Value and Limitations of Peak-to-Peak Gradient for Evaluation of Aortic Stenosis

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The transvalvular pressure gradient (\(\Delta P\)) is one of the major indices used to evaluate the severity of aortic stenosis (AS). The peak and mean \(\Delta P\) measured by Doppler echocardiography or by catheterization, and the peak-to-peak \(\Delta P_{\text{pp}}\) measured by catheterization, are used routinely for the evaluation of stenosis severity. A major limitation of \(\Delta P\) is that it is highly dependent on flow conditions. Moreover, it has frequently been reported that \(\Delta P\) may be reduced in patients with significant AS and concomitant systemic hypertension (1-4). This reduction was generally thought to depend on the simultaneous decrease in flow that may occur as a result of increased left ventricular (LV) afterload. However, Laskey et al. (2) suggested that \(\Delta P\) may decrease irrespective of flow as a direct consequence of the increased systemic vascular resistance (R). As opposed to these results, Razzolini et al. (5) found that, for each flow level, \(\Delta P\) increased linearly with R. In a recent animal study, it was observed that hypertension may cause an important reduction in \(\Delta P_{\text{pp}}\) measured by Doppler echocardiography. Multivariate analysis of these results suggested that the impact of hypertension on the peak and mean \(\Delta P\) was essentially related to changes in flow rate and valve effective orifice area (EOA), whereas the impact on the \(\Delta P_{\text{pp}}\) was also independently determined by the systemic arterial compliance (C). Moreover, the results of the animal study showed that the increase in systemic arterial load (equivalent to severe hypertension in humans) distal

Background and aim of the study: In patients with aortic stenosis (AS), it has been reported that the transvalvular pressure gradients (\(\Delta P\)) may be reduced or even abolished in the presence of concomitant arterial hypertension, but the mechanisms underlying this phenomenon remain unclear. The study aim was to: (i) examine the relationship between systemic arterial hemodynamics and the peak-to-peak \(\Delta P_{\text{pp}}\), peak \(\Delta P\) and mean \(\Delta P\); and (ii) propose and validate a new formula for the non-invasive estimation of the \(\Delta P_{\text{pp}}\) and of the peak left ventricular systolic pressure (LVSP) using Doppler echocardiography.

Methods: Two fixed stenoses (geometric orifice area 1.0 and 1.35 cm\(^2\)) and one bioprosthesis (effective orifice area (EOA) 1.2 cm\(^2\)) were tested in a mock flow circulation model. Systemic vascular resistance (R) was increased from 1,500 to 3,300 dyne·s/cm\(^5\), and systemic arterial compliance (C) was decreased from 2.9 to 0.9 ml/mmHg, while transvalvular flow was held constant.

Results: Neither C nor R had any significant impact on EOA, peak \(\Delta P\) and mean \(\Delta P\). \(\Delta P_{\text{pp}}\) was decreased markedly, however, when C was reduced (bioprosthesis: -15 mmHg (-69%); orifice 1.35 cm\(^2\): -24 mmHg (-30%); orifice 1.0 cm\(^2\): -15 mmHg (-13%)). Subsequently, an equation was proposed to predict \(\Delta P_{\text{pp}}\) from EOA, mean \(\Delta P\), and C measured by Doppler echocardiography. LVSP calculated by adding the predicted \(\Delta P_{\text{pp}}\) to systolic arterial pressure (SAP) was compared with LVSP measured directly in a dataset of 24 pigs with experimentally induced AS. There was a strong agreement between the estimated and measured LVSP (\(r = 0.97\); mean absolute error 5 ± 5 mmHg).

Conclusion: \(\Delta P_{\text{pp}}\) should not be used to evaluate AS severity because, as opposed to peak and mean \(\Delta P\), it is highly influenced by C. The new non-invasive method proposed in this study to estimate the LVSP may be useful for obtaining a more accurate estimate of global LV afterload in patients with AS.

The Journal of Heart Valve Disease 2006;15:609-616
to the valvular load (equivalent to severe AS in humans) resulted in a dramatic increase in peak LV systolic wall stress, despite a concomitant decrease in ΔPs. The reduction in ΔPs that may occur as a result of systemic hypertension in patients with AS should not be interpreted as reflecting a reduction of the load imposed on the left ventricle. On the contrary, the left ventricle of patients with AS and concomitant systemic hypertension is submitted to two additive loads: a valvular load and an arterial load. Accordingly, it was recently reported that reduced C is a frequent occurrence in patients with AS, and that it contributes independently to the increase in global LV afterload and to the occurrence of LV diastolic and systolic dysfunction in these patients (6).

These findings underline the importance of estimating global LV afterload in AS patients. The non-invasive measurement of indices of LV afterload, including LV systolic wall stress (4,7-9), or the valvular-arterial impedance recently proposed by the present authors' group (6), nonetheless requires an estimation to be made of the peak LV systolic pressure (LVSP). This is usually achieved by adding the systolic arterial pressure (SAP) measured by sphygmomanometry to the mean pressure (P) measured by Doppler echocardiography, because the mean ΔP is generally assumed to be equivalent to ΔP_{valv} (10).

The main limitation of previous in-vivo studies has been that it was difficult - or even impossible - independently to control and modify the different hemodynamic parameters (flow rate, C, R, aorta diameter, and valve EOA) involved in the complex interaction between ventricular, valvular and arterial hemodynamics. In an attempt to overcome this limitation, a numerical study and an in-vitro study were performed, using a mock flow circulation model. The study aims were to: (i) examine the relationship between systemic arterial hemodynamics (i.e., C and R) and ΔP_{valv}, peak ΔP and mean ΔP; and (ii) propose and validate a formula for accurate estimation of ΔP_{valv} and thus of the peak LVSP by Doppler echocardiography.

Materials and methods

Numerical study

A model based on the Windkessel two-element theory was used to describe the theoretical relationship between systemic arterial hemodynamics and ΔP:

$$\frac{dP}{dt} + \frac{P}{R \times C} = \frac{1}{C} Q$$

(1)

where P is the instantaneous aortic pressure, C is the systemic arterial compliance, R is the total vascular resistance, and Q is the transvalvular flow rate.

However, instead of using a constant transvalvular flow during the systolic phase (Q = cte), as suggested by the original Windkessel theory, the flow was simulated by the temporal relationship:

$$Q(t) = \begin{cases} 
Q_0 \sin\left(\frac{\pi t}{T_s}\right) & \text{for } 0 \leq t \leq T_s \\
0 & \text{for } T_s < t \leq T 
\end{cases}$$

(2)

where $Q_0$ is the maximal flow during systolic phase, T is the cardiac period, and T_s is the systolic duration.

As an initial condition to solve this problem, a condition of recurrence was used:

$$P_0 = P(0) = P(T)$$

It was therefore possible to derive analytically the aortic pressure (see Appendix I), and then to compute the LV pressure by adding ΔP. The instantaneous ΔP across the aortic valve could then be written using the expression introduced by Garcia et al. (11,12):

$$\Delta P(t) = \frac{p}{2 \cdot ELC_0^2} Q(t)^2 + \frac{2np}{\sqrt{ELC_0}} \frac{dQ}{dt}$$

(3)

where ELC_0 is the energy loss coefficient of the aortic valve (13,14), which depends on the valve EOA and the aortic cross-sectional area at the level of the sinotubular junction ($A_h$):

$$ELC_0 = \frac{A_h \cdot EOA}{A_h - EOA}$$

(4)

The expression of the ventricular pressure during the ejection phase is then:

$$P_e(t) = P(t) + \frac{p}{2 \cdot ELC_0^2} (Q_0 \sin(wt))^2 + \frac{2np}{\sqrt{ELC_0}} w Q_0 \cos(wt)$$

(5)

C was varied from 3.0 to 0.5 ml/mmHg and R from 1,332 to 3,330 dyne·s/cm^5 for three different degrees of AS (EOA = 0.5, 0.75, and 1.0 cm^2). For each of these conditions, the ventricular and aortic pressure waveforms were computed using Equations (5) and A1 (see Appendix I), respectively, and the theoretical values of ΔP_{valv}, peak ΔP and mean ΔP were then determined from these pressure waveforms.

In-vitro study

The pulse duplicator used in this study has been previously described and validated in detail (14-16). It comprises a fluid reservoir, a gear pump, and adjustable systemic arterial resistance and compliance. The pulsatile flow was provided by a computer-controlled direct-current motor coupled to a gear pump (Vi-Corr; Viking Pump, Cedar Falls, IA, USA). The LV
outflow tract and ascending aorta were both circular and rigid. The aortic compliance consisted of a rubber tube enclosed within a hermetic Plexiglas box, filled with water and air, and connected to an air vacuum/compressor.

The fluid used in the model was a mixture of water and glycerol (67/33, v/v), of viscosity 3.5 cp. The flow rate was measured using an electromagnetic flowmeter (Cliniflow II; Carolina Medical Electronics, King, NC, USA), and pressure measurements were performed using Millar catheters under a sampling frequency of 300 Hz. The LV pressure was measured 20 mm upstream of the valve, and the aortic pressure 100 mm downstream of the valve, in order to determine peak ($\Delta P_{\text{peak}}$), mean ($\Delta P_{\text{mean}}$), and peak-to-peak ($\Delta P_{\text{PPtoP}}$) transvalvular pressure gradients. The valve EOA was calculated using the following formula (16):

$$\text{EOA} = \left( \frac{1}{A_a} + \frac{2 \Delta P_{\text{mean}}}{\rho Q^2} \right)^{-1} \quad (6)$$

Systemic compliance was calculated using the two-area method introduced by Liu et al. (17). Systemic resistance was calculated using the ratio of mean aortic pressure to mean transvalvular flow rate.

**Experimental conditions**

Two fixed stenoses (plates with circular orifices with a geometric area of 1.0 and 1.35 cm²) and a Medtronic Mosaic 21-mm valve (EOA 1.2 cm²) were tested in the model. To simulate the different situations that might occur in hypertensive patients, $R$ was increased from 1,500 to 3,300 dyne·s/cm⁵ (four levels), while for each level of $R$ the $C$ was varied between 0.9 and 2.9 ml/mmHg (four levels). All experiments were performed at a heart rate of 60 bpm, with a LV ejection time of 300 ms and stroke volume of 83 ± 17 ml. The shape of the flow waveform was maintained constant ($Q = \text{Cte}$), as this may affect the $\Delta P$-values, regardless of mean flow rate.

The $\Delta P_{\text{PPtoP}}$ is difficult to determine using a simple mathematical expression, but was estimated using a non-linear regression analysis of the in-vitro results. The peak LVSP was then computed by summation of the $\text{SAP}$ and $\Delta P_{\text{PPtoP}}$ predicted from the regression equation.

**Animal study**

In order to validate the new mathematical expression proposed for the estimation of peak LVSP, a dataset obtained in 24 pigs with experimentally induced AS was used. Details of this animal study have been described previously (4). Briefly, a severe supravalvular AS was created in 24 pigs using an umbilical tape tightened around the aorta approximately 2 cm downstream of the aortic valve annulus. Systemic hypertension was induced either by banding of the distal thoracic aorta or by infusion of phenylephrine. The $\Delta P$ across the stenosis was measured with Doppler echocardiography and by catheterization, first at normal systemic arterial pressure, and subsequently during hypertension. Six of the 24 animals had two hypertension stages; hence, the protocol resulted in 54 data points. Some of these data were used in a previous publication (4).

Other investigators have also proposed estimating LVSP by adding peak $\Delta P$ or mean $\Delta P$ to the $\text{SAP}$ (10,18). Thus, three formulae were tested that could be used for non-invasive estimation of peak LVSP: $(\text{SAP} + \Delta P_{\text{peak}})$, $(\text{SAP} + \Delta P_{\text{mean}})$; and $(\text{SAP} + \Delta P_{\text{PPtoP}}$ predicted from Eq. (7)). The LVSP estimated by these formulae was compared with that measured directly by catheterization. In order to provide a more clinical perspective to these results, the valve EOA, peak $\Delta P$, mean $\Delta P$ and $C$ measured by Doppler echocardiography and sphygmanometry were used in these formulae (Table I).

**Statistical analysis**

Data were expressed as mean ± SD, and compared using a $t$-test. Statistical analysis of the association of variables was performed using the Pearson correlation

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Table I: Measurement methods of hemodynamic parameters required for non-invasive estimation of peak left ventricular systolic pressure.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Method</th>
<th>Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\text{SAP}$</td>
<td>Sphygmanometry</td>
<td>Continuity equation</td>
</tr>
<tr>
<td>$\text{EOA}$</td>
<td>Doppler echocardiography</td>
<td>Simplified Bernoulli equation</td>
</tr>
<tr>
<td>$\Delta P_{\text{mean}}$</td>
<td>Doppler echocardiography</td>
<td></td>
</tr>
<tr>
<td>$C$</td>
<td>Doppler echocardiography (SV) and sphygmanometry (PP)</td>
<td>$C = \text{SV}/\text{PP}$</td>
</tr>
</tbody>
</table>

$\Delta P_{\text{mean}}$: Mean transvalvular pressure gradient; $C$: Systemic arterial compliance; EOA: Effective orifice area; PP: Arterial pulse pressure; SAP: Systolic arterial pressure; SV: Stroke volume.
coefficient, and graphs were constructed using the corresponding regression equation. A p-value <0.05 was considered to be statistically significant.

**Results**

**Numerical study**

The numerical simulations performed using the theoretical model described in Eqs. (1), (2), (3) and (5) suggest that R and C have no direct effect on peak and mean ΔPs. These simulations also suggested that ΔP_{PP} was independently influenced by C, but not by R (Fig. 1). Figure 1 shows the normalized ΔP_{PP} (i.e., normalized with respect to the ΔP_{PP} value at the lowest level of C) as a function of C and R for three different degrees of AS (EOA = 0.5, 0.75, and 1.0 cm²). The ΔP_{PP} was markedly reduced when C decreased, and the reduction appeared to be relatively more important for less severe stenosis. For the 1.0-cm² stenosis, the ΔP_{PP} was almost completely abolished, falling from 21 to 7 mmHg, during which time C fell from 3.0 to 0.5 ml/mmHg.

**Figure 1:** Theoretical variation in normalized peak-to-peak transvalvular pressure gradient with respect to systemic arterial compliance (C) and systemic vascular resistance (R) for three different degrees of aortic stenosis severity (valve effective orifice area (EOA) = 0.5, 0.75, and 1.0 cm²).

**Figure 2:** Effect of systemic arterial compliance (C) and systemic vascular resistance (R) on peak and mean transvalvular pressure gradients (ΔPs) (panels A and C) and effective orifice area (EOA) (panels B and D). Panels A, B: Systemic vascular resistance (R) was maintained constant at 1,217 ± 333 dyne·s/cm⁵, and C was varied from 0.9 to 2.9 ml/mmHg. Panels C, D: Compliance (C) was maintained constant at 1.42 ± 0.51 ml/mmHg, and R was varied from 1,500 and 3,300 dyne·s/cm². ●, circular orifice 1.0 cm²; ▼, circular orifice 1.35 cm²; ■, Medtronic Mosaic 21-mm valve. Closed symbols indicate peak ΔP; open symbols indicate mean ΔP.
In-vitro study

The effects of C and R on peak and mean ∆Ps and valve EOA obtained in in-vitro experiments are illustrated in Figure 2 (panels A and B, respectively). As predicted by the theoretical model, C and R had no significant effect on peak and mean ∆Ps, or on valve EOA. In contrast, changes in C had a major influence on ∆PPtoP (Fig. 3); when C was reduced from 2.9 to 0.9 ml/mmHg, the ∆PPtoP fell by 15 mmHg (-69%) in the Mosaic 21-mm bioprosthetic valve, by 24 mmHg (-30%) in the circular rigid orifice 1.35 cm², and by 15 mmHg (-13%) in the circular rigid orifice 1.0 cm². As predicted by the theoretical results, the effect of C on ∆PPtoP was more pronounced in mild/moderate AS than in severe AS.

Animal study

The animal data were used to test the three formulae described above. When compared to peak LVSP measured by catheterization, the mean absolute errors were 27 ± 16 mmHg for the formula (SAP + ∆Ppeak), 13 ± 9 mmHg for the formula (SAP + ∆Pmean), and 5 ± 5 mmHg for the formula (SAP + ∆PPtoP predicted from Eq. (7)) (Table II; Fig. 4). The first formula (SAP + ∆Ppeak) overestimated peak LVSP in all situations, but the overestimation was much more important at low levels of C (Table II; Fig. 4, panel A). The second formula (SAP + ∆Pmean) overestimated peak LVSP in the presence of low C (Table II; Fig. 4, panel B). In contrast, the third formula (SAP + ∆PPtoP predicted from Eq. (7)) provided an accurate estimation of LVSP, regardless of the level of C (Table II; Fig. 4, panel C).

Table II: Mean absolute errors for different formulae used for non-invasive estimation of peak left ventricular systolic pressure (LVSP).

<table>
<thead>
<tr>
<th>Formula to estimate peak LVSP</th>
<th>Absolute error (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All data (n = 54)</td>
</tr>
<tr>
<td>SAP + ∆Ppeak</td>
<td>27 ± 16*</td>
</tr>
<tr>
<td>SAP + ∆Pmean</td>
<td>13 ± 9*</td>
</tr>
<tr>
<td>SAP + predicted ∆PPtoP</td>
<td>5 ± 5</td>
</tr>
</tbody>
</table>

*Significant difference versus peak LVSP measured by catheterization.

∆P: Transvalvular pressure gradient; ∆Pmean: Mean ∆P; ∆Ppeak: Peak ∆P; ∆PPtoP: Peak-to-peak ∆P;
C: Systemic arterial compliance; SAP: Systolic arterial pressure.
Discussion

In contrast to previous in-vivo studies, it was possible in the present study independently to control and modify the various parameters involved in valvular and vascular hemodynamics. The major findings of the study were that:

Changes in C and/or R had no direct effect on peak $\Delta P$, mean $\Delta P$, and valve EOA. Hence, these findings confirm that the significant reductions in peak and mean $\Delta P$s observed in AS patients with hypertension are likely due to a concomitant variation in flow rate and/or EOA.

A decrease in C is associated with a marked reduction in $\Delta P_{\text{to P}}$, which is independent of other hemodynamic parameters. Hence, $\Delta P_{\text{to P}}$ cannot be used to assess AS severity because it is also highly influenced by systemic arterial hemodynamics.

It is possible to estimate $\Delta P_{\text{to P}}$ and thereby peak LVSP by Doppler echocardiography and sphygmomanometry. The non-invasive estimation of LVSP may be useful to assess LV afterload in AS patients.

The major implication of the present study was that a reduction in C may indeed cause a marked decrease in $\Delta P_{\text{to P}}$ and thus lead to a major underestimation of stenosis severity. In this context, it should be emphasized that up to 40% of adult patients with AS have an abnormally low C (6), and that $\Delta P_{\text{to P}}$ is still often used to evaluate AS severity in adults (20-22) as well as in children (23, 24). Hence, $\Delta P_{\text{to P}}$ should not be used to evaluate AS severity and in particular, the recording of a low $\Delta P_{\text{to P}}$ should not be used to exclude the presence of a severe stenosis. To this effect, Christensen et al. (22) reported the clinical outcome in a subset of 12 patients in whom a conservative strategy was adopted on the basis of low invasive peak-to-peak pressure. All of these patients had a preserved LV function at baseline, and were not offered aortic valve replacement despite markedly reduced aortic valve EOA values (<1.0 cm$^2$ in 10 patients, and 1.0-1.3 cm$^2$ in two) and the presence of symptoms. At 2.5 years’ follow up, six patients had died, three had experienced further symptom progression and underwent aortic valve replacement, and three had a major worsening of their symptomatic status despite optimal medical treatment.

In patients with AS, LV afterload may be increased not only as result of the stenosis (i.e., valvular load) but also as a result of reduced C and/or increased R (i.e., vascular load). To this effect, it should be pointed out that the prevalence of hypertension has been reported as 30-40% in patients with AS (6, 25-27). When AS coexists with hypertension, the left ventricle faces a double pressure overload, and this may adversely affect LV function and patient outcome (6, 26). As emphasized in previous studies (4, 6, 26), the assessment of LV afterload should thus become an integral part of the routine Doppler echocardiographic examination of patients with AS, as it may have direct and important implications with regard to LV function and patient prognosis.

Nonetheless, the non-invasive measurement of indices of LV afterload requires the estimation of peak LVSP. In the Doppler echocardiography laboratory, this parameter is generally estimated by adding SAP and mean $\Delta P$ as a substitute for $\Delta P_{\text{to P}}$. However, as shown in Table II, this approach overestimates the peak LVSP and thus LV afterload in patients with reduced C. A more accurate estimation of LVSP can be obtained by adding SAP to $\Delta P_{\text{to P}}$ predicted by Eq. (7). Hence, by correcting mean $\Delta P$ for the influence of C, the new method proposed in the present study provides a more accurate assessment of LVSP. Interestingly, the new method is easily applicable to the clinical setting, as the three parameters included in Eq. (7) – that is, valve EOA, mean $\Delta P$ and C – are easily measurable in the context of routine Doppler echocardiography (6). The calculation of C simply requires the measurement of stroke volume and pulse pressure (Table I).

In a recent study (6), the proposal was made to esti-
mate global LV afterload in AS patients with the "valvulo-arterial impedance" calculated as the ratio of SAP + ΔPmean (as an estimate of peak LVSP) and stroke volume index. Valvulo-arterial impedance therefore represents the valvular and arterial factors that oppose ventricular ejection by absorbing the mechanical energy developed by the left ventricle. This new index was found to be the strongest independent predictor of LV diastolic and systolic dysfunction in this series of AS patients. The new method proposed in the present study (see Eq. (7) and Table I) for the estimation of peak LVSP could further improve the accuracy and predictive value of the valvulo-arterial impedance that may also be particularly useful when assessing LV afterload in the presence of AS, hypertension, or both. This method could also improve the clinical performance of other indices, such as peak LV systolic wall stress, that require peak LVSP for their calculation.

Study limitations
In the present study, the effect of systemic hypertension on the transvalvular pressure gradients was evaluated using theoretical and in-vitro approaches. As described above, in the in-vitro model the LV outflow tract and ascending aorta were both circular and rigid; hence, this model is characterized by infinite ventricular input impedance. In such circumstances, the LV flow waveform may contain some low-frequency oscillations (28) that are transmitted to the LV pressure and therefore to the instantaneous transvalvular pressure gradient. In the particular case of low transvalvular pressure gradients (TPGs), the peak-to-peak TPG may, therefore, vary as a consequence of LV pressure waveform oscillations, without any variation in the systemic arterial compliance. However, this is not the case in the present study as the TPGs were significantly high and the pump used for the in-vitro study was sufficiently powerful to generate a flow waveform without any low-frequency oscillations (12). Therefore, any variation in peak-to-peak TPG observed in vitro may be related only to a variation in the systemic arterial compliance.

In conclusion, ΔPpul should not be used to evaluate AS severity because, in contrast to peak ΔP and mean ΔP, it is heavily influenced by C. In particular, a low ΔPpul cannot be used to exclude the presence of a severe AS. The new non-invasive method proposed herein to estimate LVSP may be useful for obtaining a more accurate estimate of global LV afterload in patients with AS.

Acknowledgements
These studies were supported by an operating grant from the Canadian Institutes of Health Research (MOP-10929 and MOP-57745). Dr. Pibarot is the holder of the Canada Research Chair in Valvular Heart Disease, Canadian Institutes of Health Research, Ottawa, Canada. The authors thank Lynn Atton, Guy Noël, Justin Robillard, and Guy Rossignol for their technical assistance.

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Appendix I

The solution of Eq. (1) using the instantaneous flow Q(t) (Eq. (2)) can be written as:

\[
P(t) = \begin{cases} 
\frac{Q_0}{C \times w} \left( \frac{1}{1 + \frac{1}{\tau w}} \right) \sin(\omega t) - \frac{1}{w} \cos(\omega t) + \frac{P_0}{C \times w} \left( \frac{1}{1 + \left( \frac{1}{\tau w} \right)^2} \right) e^{-\frac{t}{\tau}} & 0 < t < T_s \\
\left( \frac{P_0}{C \times w} \left( \frac{1}{1 + \left( \frac{1}{\tau w} \right)^2} \right) \right) e^{-\frac{t}{\tau}} & T_s < t < T 
\end{cases}
\]

where

\[
P_0 = \frac{Q_0}{C \times w} \left( \frac{1}{1 + \left( \frac{1}{\tau w} \right)^2} \right) \left( \frac{\tau}{e^{\tau} + 1} \right) + \frac{h}{e^{\tau} - 1}
\]

\(\tau\) is a time constant, i.e., the product of R and C and \(\omega\) is the pulsation. This equation was used to compute the aortic pressure waveform.