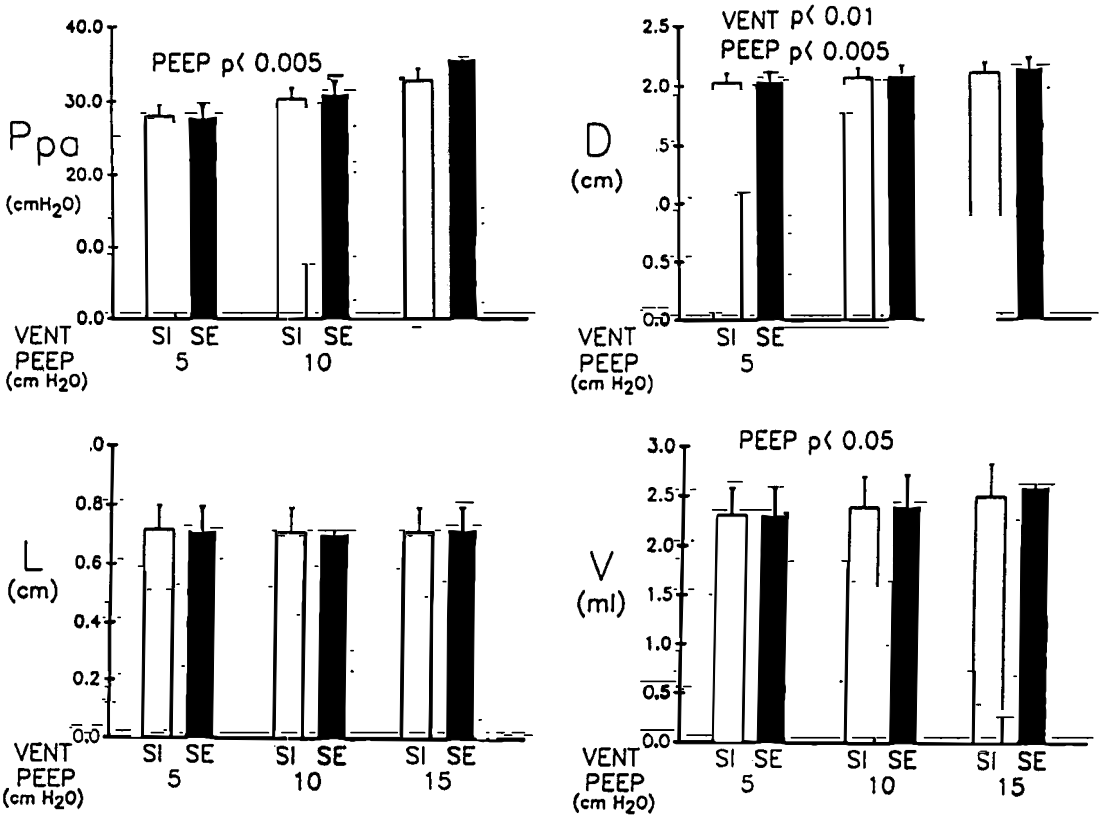

FIG. 2. Mean Ppa, D, L, and V of segment of MPA during ventilatory cycle. Significant differences between start of inspiration (SI) and start of expiration (SE) are indicated by VENT and its P value. Significant differences among 3 levels of positive end-expiratory pressure (PEEP) are indicated by PEEP and its P value. Bars, means ± standard error.

Ppa preceded L at cardiac f and differed from the phase relation at respiratory f ($P < 0.05$). The phase relation between Ppa and L at respiratory f showed marked variation and was not significantly different from zero. There were differences between respiratory and cardiac f for the phase relation between Ppa and V, but those differences varied inconsistently with the level of PEEP. The phase relation between Ppa and V was not significantly different from zero at either respiratory or cardiac f.

DISCUSSION

Comparison of our results to others. Measurements of D and L of a segment of the MPA have been made previously in vivo but not simultaneously with sonomicrometry. Patel et al. (12) found that pressure-diameter relations were linear between 10 and 60 cmH_2O. When their results are expressed in equivalent terms, they measured Ep_D, Ep_L, and Ep_V to be 204, 137, and 44 cmH_2O, respectively, at 4 cmH_2O of PEEP (10). Their results are similar to our data of 175 ± 27, 147 ± 27, and 55 ± 7 cmH_2O, respectively, at 5 cmH_2O of PEEP. There were distinct differences in the D and L waveform. They did not describe any presystolic wavelets that we encountered. They used calipers that were fixed to the operating table and may have restricted movement of the vessel. Our ultrasonic crystals are sutured to the adventicia of the MPA and are free to move independently of each other.

Measurement of C of a segment of MPA. We found that when D and L oscillations are in phase, the differences between the indirect (C_{DL}) and direct (C_V) calculation of pulmonary arterial C are <3%. The analysis given in the APPENDIX shows that use of the mean values of D and L to calculate Ep_D and Ep_L avoids the problems associated with higher-order terms (2). Nevertheless, we found that C_{DL} overestimates C_V when there are phase differences between D and L. This result emphasizes that simultaneous measurements of the changes of D and L are needed to calculate changes of vascular V.

Implicit in these calculations is the assumption that the pulmonary artery is circular in cross section at our site of measurement. Previous work by others (6) suggests that the median ratio of the major and minor axes of the pulmonary artery is 0.94 (4). They found the same changes of both major and minor axes with increment changes of pressure (6). Therefore we believe that treating the segment of the MPA as a cylinder is a reasonable approximation.

Effects of PEEP on dimensions and elasticity. PEEP resulted in small increases in mean D and V but no discernable effect on mean L. PEEP increased Ep_D but decreased Ep_L. The net result of these contrasting effects was no significant change of Ep_V with PEEP. The cause of these diverging effects is unclear; PEEP may have altered the geometry of the MPA due to the increase in lung volume changing its position.

Frequency dependence of pulmonary arterial elasticity and C. Although not central to our investigation of time-varying C, this study provided a comparison between pul-

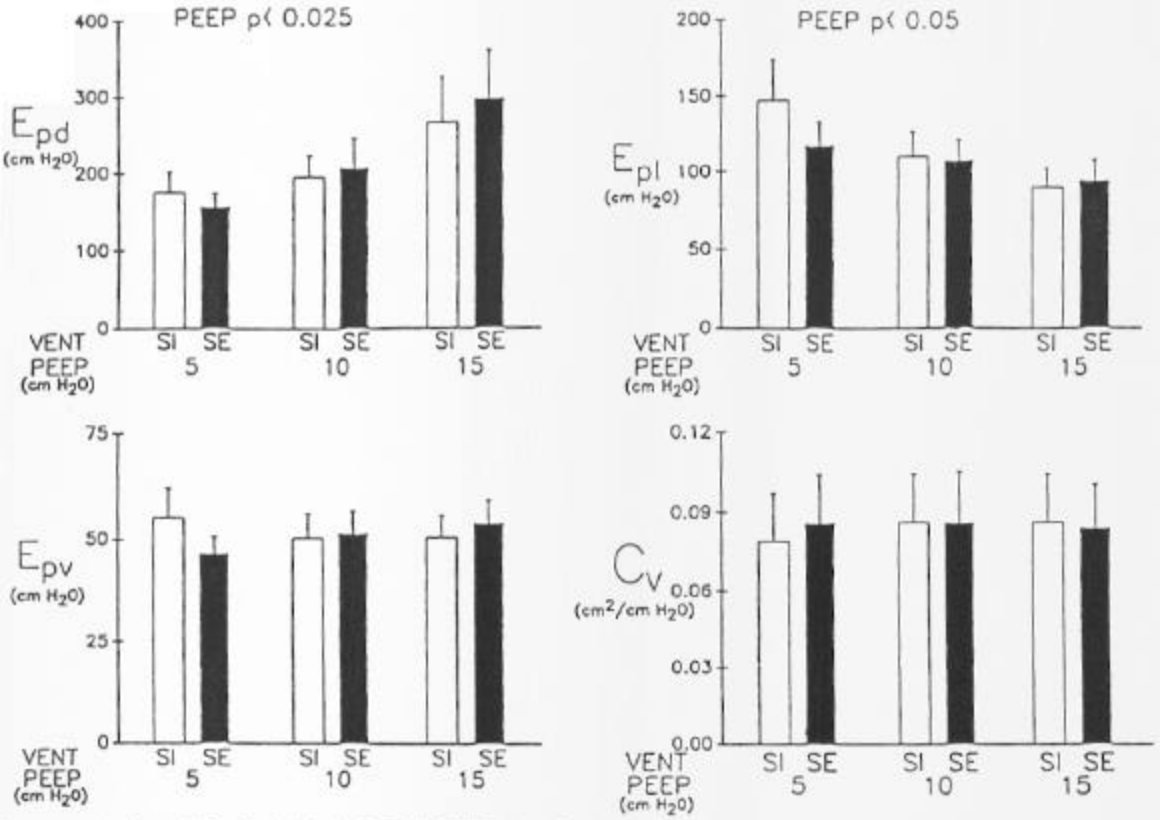

FIG. 3. Pressure strain modulus for D (Ep_D), L (Ep_L), V (Ep_V), and compliance (C_V) of segment of MPA measured at cardiac frequency during ventilatory cycle. There were no significant differences between SI and SE. Significant differences among 3 levels of PEEP are indicated by PEEP and its P value. Bars, means ± standard error.

TABLE 1. *Phase relation between Ppa and D, Ppa and L, and Ppa and V at cardiac frequency during the ventilatory cycle*

PEEP	Ppa-D Relation, rad	Ppa-L Relation, rad	Ppa-V Relation, rad
5 cmH_2O			
SI	−0.36±0.39	0.27±0.32	−0.09±0.18
SE	−0.30±0.35	0.30±0.27	−0.02±0.21
10 cmH_2O			
SI	−0.33±0.43	0.27±0.16	−0.01±0.25
SE	−0.28±0.47	0.42±0.26	0.07±0.31
15 cmH_2O			
SI	−0.39±0.54	0.27±0.29	0.00±0.35
SE	−0.23±0.52	0.40±0.24	0.12±0.36
		P values	
Vent effect (n = 36)	NS	<0.001	NS
Nonzero SI (n = 36)	<0.001	<0.001	NS
Nonzero SE (n = 36)	<0.05	<0.001	NS

Values are circular means ± SD; n, no. of tests. Phase relations are between pressure and diameter (Ppa-D), pressure and length (Ppa-L), and pressure and volume (Ppa-V) of the segment of the main pulmonary artery. Positive phase angles (rad) indicate extent to which changes in Ppa lead oscillations of D, L, or V. PEEP, positive end-expiratory pressure; SI, start of inspiration; SE, start of expiration; Vent effect, statistical differences between SI and SE. Nonzero SI and SE indicate that mean phase angle is significantly different from zero at that particular stage of ventilation.

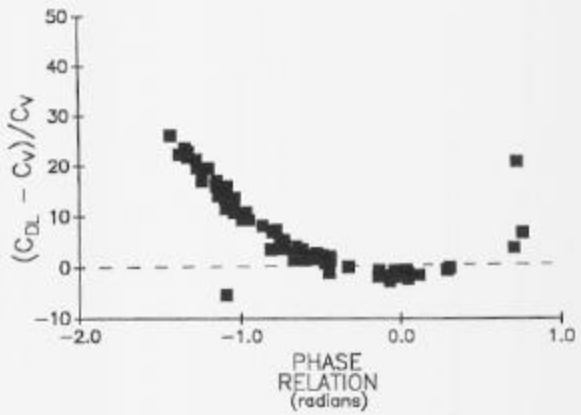

FIG. 4. Difference between compliance (C) calculated from D and L (C_{DL}), with C calculated from instantaneous V (C_V). This difference is expressed as percentage of C_V and plotted against phase difference between L and D changes.

monary arterial elasticity and C measured at respiratory f with corresponding measurements at cardiac f. There was a significant difference between measurements made at cardiac and respiratory f for Ep_D. In a previous study, we found that Ep_D was greater at respiratory than cardiac f (7). In that study, lung tissue may have impinged on the external surface of the MPA and impeded radial dilatation. As a result, Ep_D was elevated. Spectral analysis of Ep_D supports this possibility; the increase in Ep_D occurred close to respiratory f (7). In the present study, we were careful to prevent surrounding lung tissue from impinging on the pulmonary artery, particularly during PEEP. Although we did not encounter f dependence of Ep_D or Ep_L that attained statistical significance,

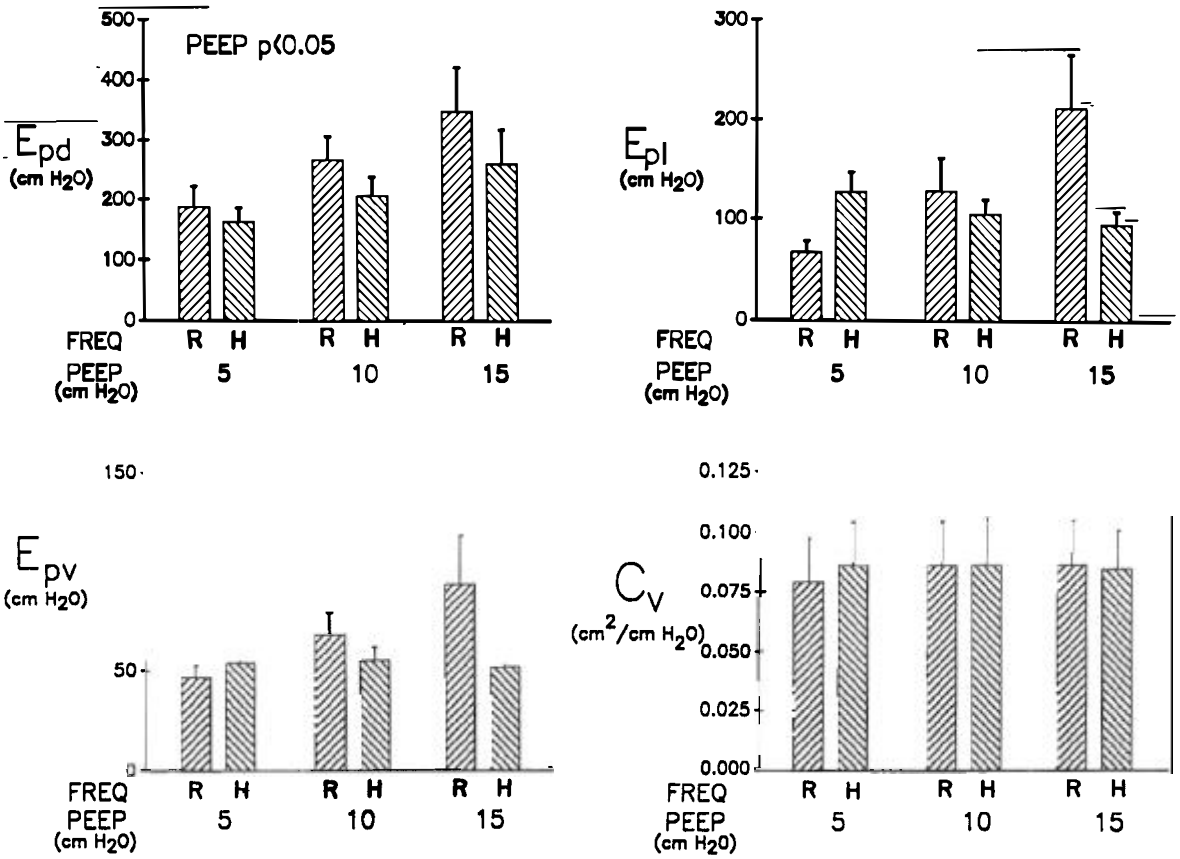

FIG. 5. Comparison of elastic moduli for D, L, V, and C of segment of MPA (Ep_D, Ep_L, Ep_V, and C_V, respectively) at respiratory (R) and cardiac (H) frequencies. None of differences in elastic moduli between R and H attained statistical significance. Significant differences between 3 levels of PEEP are indicated by PEEP and its P value. Bars, means ± standard error.

the pattern of results suggests that we may not have eliminated this possibility entirely.

Phase relations at cardiac f. The phase relation between Ppa and D was negative at cardiac f. This result indicates that changes of D precede changes of Ppa at that f. The phase relation between pressure and D depends on the interplay among three factors: vascular wall elasticity, wall viscosity, and longitudinal wall constraint (9). When the arterial wall is tethered, there is no longitudinal movement of the vascular wall. Figure 1 shows this case is not true for the MPA. It is unlikely that the MPA is freely mobile because the MPA is attached to some extent by the heart and lungs. Changes of D lag changes in pressure in a tethered viscoelastic tube. There are at least two circumstances when changes of D precede changes of transmural pressure (9). First, it occurs when the tube has pure elastic properties with no viscous component, regardless of the degree of wall constraint due to tethering by perivascular tissue (13). Second, it can occur even in a viscoelastic tube when there is some intermediate degree of wall constraint, as seems to be the case of the MPA (9). Radial dilatation precedes the change in local transmural pressure during increasing blood flow because more blood enters the vascular segment than leaves it. The fact that radial dilatation precedes pressure should not be too surprising because changes of flow precede pressure at cardiac f in the MPA.

Alternatively, changes of D could precede Ppa if external forces are imposed on the vascular wall by cardiac motion (11). Our experiments cannot distinguish b tween these two possibilities. The presystolic wavel may result from cardiac motion. The timing of the p

TABLE 2. *Phase relations between Ppa and D, Ppa and L, and Ppa and V at respiratory and cardiac frequenci*

PEEP	Ppa-D Relation, rad	Ppa-L Relation, rad	Ppa-V Relation,
	Respiratory frequency		
5 cmH_2O	0.55±0.96	−0.38±0.87	0.03±0
10 cmH_2O	0.60±1.10	−0.58±1.06	−0.12±1
15 cmH_2O	0.54±0.66	−0.22±0.97	0.22±0
	Cardiac frequency		
5 cmH_2O	−0.33±0.58	0.51±0.52	−0.03±0
10 cmH_2O	−0.19±0.46	0.26±0.19	−0.03±0
15 cmH_2O	−0.33±0.53	0.35±0.37	0.08±0
		P values	
Nonzero R (n = 34)	<0.001	NS	NS
Nonzero H (n = 35)	<0.05	<0.001	NS
Freq effect (n = 35)	<0.001	<0.05	0.001

Values are circular means ± SD. Phase relation is the extent to wh oscillations of Ppa lead oscillations of D, L, or V at either respirat frequency (R) or cardiac frequency (H) in rad. Freq effect, statisti differences between phase angles at R and H. Nonzero R and H in cate that the mean phase angle is significantly different from zero that particular frequency.

systolic wavelets is associated with atrial contraction but with only a small change of Ppa (Fig. 1). The cause of these wavelets is unknown. Echocardiographers have recorded presystolic pulmonary valve motion due to atrial contraction (1). It is possible that this motion generates a transient distortion of the MPA. Therefore cardiac motion or surrounding tissue could alter the measured values of elasticity and C. In these circumstances, C_V still reflects the C of the MPA segment, but the C may not be attributed solely to the properties of the vascular wall.

Although there were phase differences between pressure and D and between pressure and L, the phase relation between pressure and V was not different from zero at either cardiac or respiratory f. The zero phase relation at respiratory f is of interest because a time-varying C of the pulmonary arteries may result in a hysteresis of the Ppa-V relation during the ventilatory cycle. Hysteresis would cause a phase shift between pressure and V that would result in a nonzero phase relation.

Time invariance of pulmonary arterial C. Not only were there no phase differences between pressure and V during the ventilatory cycle, but we also found no difference in C_V at cardiac f between SI and SE. The experimental preparation was similar to our previous study (5), and the same method was used to identify SI and SE. Despite these conditions, we did not find any evidence that the C of the MPA was time varying during the ventilatory cycle. To the extent that the C of the MPA reflects total pulmonary arterial C, one of the following conditions should occur if C varied during the ventilatory cycle: either C_V is greater at SE than SI or the elasticity of the MPA segment is less at SE than SI. The increase of D that occurred due to ventilation was small and did not result in any significant change of V. In light of this result, it seems highly improbable that the changes of pulmonary arterial C are related to time-varying properties of the pulmonary arterial wall.

Another potential mechanism was the shifts of blood volume between the extra-alveolar and alveolar vessels during the ventilatory cycle. At SE, the alveolar capillaries are expanding from a compressed state due to positive pressure inflation. In contrast, the extra-alveolar arteries are decreasing in volume as lung volume decreases during expiration. This action would create longitudinal pressure gradients along the pulmonary vasculature that vary during the ventilatory cycle. Therefore there will be a transfer of blood from the extra-alveolar to the alveolar vessels. This transfer of blood volume would effectively increase pulmonary arterial C due to changes in the longitudinal pressure gradient. It is possible that the longitudinal gradients did not affect the pressure-V relationship of the segment of the MPA. The pressure changes were alterations of transmural pressure rather than changes of pressure in the longitudinal axis.

A more likely explanation is that the C of the MPA in this preparation does not reflect the C of the pulmonary arterial tree. The segment of MPA contained an average V of ~2.5 ml (Fig. 2), which is <5% of the estimated pulmonary arterial blood volume for dogs of this size (8). The MPA is not only extra-alveolar but also extrapulmonary. Therefore the MPA is not subjected to the same extramural forces that the extra-alveolar intrapulmonary arteries are exposed to because the chest was open in this preparation.

In summary, we have made simultaneous measurements of the pressure, D, and L of a segment of the MPA in the anesthetized dog. We used this preparation to clarify the mechanism of the previously reported variation of pulmonary arterial C during the ventilatory cycle. No significant differences of the MPA C at cardiac f were found during the ventilatory cycle. Furthermore, there were no phase differences between changes of MPA segment pressure and V at respiratory f. We conclude that the changes of pulmonary arterial C during the ventilatory cycle cannot be attributed to time-varying properties of the pulmonary arterial wall. The lack of a time-varying C of the MPA may be related to its extrapulmonary location because it is not subject to the fluctuations of extramural pressure that the extra-alveolar intrapulmonary arteries sustain during the ventilatory cycle when the chest is open.

APPENDIX

Recently, Gentile et al. (2) pointed out errors that can occur if elasticity for V is calculated from the elasticity of D and L. This approach may neglect second- and third-order terms. As a result, elasticity for V is underestimated by ~10% (2). Like Patel et al. (10), they used the end-diastolic value of D, L, and V in their calculation of elasticity. In contrast, we used the mean value, which results in different second- and third-order terms. The dependence of this error on the choice of value of D used to normalize the changes of D are shown algebraically.

The segment of the MPA is considered to be cylindrical with an instantaneous L and radius (R). The mean value of both R and L is denoted by a bar, diastolic value by subscript, d, and systolic value by subscript, s. $\bar{R}$ is assumed to be the average of R_s and R_d. ΔR is the change of R between R_s and R_d. The total change of the L is defined as ΔL

$$R_s = \bar{R} + \Delta R/2 \tag{A1}$$

$$R_d = \bar{R} - \Delta R/2 \tag{A2}$$

Similarly

$$L_s = \bar{L} + \Delta L/2 \tag{A3}$$

$$L_d = \bar{L} - \Delta L/2 \tag{A4}$$

Since

$$\bar{V} = \pi \bar{R}^2 \bar{L} \tag{A5}$$

$$V_s = \pi R_s^2 L_s \tag{A6}$$

By substituting in *Eq. A6* for R_s and L_s, we obtained the following expression

$$V_s = \pi(\bar{R} + \Delta R/2)^2(\bar{L} + \Delta L/2)$$

Similarly

$$V_d = \pi(\bar{R} - \Delta R/2)^2(\bar{L} - \Delta L/2) \tag{A8}$$

The total change of volume is ΔV

$$\Delta V = V_s - V_d \tag{A9}$$

By substituting in *Eq. A9* for V_s and V_d, we obtained the following expression

$$\Delta V = \pi[2\bar{R}\bar{L}\Delta R + \bar{R}^2 \Delta L + 0.25(\Delta R)^2 \Delta L] \tag{A10}$$

Therefore

$$\Delta V/\bar{V} = 2\Delta R/\bar{R} + \Delta L/\bar{L} + 0.25(\Delta R/\bar{R})^2 \Delta L/\bar{L} \tag{A11}$$

This equation should be compared with the expression that is obtained if R_d and L_d are used to normalize ΔR and ΔL (*Eq. 4* of Ref. 1)

$$\Delta V/\bar{V} = 2\Delta R/R_d + \Delta L/L_d + \Delta R^2/R_d^2 + 2(\Delta R/R_d)\Delta L/L_d + (\Delta R/R_d)^2\Delta L/L_d \quad (A12)$$

Compared with *Eq. A12, Eq. A11* has no second-order term, and the third-order term is <25% of the corresponding term in *Eq. A12.*

This last term in *Eq. A12* can be estimated in terms of the mean R and L

$$(\Delta R/R_d)^2\Delta L/L_d = (\Delta R/\bar{R})^2\Delta L/\bar{L}(o) \quad (A13)$$

where o is a numerical parameter that is always larger than unity if the changes in R and L are smaller than their mean values.

There is no correct way to calculate vascular elasticity. We chose to use mean rather than the end-diastolic dimension because it avoids the problems associated with higher-order terms.

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