Effective arterial elastance as an index of pulmonary vascular load.

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Abstract

The aim of this study was to test whether the simple ratio of right ventricular (RV) end-systolic pressure to stroke volume, known as the effective arterial elastance (Ea), provides a valid assessment of pulmonary arterial load in case of pulmonary embolism- or endotoxin-induced pulmonary hypertension. Ventricular pressure-volume data (obtained with conductance catheters) and invasive pulmonary artery pressure and flow waveforms were simultaneously recorded in 2 groups of 6 pure pietran pigs submitted either to pulmonary embolism (group A) or endotoxic shock (group B). Measurements were obtained at baseline, and each 30 minutes after injection of autologous blood clots (0.3 g/kg) in the superior vena cava in group A and after endotoxin infusion in group B. Two methods of calculation of pulmonary arterial load were compared. On one hand, Ea provided by using three-element windkessel model of the pulmonary arterial system [Ea(WK)] was referred to as standard computation. On the other hand, similarly to the systemic circulation, Ea was assessed as the ratio of RV end-systolic pressure to stroke volume [Ea(PV) = Pes/SV]. In both groups, although the correlation between Ea(PV) and Ea(WK) was excellent over a broad range of altered conditions, Ea(PV) systematically overestimated Ea(WK). This offset disappeared when left atrial pressure (Pla) was incorporated into Ea [Ea*(PV) = (Pes-Pla)/SV]. Thus, Ea*(PV), defined as the ratio of RV end-systolic pressure - minus left atrial pressure - to stroke volume, provides a convenient, useful and simple method to assess the pulmonary arterial load and its impact on the RV function.

Keywords: hemodynamics, pulmonary hypertension, right ventricle, ventriculo-arterial coupling
**Introduction**

In current clinical practice, pulmonary arterial load [or right ventricular (RV) afterload] is usually expressed as the mean pulmonary vascular resistance, computed as the ratio of the pressure drop through the pulmonary circulation [difference between mean pulmonary arterial pressure (PAP\text{mean}) and left atrial pressure (Pla)] to the mean pulmonary blood flow (CO). Such an evaluation ignores the pulsatile nature of both pressure and flow. Although oscillatory components of the pulmonary arterial load are low and mean resistance may be a valuable index of the pulmonary vascular load, the pulsatile nature of the load may be prominent in numerous pathological situations. Wave reflections play an important role and should be taken into account in pulmonary hypertension resulting from several pathological conditions like pulmonary embolism and septic shock (1, 2, 5, 14). In this way, the pulmonary artery impedance spectrum, which is defined in the frequency domain, provides a more precise and complete description of the pulmonary vascular load (12, 17). However, because of its complexity, this approach is difficult to use in clinical practice and to link with data obtained in the time domain. Sunagawa introduced the concept of the effective arterial elastance (Ea), defined as a steady-state arterial parameter that incorporates the principal elements of the windkessel model of the pulmonary vascular bed (24, 25). According to this concept, Ea is computed by combining the pulmonary vascular compliance, the characteristic impedance, and the resistance of the main pulmonary vessels as well as the pulmonary peripheral resistance. An alternative method consists in assessing Ea by the steady-state ratio of end-systolic pressure to stroke volume (Pes/SV). This ratio can be simply obtained from steady-state ventricular pressure-volume (PV) measurements while the pulmonary impedance spectrum or the windkessel parameters requires simultaneous pulmonary arterial flow and pressure waveforms acquisition. Moreover, the ratio of end-systolic ventricular elastance (Ees) to Ea, obtained from PV relations, characterizes the ventriculo-arterial interaction. In the systemic circulation, both normal and hypertensive human subjects show good agreement between Ea computed using left ventricular PV loop and Ea calculated from windkessel model parameters value recorded in the aorta (10). In the pulmonary circulation, it remains unknown whether or not Ea(PV) can be used as a substitute of Ea(WK) [“WK” denotes Ea derivation from windkessel model]. Therefore, the purpose of the present study was to assess the validity of Ea(PV) derived from steady-state RV PV data in experimental animals insulted with endotoxin or submitted to clot embolism.
Methods

All experimental procedures and protocols used in this investigation were reviewed and approved by the ethical committee of the Medical Faculty of the University of Liege and conformed to the Guide for the Care and Use of Laboratory Animals published by the US National Institutes of Health (NIH Publication No. 85-23, revised 1996). Experiments were performed on 2 groups of 6 healthy pure pietran pigs of either sex weighing from 16 to 28 kg. The animals were premedicated with intramuscular administration of ketamine (20 mg/kg) and diazepam (1 mg/kg). Anaesthesia was then induced and maintained by a continuous infusion of sufentanil (0.5 µg/kg/h) and pentobarbital (5 mg/kg/h). Spontaneous movements were prevented by pancuronium bromide (0.2 mg/kg/h). After endotracheal intubation via a cervical tracheostomy, the pigs were connected to a volume-cycled ventilator (Evita 2, Drager, Lubeck, Germany) set to deliver a tidal volume of 10 ml/kg at a respiratory rate of 20/min with a FiO₂ of 0.4. End-tidal CO₂ measurements (Capnomac, Datex, Helsinki, Finland) were used to monitor the adequacy of ventilation. Respiratory settings were adjusted to maintain end-tidal CO₂ between 30 and 35 mmHg. The pulmonary trunk was exposed via a median sternotomy. A micromanometer-tipped catheter (Sentron pressure measuring catheter, Cordis, Miami, FL, USA) was inserted into the main pulmonary artery through a stab wound in the RV outflow tract. A 14 mm diameter perivascular flow-probe (Transonic Systems, Ithaca, NY, USA) was placed around the main pulmonary artery 2 cm downstream to the pulmonary valve. The micromanometer-tipped catheter was manipulated so that the pressure sensor was finally positioned at the level of the flow-probe. Pla was measured with a micromanometer-tipped catheter inserted into the cavity through the left atrial appendage. Systemic arterial blood pressure was monitored via a micromanometer-tipped catheter inserted into the abdominal aorta through the left femoral artery. A 7F, 12-electrode (8-mm interelectrode distance) conductance micromanometer-tipped catheter (CD Leycom, Zoetermeer, The Netherlands) was inserted through the RV infundibulum into the right ventricle and positioned so that all electrodes were in the RV cavity. A 6F Fogarty balloon catheter (Baxter Healthcare Corp., Oakland, CA) was advanced into the inferior vena cava through a right femoral venotomy. Inflation of this balloon produced a gradual preload reduction.

Experimental protocol

After surgical preparation, the animals were allowed to stabilize for 30 min. Baseline hemodynamic recording was performed including PAPₘᵉᵃⁿ, pulmonary blood flow (Q), Pla, mean arterial blood
pressure, and heart rate (HR). In the first group (group A), autologous blood clots (0.3 g/kg) were injected in the superior vena cava immediately after baseline measurements. In the second group (group B), the animals had an intravenous infusion of 0.5 mg/kg of a freshly prepared endotoxin solution (lipopolysaccharide from E. coli serotype 0127:B8, Sigma, St Louis, MO, USA) over 30 min at T0.

**Data collection**

All analog signals were continuously digitalized with an appropriate system (Codas, DataQ, Akron, OH, USA). The pulmonary pressure and flow waves were sampled at 200 Hz and stored. Cardiac cycles were defined by R wave detection provided by a permanent recording of a one lead electrocardiogram. Ten consecutive cycles were recorded during apnea and were numerically averaged to obtain representative diagrams of pressure and flow waves corresponding to specific experimental conditions. Simultaneously, ten consecutive RV PV loops were recorded. The same measurements were then repeated during transient occlusion of the inferior vena cava using the Fogarty balloon.

**Data analysis**

We used a lumped parameter model, i.e. the three-element windkessel model (WK3), in order to analyze the flow conditions in the pulmonary circulation throughout the experimental protocol (figure 1). The 10 steady-state beats were analyzed under each condition (baseline, each 30 minutes after pulmonary embolism in group A or endotoxin infusion in group B), and the results were averaged. Maximal and minimal pulmonary arterial pressure defined systolic and diastolic pressures, respectively. Systolic ejection interval (ts) was measured from the foot of the pulmonary arterial pressure wave to its incisura, and the diastolic interval was td = T - ts, where T is the cardiac length. Pairs of pressure and flow data for each beat were analyzed and the three elements of the model were simultaneously calculated by using an original analytic procedure as described previously (12). RV PV loops were obtained using the conductance catheter method (3). End-systolic pressure (Pes) was the pressure at maximal ventricular elastance for a steady state loop. Maximal RV elastance was determined as 

\[
\left[\frac{P(t)}{V(t) - V_0}\right]_{\text{max}}
\]

where P(t) and V(t) are instantaneous RV pressure and volume respectively, Vo is the volume intercept of the end-systolic PV relation (ESPVR) obtained using the preload reduction method (3).

**Ea definitions**
An expression of the effective arterial elastance with the three parameters of the WK3 is given by (12, 21, 24):

\[
Ea(WK) = \frac{RT}{ts + \tau (1 - e^{-\frac{td}{\tau}})}
\]  

(Eq. 1)

where RT is the total pulmonary vascular resistance, i.e. the sum of the characteristic resistance (R₁) and the pulmonary arteriolar resistance (R₂) and \( \tau \) is the diastolic pressure decay time constant or the product of R₂ and the total capacitance (C) which represents the compliant properties of the pulmonary arterial tree. The electrical representation of the WK3 is displayed in figure 1. The three elements of the model are calculated using an analytical procedure (12, 14). Details of this analysis are provided in Appendix A.

Eq. 1 can be simplified as follows: If the diastolic pressure decay time constant is long compared with the diastolic time interval (\( \tau >> td \)), then the denominator reduces to \( ts+td=T \), the cardiac cycle length. Thus

\[ Ea(WK) = \frac{RT}{T} \]  

(Eq. 2)

However,

\[ RT = \frac{(PAP_{\text{mean}} - Pla)}{CO} \]  

(Eq. 3)

Therefore,

\[ Ea(WK) = \frac{(PAP_{\text{mean}} - Pla)}{(CO\cdot T)} = \frac{(PAP_{\text{mean}} - Pla)}{(SV\cdot HR\cdot T)} = \frac{(PAP_{\text{mean}} - Pla)}{SV} \]  

(Eq. 4)

If PAP\(_{\text{mean}}\) is further approximated by RV end-systolic pressure (Pes), then

\[ Ea(WK) = \frac{(PAP_{\text{mean}} - Pla)}{SV} \approx \frac{(Pes - Pla)}{SV} = Ea^*(PV) \]  

(Eq. 5)

If the downstream pressure can be neglected as in the systemic circulation, then

\[ Ea(WK) \approx \frac{Pes}{SV} = Ea(PV) \]  

(Eq. 6)

**Statistical analysis**

For each group, we performed a linear regression between Ea(WK) and Ea(PV) and we completed the analysis with a Bland-Altman test (Statistica version 7, StatSoft). Changes in hemodynamics and WK3 parameters were evaluated by a repeated-measures analysis of variance (Statistica version 7, StatSoft). Data are expressed as mean ± standard error of the mean (SEM).

**Results**

*Windkessel parameters and PV loops effects of arterial load variations.*
The effects of induced pulmonary hypertension in both groups on HR, cardiac output, mean arterial pressure, PAP$_{\text{mean}}$, Pla and right atrial pressure (Pra) are shown in table 1. In group A, pulmonary embolism was responsible for several alterations in the shape of pressure and flow waves including early pressure inflection, late systolic peak pressure and sharper pulmonary artery flow waveform (figure 1). The corresponding PV loops became oblong due to a rise in ejection pressure (figure 2, left panel). In group B, administration of endotoxin induced a bulging of PV loops (figure 2, right panel). These modifications were related to both reduced arterial compliance and enhanced wave reflections. Clot embolism and infusion of endotoxin lead to significant changes in the windkessel parameters, as shown in figure 3. In group A (figure 3, left panel), R$_1$ did not change significantly, however there was a rapid rise in R$_2$ and a fall in C after pulmonary embolism. In group B (figure 3, right panel), both R$_1$ and R$_2$ significantly increased following endotoxin insult, while C progressively decreased after endotoxin insult.

**Ea**(PV) compared to **Ea**(WK)

The time-course of both Ea**(PV)** and Ea**(WK)** in each group is shown in figure 4. In group A, Ea**(PV)** and Ea**(WK)** increased rapidly after the first vena cava injection of embol. In group B, both parameters progressively increased after endotoxin infusion. In each group, the pulmonary arterial elastance calculated with the complex method (Ea**(WK)**) and the simple method (Ea**(PV)**) displayed a parallel evolution.

Despite the varying vascular properties due to either pulmonary embolism or endotoxin infusion, the ratio of ventricular end-systolic pressure to stroke volume [Pes/SV = Ea**(PV)**] was remarkably similar to Ea**(WK)** derived from the windkessel parameters using Eq. 1. However, Ea**(PV)** was somewhat higher than Ea**(WK)**. The offset between Ea**(PV)** and Ea**(WK)** was significantly reduced by using Ea**(PV)** instead of Ea**(PV)** (figure 4). Indeed, incorporating Pla into Ea**(PV)**, the mean difference between both methods decreased from $0.24 \pm 0.07$ to $0.11 \pm 0.08$ mm Hg/ml (mean ± SD) in group A and from $0.29 \pm 0.11$ to $0.08 \pm 0.1$ mm Hg/ml (mean ± SD) in group B (figure 5).

The linear relations were nearly identical in both groups and given by Ea**(PV)** = 0.92 Ea**(WK)** + 0.1 ($r^2 = 0.96$, n = 56, SEE = 0.1, p < 0.0001) in group A and Ea**(PV)** = 0.88 Ea**(WK)** + 0.19 ($r^2 = 0.97$, n = 56, SEE = 0.21, p < 0.0001) in group B (figure 5). The limits of agreement indicated that the differences between both methods did not exceed 0.08 mm Hg/ml in group A and 0.11 mm Hg/ml in group B for 95% of the cases (figure 5).
**Ea(PV) and RT/T**

Like for the relation between Ea(PV) and Ea(WK), there was a strong linear correlation between Ea(PV) and RT/T: 
\[ \text{Ea(PV)} = 1.1 \times \text{RT/T} + 0.27 \quad (r^2 = 0.96, \ p < 0.0001, \ n = 58, \ \text{SEE} = 0.032) \] in group A and 
\[ \text{Ea(PV)} = 0.98 \times \text{RT/T} + 0.36 \quad (r^2 = 0.97, \ p < 0.0001, \ n = 56, \ \text{SEE} = 0.059) \] in group B. The bias was still reduced using Ea*(PV) instead of Ea(PV): 
\[ \text{Ea*(PV)} = 1.04 \times \text{RT/T} + 0.11 \quad (r^2 = 0.93, \ p < 0.0001, \ n = 58, \ \text{SEE} = 0.038) \] in group A and 
\[ \text{Ea*(PV)} = 0.95 \times \text{RT/T} + 0.23 \quad (r^2 = 0.96, \ p < 0.0001, \ n = 56, \ \text{SEE} = 0.051) \] in group B. The low SEE obtained in each group corresponded to a good agreement between both parameters (figure 6).

**Discussion**

In the present study, we tested whether or not pulmonary vascular load could be assessed by the effective arterial elastance determined by the simple ratio of end-systolic pressure to stroke volume [Ea(PV)]. Our results demonstrated that there was an excellent correlation between Ea(WK) calculated from the windkessel model and Ea calculated as Pes/SV over a wide range of loading conditions resulting from either pulmonary embolism or endotoxin insult. However, the effective arterial elastance determined by the simple ratio of end-systolic pressure to stroke volume consistently exceeded the elastance calculated from the windkessel parameters. The offset between Ea(PV) and Ea(WK) nearly vanished using Ea*(PV) - which incorporates Pla - instead of Ea(PV). This observation implies that, contrary to what is observed in the systemic circulation, the effect of the downstream pressure on the pulmonary circulation is not negligible. Sagawa et al. suggested to incorporate the presence of an effective downstream pressure into Ea (21). Nevertheless, Pla is frequently ignored in the pulmonary circulation and Ea is calculated as the ratio of Pes to SV similarly to the systemic circulation (4, 9, 18, 28).

The linear relations between Ea*(PV) and Ea(WK) were nearly identical in both groups. These results are concordant with those of Kelly et al. in the systemic circulation, who showed that Ea(PV) provides a useful method to assess arterial load and its interaction with the human ventricle (10). These authors suggested that Ea(PV) is a powerful tool to assess the effects of increased pulsatile load caused by aging or hypertension on PV loops. They also pointed out that mean arterial resistance often underestimates the real effects of the load on cardiac performance (10). Segers et al. found that Ea(PV) underestimated Ea(WK) provided by the four-element windkessel model (23).
Our results demonstrated that $E_{a}^{*}(PV)$ can be used in place of $E_{a}(WK)$ in the pulmonary circulation. The correlation between $E_{a}^{*}(PV)$ and $E_{a}(WK)$ was excellent in normal condition as well as after pulmonary embolism or endotoxin infusion as shown by the Bland-Altman test in both groups. In group A, $E_{a}$ showed an asymptotic increase early after the pulmonary embolism in concordance with an acute loss of compliance and a rapid rise of the total resistance offered by the pulmonary vascular tree. In group B, $E_{a}$ progressively increased with an exponential trend associated with a progressive decrease in compliance and a slow rise in the total resistance of the pulmonary vasculature. Although experimental conditions were totally different, the linear correlations between both methods in each group were nearly similar.

Our data showed that pulmonary embolism or endotoxin insult led to a complex pulmonary vascular response involving a dynamic, time-dependent interplay between $R_{1}$, $C$ and $R_{2}$. Nevertheless, the correlation between both methods in each group remained excellent over the range of important variations in the windkessel parameters.

The ratio of maximum ventricular elastance on $E_{a}$ ($E_{max}/E_{a}$) is superior to 1 in the normal heart suggesting that the ventricle operates close to the optimal efficiency. We previously showed that in heart failure due to pulmonary embolism and sepsis, the decreased value of $E_{max}/E_{a}$ was related to an impaired use of energy by the failing heart (6, 13, 15). In combination with $E_{max}$, $E_{a}(PV)$ appears to be a simple way to characterize ventriculo-arterial interaction (18, 26, 27). For the systemic circulation, Segers et al. suggested that $E_{a}$ can be approximated by $RT/T$ only for high $C$ values (23). For the pulmonary circulation, our results evidenced significant correlation between both methods as well as between $E_{a}^{*}(PV)$ and $RT/T$, despite dramatic changes in pulmonary vascular compliance. We previously showed a concordant evolution between $E_{max}/E_{a}$ and stroke work in pulmonary embolism or septic shock (6, 13, 15). This could be explained by higher basal pulmonary vascular compliance compared with the values obtained on the systemic circuit.

The determination of $E_{a}$ as the simple ratio of end-systolic pressure - minus left atrial pressure - to stroke volume to assess pulmonary vascular load is rapidly and easily feasible in clinical settings. In contrast, $E_{a}(WK)$ requires invasive measurement of pulmonary flow and pressure waves, which limits its potential use (8, 19).

RV tolerance and adaptation to chronic or acute increase in pulmonary vascular load may be a cornerstone in the prognosis of patients suffering from pulmonary hypertension. Therefore, evaluation
of RV-pulmonary arterial coupling by using the ratio of contractility, assessed by the slope of the end-
systolic PV relationship, to Ea seems essential to evaluate correctly the facilitation of energy transfer
from the right ventricle into the pulmonary circuit (11). However, determination of Emax requires
preload variation that is difficult to apply in clinical practice. It is the reason why single beat methods
have been developed but unfortunately not yet validated for the right ventricle. Therefore further
studies should be encouraged (16, 22). As pathophysiological RV conditions are often associated with
valve insufficiencies, SV was derived from pulmonary arterial flow divided by HR.

Some study limitations should be acknowledged. Ea(WK) and Ea*(PV) were related through two
assumptions (Eq. 2 to 6): Diastolic time constant \( t = R_2 C \) is long relative to the diastolic time period
\( t_d \) and Pes is approximately equal to PAP\(_{\text{mean}}\). In comparison with the systemic vasculature, lower
pulmonary vasculature resistance is counterbalanced by higher pulmonary vascular compliance in
such a way that the first assumption can be considered as valid in basal conditions. In pulmonary
hypertension, rise in \( R_2 \) prevails on loss in \( C \), so the first assumption holds. Because of lower
pulmonary arterial pressure levels, discrepancy between Pes and PAP\(_{\text{mean}}\) is more important than in
the systemic vascular tree. However, in pulmonary hypertension, due to higher pulmonary arterial
pressure levels and enhanced wave reflections occurring during systole, PAP\(_{\text{mean}}\) tends to be nearer to
Pes.

In summary, several recent studies highlighted the importance of abnormal pulsatile load effect in
the mechanism of right heart failure (1, 2, 7, 20). Several methods to assess pulmonary vascular load
have been proposed, but require a complete acquisition of pulmonary arterial pressure and flow
waveforms. As a result, such methods are difficult to apply in current clinical practice. In the present
study, we suggest that pulmonary arterial load can be simply assessed from the ratio of RV end-
systolic pressure - minus left atrial pressure - to RV stroke volume in the setting of pulmonary
hypertension. The downstream pressure plays an important role in the pulmonary circulation and
should be incorporated into the pulmonary effective arterial elastance.

**APPENDIX A: CALCULATION OF WK3 PARAMETERS**

The relationship between pressure and flow in the electrical representation of WK3 is described by the
following equation:

\[
P(t) + R_2 C \frac{dP(t)}{dt} = (R_1 + R_2)Q(t) + R_1 R_2 C \frac{dQ(t)}{dt}
\]  

(1)
where \( Q \) = pulmonary flow, \( P \) = pulmonary arterial pressure and \( t_0 \) = the beginning of the cardiac cycle defined as the R wave on the ECG. \( R_1, R_2 \) and \( C \) are the three elements of the WK3 (figure 1). Eq.1 is integrated and becomes:

\[
\int_{t_0}^{t} Q(\tau) d\tau = k_1 \int_{t_0}^{t} P(\tau) d\tau + k_2 (P(t) - P(t_0)) + k_3 (Q(t) - Q(t_0)) \tag{2}
\]

where

\[
k_1 = \frac{1}{R_1 + R_2}, \quad k_2 = \frac{CR_2}{R_1 + R_2} \quad \text{and} \quad k_3 = -\frac{CR_2}{R_1 + R_2} \tag{3}
\]

The multiple regression technique estimates the constants \( k_i \) in order to minimize the residual sum of squares (RSS) i.e., the sum of squared differences between the observed values of both parts of this equation

\[
RSS = \sum \left[ \int_{t_0}^{t} Q(\tau) d\tau - k_1 \int_{t_0}^{t} P(\tau) d\tau - k_2 (P(t) - P(t_0)) - k_3 (Q(t) - Q(t_0)) \right]^2 \tag{4}
\]

\( R_1, R_2 \) and \( C \) values are then derived by solving Eq.3.
Acknowledgements

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References


Figure Legends

**Figure 1.** Representation of the three element windkessel model with an example of computer record used to obtain averaged pulmonary pressure (upper panel) and flow (lower panel) waveforms from ten consecutive systoles in a typical group A pig. Key to symbols used: R1, characteristic vascular resistance; C, vascular compliance; R2, peripheral vascular resistance.

**Figure 2.** Example of RV pressure-volume loops in a group A pig (left panel) before (bold lines) and after (thin lines) pulmonary embolism and in a group B pig (right panel) before (bold lines) and after (thin lines) endotoxin infusion. Lines representing Ea (= (Pes-Pla)/SV) are shown for each pressure-volume loop (dotted lines).

**Figure 3.** Time-course of windkessel parameters (R1, R2, C) in group A (left panel) and in group B (right panel). (*p<0.05 compared with baseline)

**Figure 4.** Time-course of Ea(WK), Ea*(PV) and Ea(PV) in group A (left panel) and in group B (right panel). (#p<0.05 compared with baseline)

**Figure 5.** On the left upper panel: correlation between Ea*(PV) and Ea(WK): Ea*(PV) = 0.92 Ea(WK) + 0.1 (r² = 0.96, n = 56, SEE = 0.1, p < 0.0001) in group A and Ea*(PV) = 0.88 Ea(WK) + 0.19 (r² = 0.97, n = 56, SEE = 0.21, p < 0.0001) in group B. Bland-Altman test comparing Ea*(PV) and Ea(WK) in group A on the left lower panel and in group B on the right lower panel. The solid line is the mean difference; the dashed lines represent mean difference ± 2SD.

**Figure 6.** Correlation between Ea*(PV) and RT/T is given by the following linear relations: Ea*(PV) = 1.04 · RT/T + 0.11 (r² = 0.93, p < 0.0001, n = 58, SEE = 0.038) (group A) and Ea*(PV) = 0.95 · RT/T + 0.23 (r² = 0.96, p < 0.0001, n = 56, SEE = 0.051) (group B) on the left and right panel respectively.
Tables

*Table 1. Effects of induced pulmonary hypertension on heart rate, cardiac output, systemic and pulmonary arterial pressures, left and right atrial pressures.*

<table>
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<th>HR (beat/min)</th>
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<td>T0</td>
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<td>5.1 ± 0.2</td>
<td>82 ± 9</td>
<td>11 ± 3</td>
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<tr>
<td>PHT</td>
<td>114 ± 4</td>
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<td>p</td>
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<td>114 ± 2</td>
<td>4.8 ± 0.9</td>
<td>75 ± 13</td>
<td>15 ± 2</td>
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Values are mean ± SE. T0, baseline conditions; PHT, pulmonary hypertension; HR, heart rate; CO, cardiac output; MAP, mean arterial pressure; PAPmean, mean pulmonary arterial pressure; Pla, left atrial pressure; Pra, right atrial pressure.
three-element windkessel model

Baseline
After pulmonary embolism
Ea(WK)  
Ea*(PV)  
Ea(PV)  

0.2  0.4  0.6  0.8  1.0  1.2  1.4  1.6  2.0

time (min)  

Tables

Table 1. Effects of induced pulmonary hypertension on heart rate, cardiac output, systemic and pulmonary arterial pressures, left and right atrial pressures.

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<tr>
<td><strong>Group B</strong></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>T0</td>
<td>114 ± 2</td>
<td>4.8 ± 0.9</td>
<td>75 ± 13</td>
<td>15 ± 2</td>
<td>8 ± 2</td>
<td>8 ± 2</td>
</tr>
<tr>
<td>PHT</td>
<td>124 ± 3</td>
<td>4.3 ± 1</td>
<td>60 ± 8</td>
<td>25 ± 2</td>
<td>6 ± 2</td>
<td>22 ± 2</td>
</tr>
<tr>
<td>p</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
<td>&lt; 0.001</td>
<td>NS</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Values are mean ± SE. T0, baseline conditions; PHT, pulmonary hypertension; HR, heart rate; CO, cardiac output; MAP, mean arterial pressure; PAPmean, mean pulmonary arterial pressure; Pla, left atrial pressure; Pra, right atrial pressure.