Total arterial compliance estimated by stroke volume-to-aortic pulse pressure ratio in humans

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Chemla, Denis, Jean-Louis Hébert, Catherine Coirault, Karen Zamani, Isabelle Suard, Patrice Colin, and Yves Lecarpentier. Total arterial compliance estimated by stroke volume-to-aortic pulse pressure ratio in humans. Am. J. Physiol. 274 (Heart Circ. Physiol. 43): H500–H505, 1998.—On the basis of the windkessel model, the stroke volume-to-aortic pulse pressure ratio (SV/PP) has been proposed as an estimate of total arterial compliance, but recent studies have questioned this approximation. Aortic pressure was obtained at rest in 31 adults undergoing cardiac catheterization (47 ± 14 yr): controls (n = 7), patients with dilated cardiomyopathy (n = 10), and patients with other cardiovascular diseases (n = 14). We calculated PP, mean aortic pressure (MAoP), heart period (T), SV (thermodilution cardiac output/heart rate), total peripheral resistance (R), total arterial compliance estimated by area method (Carea), and the time constant of aortic pressure decay in diastole (RCarea). In the overall population (n = 31), there was no significant difference between SV/PP and Carea. SV/PP was linearly related to Carea (SV/PP = 0.99Carea + 0.05; r = 0.98; P < 0.001); the slope and intercept did not differ from unity and zero, respectively. Similar results were obtained in the three subgroups. These results imply that PP/MAoP and T/RCarea were proportionally related (TPCarea = 1.18PP/MAoP = 0.07; r = 0.96; P < 0.001). We conclude that for humans at rest 1) SV/PP gave an accurate estimate of Carea, and 2) T normalized by the time constant of aortic pressure decay in diastole was proportionally related to PP/MAoP. This last relationship could be considered an aspect of the coupling between the left ventricle and its load.

heart period; ventricular-arterial coupling; wave reflection

THE WAY IN WHICH THE HEART and its load are coupled contributes to efficient blood flow delivery to the tissues. A complete description of arterial load requires the evaluation of aortic input impedance spectra defined in the frequency domain (22, 25). In current clinical practice, this approach is complex, and thus time-domain evaluations of arterial load are most often used. Total peripheral resistance (R) reflects the steady component of arterial load, whereas the pulsatile component of arterial load is quantified by estimating total arterial compliance (13, 16, 36) and the indexes of wave propagation and reflection (12, 14, 24). Aging and essential hypertension are associated with lowered arterial compliance (17, 20, 26), thus contributing to increased pulsatile load, which may in turn adversely affect the myocardial supply-demand balance and ventricular-arterial coupling. Total arterial compliance is an important determinant of the load on the heart, and therefore its determination is of major interest for physiologists and clinicians.

Although R is commonly calculated from the ratio of mean aortic pressure to cardiac output, time-domain measurements of total arterial compliance are more difficult and are based on the windkessel model of systemic circulation (10). Although the limitations of this model have been discussed (22, 25), its applicability has been widely demonstrated in humans (2, 13, 16). Recently, Liu et al. (16) have evaluated a method estimating total arterial compliance from systolic and diastolic areas under the aortic pressure wave. The so-called area method is now considered as the reference for time-domain estimation of total arterial compliance in humans (3, 19, 36). This method requires continuous pressure data recordings throughout the cardiac cycle, and this limits its clinical use and its diagnostic and therapeutic benefits (19).

The ratio of stroke volume to aortic pulse pressure (SV/PP) was initially proposed as an estimate of arterial compliance (32). Recent studies have questioned the accuracy of this approximation (2, 16). It has been stated that estimating total arterial compliance by SV/PP violates the fundamental concept of the windkessel model (2); others have predicted that SV/PP would be markedly larger than total arterial compliance (16). This contrasts with the results showing that SV/PP appears to be a relatively good estimate of total arterial compliance (calculated by using a monoexponential fit of aortic pressure decay) (9).

The aim of our study was to assess SV/PP as an estimate of total arterial compliance (area method; Carea) in humans at rest. If SV/PP is indeed an accurate estimate of Carea, one important implication is that the heart and its load could be coupled in such a way that the ratio of pulse pressure to mean aortic pressure equals the ratio of heart period to arterial decay time. We therefore investigated these ratios and their proportionality.

METHODS

Patients. Thirty-one patients (27 men and 4 women) were enrolled in our prospective study after informed consent was obtained. The investigation was approved by our institution. Patients were referred to our laboratory for diagnostic right and left heart catheterization for symptoms of chest pain, heart failure, or other cardiovascular symptoms. Patients with end-stage heart failure, rhythm disturbances, and aortic and mitral valve insufficiency were excluded from the study. Three groups were defined as follows: normal subjects (n = 7),
idiopathic dilated cardiomyopathy (n = 10), and miscellaneous cardiac diseases (mainly coronary artery disease, hypertrophic cardiomyopathy, and right ventricular disease) (n = 14). Preliminary results have been published elsewhere (5).

Catheterization technique and protocol. Patients were studied in the early morning in a basal state. They were unseated and investigated ≥12 h after the last intake of their usual treatment. Routine right heart catheterization was performed using the Seldinger technique with an 8-Fr sheath from the femoral vein. The right heart catheter was a 7.5-Fr five-lumen thermodilution pressure-measuring tipped catheter with a high-fidelity transducer (Cordis/Sentron, Roden, The Netherlands) (4). The catheter was advanced into either the right or the left pulmonary artery to measure cardiac output. The left heart catheter was either an 8-Fr single-lumen catheter with a lateral high-fidelity transducer and a hole at the distal end or a closed 5-Fr catheter tipped with a high-fidelity transducer (Cordis/Sentron). The left heart catheter was advanced from the femoral artery to the aortic root in 28 of 31 patients. In three patients with peripheral arterial disease of the lower limbs, we used the percutaneous brachial artery approach (11). Pressure data were obtained at baseline after a 10-min equilibrium period. The data were computed on a Toshiba 3200SX with homemade software (sampling rate 500 Hz).

High-fidelity recordings at the aortic root level and cardiac output. We measured systolic (SAoP), diastolic (DAoP), pulse (PP = SAoP – DAoP), and end-systolic aortic pressures (ESAoP). ESAoP was defined as the nadir of the incisura (dicrotic notch). We computed systolic (A_s) and diastolic (A_d) areas under the pressure curve. We measured heart period (T) as the time between two consecutive aortic pressure upstrokes. The time to SAoP was measured from the foot of the pressure upstroke to SAoP. Mean aortic pressure (MAoP) was calculated as the total area under the pressure curve (i.e., \( A_s + A_d \)) divided by T. We calculated the ratio PP/MAoP. Cardiac output was measured in triplicate using the thermodilution technique in all patients. SV was calculated by dividing cardiac output by heart rate.

Wave reflection and augmentation index. The human aortic pressure waveform exhibits an inflection point (P_i) indicating the end of the forward (or incident) wave and resulting from peak flow input into the vasculature previous to the effects of wave reflection. The relative increase in pressure amplitude above the inflection point (∆P = SAoP – P_i) is an estimate of the magnitude of the reflected pressure wave. The ratio of ∆P to aortic pulse pressure defines a so-called augmentation index (∆P/PP), thus allowing quantification of the extent of wave reflection in central arteries (24). The systolic inflection point was clearly defined in 25 of 31 subjects (81%). They were divided into three groups according to the classification previously proposed by Murog et al. (24): type A (n = 21), ∆P/PP > 0.12; type B (n = 4), 0 < ∆P/PP < 0.12; type C (n = 0), ∆P/PP < 0. Thus, according to this classification, all our subjects were type A or type B. Given that all but three patients were older than 30 yr of age, this finding is consistent with earlier works (24, 26). In these 25 subjects, the time to SAoP (220 ± 39 ms) occurred during the second half of the systolic period and encompassed 75 ± 8% of left ventricular ejection time (LVET) (59–97%). In six subjects the inflection point could not be discerned; in these subjects, the time to SAoP (216 ± 58 ms) occurred during the second half of the systolic period and encompassed 74 ± 9% of LVET (58–83%).

Total arterial compliance estimated by the area method. We assumed the windkessel model of systemic circulation. To ensure zero flow in diastole, we obtained pressure data at the aortic root level, and the patients with aortic insufficiency were excluded from the study. According to the area method (16) it can be derived that total arterial compliance is

\[
\begin{align*}
C_{area} & = \frac{SV}{K (ESAoP – DAoP)} \\
& \text{with} \\
K & = \frac{(A_s + A_d)}{A_d}
\end{align*}
\]

where K is the area coefficient. Theoretical considerations and hypotheses tested. The first hypothesis tested was the equality of \( C_{area} \) and SV/PP, i.e.

\[
C_{area} = SV/PP
\]

This equality, in conjunction with Eq. 1, implies that

\[
PP = K (ESAoP – DAoP)
\]

R is MAoP divided by mean flow, or MAoP divided by SV/T, i.e.

\[
R = MAoP/(SV/T)
\]

The time constant of aortic pressure decay in diastole (T_c) equals the product of R and total arterial compliance. Using \( C_{area} \) as a good approximation of total arterial compliance, and inserting Eqs. 2 and 4 in \( T_c \), we obtain

\[
T_c = (MAoP/SAoP)(SV/PP)T = T \times MAoP/PP
\]

or

\[
T/T_c = PP/MAoP = T/RC_{area}
\]

Thus, if SV/PP is indeed an accurate estimate of \( C_{area} \), one potentially important implication is the equality between two dimensionless ratios: the ratio of two pressures (PP/MAoP) and the ratio of two times (T/RC_{area}). Furthermore, Eq. 6 may be considered as an aspect of the coupling between the heart and its load. In the present study, we critically evaluated the accuracy of Eqs. 2, 3, and 6.

Data analysis and statistics. Results are expressed as means ± SD. Pressures, pressure areas, and time parameters were averaged over 10 consecutive cardiac cycles. Comparisons were performed using Student’s t-test. Linear regressions were performed using the least-squares method. A P value < 0.05 was considered statistically significant.

RESULTS

Characteristics of the study population are listed in Table 1. Compliance estimates. In the overall population, SV/PP ranged from 0.34 to 2.80 ml/mmHg (mean ± SD: 1.46 ± 0.69 ml/mmHg) and \( C_{area} \) ranged from 0.31 to 2.74 ml/mmHg (mean ± SD: 1.43 ± 0.68 ml/mmHg) (Table 2). \( C_{area} \) was negatively related to age, MAoP, PP, and ∆P/PP and positively related to SV and body length (Table 3). There was a positive linear relationship between PP and MAoP (r = 0.74, P < 0.001) and between PP and ∆P/PP (r = 0.67, P < 0.001).

In the overall population as well as in the three study groups, there was no significant difference between \( C_{area} \) and SV/PP (Table 2). There was a strong linear relationship between \( C_{area} \) and SV/PP (r = 0.99; P < 0.001; n = 31), and the slope and intercept of the regression lines did not differ from unity and zero, respectively (Fig 1 and Table 2).
The equality between $C_{\text{area}}$ and SV/PP (see Fig. 1 and Table 2) was partly explained by the close relationship between PP and the product of $K$ times the difference between ESAoP and DAoP (see Eq. 3) [$PP = 0.82[\{\text{ESAoP} - \text{DAoP}\} + 7.6 \text{ mmHg}; n = 31; r = 0.97; P < 0.001]$; the slope was different from unity ($P < 0.05$), whereas the intercept was not different from zero. There was also a close linear relationship between PP and (ESAoP − DAoP) ($r = 0.91, P < 0.001$). The equality between the two compliance estimates was independent of the pressure wave shape, given that 1) no relationship was found between PP and $K$ ($r = 0.30$), and 2) the difference between SV/PP and $C_{\text{area}}$ was not related to $\Delta P/PP$ ($r = 0.10$) (Fig. 2).

Relationship between PP/MAoP and $T/RC_{\text{area}}$. In the study population ($n = 31$), PP/MAoP ranged from 0.33 to 0.78. $T/RC_{\text{area}}$ ranged from 0.29 to 0.82. $T/RC_{\text{area}}$ was not significantly different from PP/MAoP (Table 4).

### Table 1. Characteristics of the study population

<table>
<thead>
<tr>
<th>Attribute</th>
<th>Values</th>
<th>Range</th>
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<tbody>
<tr>
<td>Age, yr</td>
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<tr>
<td>Weight, kg</td>
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<td>Body length, cm</td>
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<td>T, ms</td>
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<td>SV, ml</td>
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<td>Ejection fraction, %</td>
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<td>SAoP, mmHg</td>
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<td>DAoP, mmHg</td>
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<tr>
<td>PP, mmHg</td>
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</tr>
<tr>
<td>(ESAoP − DAoP), mmHg</td>
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<td></td>
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<tr>
<td>$K$</td>
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<tr>
<td>$\Delta P/PP$, %</td>
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</table>

Values are means ± SD; $n = 31$ subjects. Pressure data and heart period (T) were averaged over 10 consecutive cardiac cycles. SV, stroke volume; SAoP, systolic aortic pressure; DAoP, diastolic aortic pressure; PP, aortic pulse pressure; ESAoP, end-systolic aortic pressure; $K$, area coefficient; $\Delta P$, late systolic pressure augmentation; $\Delta P/PP$, augmentation index.

### Table 2. Compliance estimates

<table>
<thead>
<tr>
<th></th>
<th>Overall Population</th>
<th>Controls</th>
<th>IDCM</th>
<th>Miscellaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>($n = 31$)</td>
<td>($n = 7$)</td>
<td>($n = 10$)</td>
<td>($n = 14$)</td>
<td></td>
</tr>
<tr>
<td>$C_{\text{area}}$, ml/mmHg</td>
<td>1.43 ± 0.68</td>
<td>1.84 ± 0.76</td>
<td>1.48 ± 0.75</td>
<td>1.21 ± 0.53</td>
</tr>
<tr>
<td>$SV/PP$, ml/mmHg</td>
<td>1.46 ± 0.69</td>
<td>1.91 ± 0.76</td>
<td>1.48 ± 0.74</td>
<td>1.23 ± 0.53</td>
</tr>
<tr>
<td>($SV/PP − C_{\text{area}}$) ml/mmHg</td>
<td>0.03 ± 0.15</td>
<td>0.02 ± 0.10</td>
<td>0.02 ± 0.19</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SD; $n =$ no. of patients. Controls, patients with normal left ventricle (LV) and normal coronary arteries; IDCM, patients with idiopathic dilated cardiomyopathy; miscellaneous, patients with various forms of cardiac disease (see METHODS). $C_{\text{area}}$, total estimated arterial compliance (by area method). Characteristics of linear relationship between SV/PP and $C_{\text{area}}$ are given as $SV/PP = aC_{\text{area}} + b$, where $a$ is slope and $b$ is intercept. For overall population and for each patient group, SV/PP value was not significantly different from corresponding $C_{\text{area}}$ value ($P > 0.05$).

There was a strong linear relationship between the two ratios ($r = 0.96, P < 0.001$; $n = 31$) (Fig. 3). The intercept was not different from zero. The slope was different from unity ($P < 0.05$) such that the regression line progressively diverged from the identity line, especially for high PP/MAoP values (Fig. 3). As a result, $T/RC_{\text{area}}$ was equal to PP/MAoP in subjects with PP/MAoP < 0.50 ($n = 17$; mean difference ± SD = 0 ± 0.03), whereas $T/RC_{\text{area}}$ slightly overestimated PP/MAoP in subjects with PP/MAoP > 0.50 ($n = 14$; mean difference ± SD = 0.05 ± 0.06) (Fig. 3).

### DISCUSSION

The main results of our study were as follows. 1) On the basis of the windkessel model of systemic circulation, SV/PP was equal to $C_{\text{area}}$ in humans at rest. 2) Our results may be explained by the fact that heart period normalized by the time constant of aortic pressure fall in diastole is proportionally related to PP/MAoP in humans at rest, a finding consistent with recent results in comparative physiology (39).

Relationship between SV/PP and total arterial compliance comparison with previous results. Since its validation by Remington et al. (32), SV/PP has been used to estimate arterial compliance (9, 28, 29, 34). Others have estimated arterial stiffness (or rigidity) by...
using PP/SV (1, 7, 21, 38). On the basis of theoretical and experimental arguments, recent studies have advised against the use of SV/PP as an estimate of total arterial compliance, such that alternative methods must be used (2, 13, 16, 36). Conversely, a previous study has shown that SV/PP is linearly related (r = 0.80) to total arterial compliance estimated by exponential fitting of diastolic pressure decay (9). Our study indicates that SV/PP was a simple, accurate estimate of $C_{\text{area}}$ in humans at rest. The results were obtained despite marked differences in cardiac status and over a wide range of aortic pressures, heart rates, $C_{\text{area}}$ values, and extents of wave reflection.

Differences between our conclusions and others may be explained by the greater accuracy of the area method. This method avoids potential artifacts stemming from the choice of cutoff values for the onset and end of monoexponential analysis (16, 36). As this method does not depend on the exact form of the pressure wave, it is not influenced by deviations from a true exponential function (16). The mean value of the area coefficient K we reported (1.72; $n = 31$) is consistent with that of Liu et al. (1.68; $n = 17$) (16). Liu et al. (16) have predicted that SV/PP should be markedly larger than $C_{\text{area}}$. However, the mean difference between SV/PP (calculated from Table 1 in Ref. 16) and total arterial compliance ($C_1$ in Table 2 of Ref. 16) is $-0.07 \text{ ml/mmHg}$, which strengthens our findings. Importantly, SV/PP and $C_{\text{area}}$ cannot be considered interchangeable estimates of "real" total arterial compliance. SV/PP determines compliance at MAoP, whereas $C_{\text{area}}$ determines compliance at average diastolic pressure (which is known to be lower than MAoP). Because compliance normally decreases when arterial pressure increases (13, 16, 36), one can expect that $C_{\text{area}}$ was in fact lower than SV/PP in our patients.

PP, arterial compliance, and wave reflection. Aortic PP is determined by the patterns of left ventricular ejection, aortic stiffness, and wave reflections (22, 27, 33). Cardiac ejection into a low-compliance system generates a wider PP than in a normally compliant system (30, 31, 37). Furthermore, reduced arterial compliance is associated with increased pulse wave velocity and wave reflection, and this also contributes to increased PP (12, 18, 24, 33). These mechanisms could account, at least in part, for the linear relationship observed in our study between $C_{\text{area}}$ and SV/PP. Furthermore, in aged and hypertensive subjects, it is well documented that increased PP is associated with a shortening of the $R \times C$ product (1, 9, 35). In these patients, both higher ESAoP and less compliant arterial vasculature contribute to this close link (17, 20, 26, 27).

The reflection of pressure waves leading to inflection point and the augmentation index result from wave transmission characteristics that are not contained in the windkessel models (24). Yet a good correlation between $C_{\text{area}}$ and SV/PP was found. We feel it unlikely that the observed equality between SV/PP and $C_{\text{area}}$ was casual or related to the mutual canceling of the many assumptions and approximations on which the two compliance estimates were based. We suggest that this equality may furnish a basis for recent results in comparative physiology (39).

**Table 4. Aortic pulse pressure-to-mean aortic pressure ratio and time parameter ratio**

<table>
<thead>
<tr>
<th>Overall Population (n = 31)</th>
<th>Controls (n = 7)</th>
<th>IDCM (n = 10)</th>
<th>Miscellaneous (n = 14)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PP/MAoP</td>
<td>0.51 ± 0.012</td>
<td>0.46 ± 0.10</td>
<td>0.49 ± 0.12</td>
</tr>
<tr>
<td>T/RC_{area}</td>
<td>0.53 ± 0.15</td>
<td>0.48 ± 0.14</td>
<td>0.50 ± 0.14</td>
</tr>
<tr>
<td>T/RC_{area}−PP/MAoP</td>
<td>0.02 ± 0.05</td>
<td>0.02 ± 0.04</td>
<td>0.01 ± 0.03</td>
</tr>
</tbody>
</table>

Values are means ± SD. R, total peripheral resistance. For overall population and for each patient group, T/RC_{area} value was not significantly different from corresponding PP/MAoP value ($P > 0.05$).
ratio \((T/T_c)\) and \(T/T_c\) were independent of body mass in all mammalian species. These authors have hypothesized that the independence of \(T/T_c\) and \(T/T_c\) relative to body mass suggests that heart rate is compelled by the arterial tree to maintain similar diastolic and/or pulse pressure in all mammalian species, thus warranting coronary perfusion \((8, 39)\). In our study, we have taken advantage of some redundancies in hemodynamic formulas to predict that the equality between \(SV/PP\) and \(PP/MAoP\) could be equal to that of two pressures \((PP/MAoP)\) could be viewed as a contributory factor in ventricular-arterial coupling. Viewed as a contributory factor in ventricular-arterial coupling, \(PP/MAoP\) \((T/T_c)\) equals \(PP/MAoP\) in numerous subjects, especially those with \(PP/MAoP < 0.50\) (see Fig. 3). Given that \(T\) determines the frequency of blood spurts from the ventricle into the aorta and that both the resistive and viscoelastic properties of the arterial tree determine the value of \(RC_{a_\text{area}}\), the fact that the dimensionless ratio of two times \((T/RC_{a_\text{area}})\) was equal to that of two pressures \((PP/MAoP)\) could be viewed as a contributory factor in ventricular-arterial coupling.

Importantly, however, our patients exhibited a wide range of \(T/RC_{a_\text{area}}\) values, and this indicated that \(T/T_c\) values could not be considered constants in humans, contrary to what has been hypothesized in comparative physiology \((39)\). Furthermore, the \(T/RC_{a_\text{area}}\) vs. \(PP/MAoP\) regression line diverged from identity at high \(PP/MAoP\) values (Fig. 3), and this may testify to an uncoupling between the left ventricle and its load, a point that deserves further study. Finally, our study also strengthens the physiological relevance of \(PP/MAoP\). Several studies have stressed the fact that \(PP\) depends on mean pressure: the higher the mean pressure, the higher the fluctuations around the mean \((6, 18, 33)\). It has also been shown that \(PP/MAoP\) is linearly related to the characteristic impedance-to-R ratio in dogs with ascending aorta-abdominal aorta bypass \((23)\).

Study limitations. The windkessel model implies infinitely high wave speed in diastole and an absence of wave reflection \((2, 22, 25)\), whereas wave reflections are known to occur in both health and disease \((14, 15, 24)\). However, this model has been assumed to be applicable to humans, especially at low frequencies corresponding to normal ranges of heart rate \((2, 13, 16, 36)\). Other shortcomings of the area method need to be pointed out.

1) No attempt was made to evaluate the runoff of blood forwarded into the peripheral circulation during systole.
2) It was assumed that the pressure asymptote is so small as to be negligible. The pressure dependence of compliance estimates \((16, 36)\) was not tested in our study. The results pertain strictly to the study population, of which we had excluded patients with aortic and mitral valve insufficiency.
3) We cannot exclude the possibility that our findings do not apply to subjects with negligible wave reflections (type C subjects) \((24)\), and this deserves further studies focused on younger populations.

Implications. From a practical point of view, two implications must be discussed. First, it is suggested that \(SV/PP\) furnishes a rapid, valuable estimate of \(C_{a_\text{area}}\). We wish to emphasize that the purpose of our study was not to recommend that \(SV/PP\) replace \(C_{a_\text{area}}\), which remains the reference estimate of total arterial compliance in the time domain. However, the area method requires continuous pressure data recordings throughout the cardiac cycle, and this limits its clinical applications. Second, the SV-to-brachial \(PP\) ratio \((SV/PP_b)\) has been previously used to estimate total arterial compliance. This approximation is likely to be accurate only in patients with no or minor amplification of pulse pressure from aorta to periphery; conversely, it must be used cautiously with subjects exhibiting physiological pulse wave amplification, given that \(SV/PP_b\) is likely to underestimate total arterial compliance in these subjects. From a physiological point of view, and to the best of our knowledge, this is the first study to have proposed a hemodynamic formula relating pulse pressure to the heart period of the corresponding beat \((i.e., PP/MAoP = T/RC_{a_\text{area}}\) (see Eq. 6). Although pertaining strictly to the windkessel model, this new relationship could reasonably describe one aspect of the coupling between the left ventricle and its load. Further studies are needed to assess the relevance of this relationship in various populations and under dynamic conditions. Finally, from an epidemiological point of view, increased blood pressure and heart rate are considered major cardiovascular risk factors. Our results suggest that the changes in blood pressure (mean, pulse), heart period, and arterial time constant may well be coordinated (e.g., during aging or hypertension), and this point deserves further study.

Conclusion. On the basis of the windkessel model of systemic circulation, \(SV/PP\) was equal to \(C_{a_\text{area}}\) in humans at rest. This implied that heart period normalized by the time constant of aortic pressure fall in diastole is proportionally related to \(PP/MAoP\), a finding in keeping with recent results in comparative physiology.

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