Peripheral “Oscillatory” Compliance Is Associated With Aortic Augmentation Index

Patrick Segers, Ahmad Qasem, Tine De Backer, Stephane Carlier, Pascal Verdonck, Albert Avolio

Abstract—The augmentation index (AIx) and “oscillatory” compliance (C2) are wave contour analysis parameters for the central aorta (Pao) and radial artery pressure wave (Prad), respectively. Both are sensitive to cardiovascular risk factors such as aging, hypertension, and diabetes and have been proposed as prognostic markers for cardiovascular disease. In this work, we studied the relation between both. We first calculated Prad corresponding to a typical aortic A-type (AIx >0.15) and C-type wave (AIx <0), taken from the literature, by using a generalized aorta-radial pressure transfer function. Prad corresponding to C-type waves yielded the highest C2 value. We further used simultaneously measured aortic and radial artery pressure in 45 human subjects age 34 to 84 years (63±12 [SD]) at baseline and after administration of nitroglycerin to calculate AIx meas and C2, respectively. Transfer function was used to calculate reconstructed aortic pressure and AIx rec. AIx rec underestimates AIx meas by 0.03±0.16, but both values correlate well (r=0.64; P<0.001). C2 and AIx were inversely correlated (r=−0.36; P<0.001 for AIx meas, r=−0.30; P<0.01 for AIx rec). Both AIx meas (0.06±0.17 versus 0.20±0.21; P<0.01) and AIx rec (0.04±0.12 versus 0.16±0.16; P<0.001) were lower after nitroglycerin, whereas C2 increased only nonsignificantly (0.080±0.036 versus 0.071±0.042). C2 is related to AIx and reflects, at least in part, hemodynamic changes affecting central aortic pressure. Nevertheless, given the model assumptions and computational steps associated with calculating C2, AIx could be a more appropriate parameter to use in the clinical setting because it is determined directly from the pressure wave contour. (Hypertension. 2001;37: 1434-1439.)

Key Words: compliance ■ arterial pressure ■ hypertension, arterial ■ blood pressure ■ aorta

The aortic pressure wave results from the interaction of the ejecting left ventricle and the systemic arterial system. On ejection, an incident, forward wave propagates from the heart toward the periphery, where it is reflected at bifurcations and arteriolar beds, giving rise to reflected, backward waves propagating toward the heart. The measured aortic pressure thus consists of incident and reflected waves, and their magnitude and timing determines the aortic wave contour.1 In the young, the reflected waves arrive in the ascending aorta in late systole and generate the typical C-type pressure wave. With aging and in hypertension, the magnitude and speed of the reflected waves increase, and they add to the forward wave in early systole, boosting systolic pressure and generating the typical A- and B-type aortic pressure wave profile. The relative contribution of the reflected waves to blood pressure is quantified by the augmentation index, which is related to the overall mechanical properties of the vasculature.2 Though equally present, the effect of wave reflection on the flow wave has been less studied. The reflected pressure and flow wave are similar in form but are the inverse of each other: The reflected pressure wave increases pressure, whereas the reflected flow decreases flow.3

An alternative pressure wave contour analysis method is routinely used by Cohn,4 with the use of the parameters of a 4-element Windkessel model, originally introduced by Goldwyn and Watt5 in 1967. By fitting the model parameters to the diastolic portion of the brachial or radial artery pressure wave, they obtain parameters that quantify the morphology of the radial artery pressure wave. It has been shown that one of the model parameters (C2) characterizes the oscillatory nature of the pressure wave and is generally related to peripheral effects of wave reflection. C2 is reduced with age,6 in hypertension,7 and in diabetes8 and has been proposed as an early and sensitive marker of cardiovascular disease, with diagnostic information. On the other hand, the method is somewhat controversial, probably because of the unclear physical meaning of the model parameters, and has been questioned on theoretical grounds.9 In this study, we ran a parameter study to assess how changes in the 4 parameters of the Goldwyn-Watt model affect the morphology of the diastolic portion of the radial artery pressure wave.

It is known that aortic and radial artery pressures are related through the radial-aorta pressure transfer function. Generalized, average forms of this transfer function have
Aortic Pressure Wave Analysis: Augmentation Index

The augmentation index (Alx) is given by

$$\text{Alx} = \frac{P_P - P_i}{P_P - P_a}$$

with $P_a$, $P_i$, and $P_P$ indicating systolic, diastolic, and inflection pressure, respectively (Figure 1). Alx is positive when $P_P$ precedes $P_i$ (A- or B-type wave) and negative when $P_i$ precedes $P_P$ (C-type wave).

Radial Artery Pressure Wave Analysis

Assessing 4 Parameters of Goldwyn-Watt Model

The 4-element Windkessel model, introduced in 1967 by Goldwyn and Watt, consists of 4 elements: total peripheral resistance (R) and proximal and distal elastic chambers ($C_1$ and $C_2$, respectively) separated by an inertial element L. It has been shown that during the augmentation index and $C_2$ describing aortic and radial artery pressure waves that were reconstructed from the radial pressure curve by using the generalized transfer function.

Methods

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Figure 1. Left, Central aortic pressure with definition of augmentation index. Right, Solid line is measured radial artery pressure. Symbols show result of fitting Equation 2 to diastolic part of curve. Upper right corner shows 4-element lumped parameter model as originally proposed by Goldwyn and Watt.

R is calculated as the ratio of mean pressure and mean flow. In analogy with previous studies, we estimated cardiac output (CO) from patient data and the radial artery pressure wave by using the following empirical expression:

$$\text{CO} = \frac{HR}{1000} \left( -0.6 + 0.25(T_{SN} - 35) - 0.02 \cdot HR + 0.4 \cdot \text{BSA} - 0.51 \cdot \text{age} \right)$$

with $HR$, $T_{SN}$, and body surface area (BSA) the subject's heart rate (beats/min), systolic ejection time (ms), and body surface area (m²), respectively. $T_{SN}$ is estimated from the radial artery pressure wave as the time interval between the onset of the systolic rise and the dicrotic notch. Age is in years.

Effect of Parameter Changes on Radial Artery Wave Contour

Applying the Goldwyn-Watt model to the radial artery pressures measured in 45 human subjects at baseline and after administration of NTG (see below), we found average and upper and lower limit values for $R$ (0.95 [0.5 to 2.5] mm Hg/mL/s), $C_1$ (2.27 [0.4 to 6.5] mL/mm Hg), $C_2$ (0.075 [0.005 to 0.2] mL/mm Hg), and $L$ (0.066 [0.01 to 0.25] mm Hg/mL/s²). We studied the effect of individual changes in each of these parameters (giving the other parameters the average value) on the contours of the diastolic portion of the radial artery pressure wave. In normal, routine application of the Goldwyn-Watt model, parameters $A_1$ to $A_6$ are determined, and the 4 model parameters follow directly from Equations 3 through 5. In this application, values for the 4 model parameters were prescribed, whereas $A_1$, $A_5$, and $A_6$ were derived from Equations 3 through 5 in an implicit way (Matlab, Mathworks Inc). $A_1$, $A_5$, and $A_6$ depending only on initial conditions and measuring location, were given fixed values of 100, −10, and 0.5, respectively. The radial artery wave contours were then calculated by Equation 2.

Augmentation Index Versus $C_2$

Calculated Radial Pressure Corresponding to C- and A-Type Aorta Pressure

Typical type-A and type-C aortic pressure wave forms, given by Murgo et al., were digitized. A generalized time-domain formulation in the form of an autoregressive model of an aortic-radial pressure transfer function was applied on these curves, yielding the corresponding reconstructed radial artery pressure waves. The augmentation index and the 4 Goldwyn-Watt model parameters, with aorta and radial artery pressures, respectively, were determined as described above, except for the value of peripheral resistance, which was calculated from mean aorta pressure and cardiac output, estimated by Equation 6.

In Vivo Study

Forty-five sets of data, consisting of simultaneously recorded invasive aortic and radial artery pressure waves with identical fluid-filled catheters (frequency response >15 Hz), were included in this study. The study protocol was approved by the Institutional Ethics Committee (A. Pauca, Wake Forest University, School of Medicine; personal communication). Data were recorded in anesthetized patients before cardiac procedures. The population (35 male, 10 female subjects) consisted of patients with coronary artery disease (n=41) and/or patients treated for hypertension (n=43). Age, weight, height, and BSA were 63±12 (34 to 84) years, 84±16 (54 to 112) kg, 172±8 (155 to 189) cm, and 1.97±0.20 (1.53 to 2.39) m², respectively. Baseline data were recorded during steady-state conditions for at least 15 seconds. Measurements were repeated after administration of NTG in 40 subjects (6 to 16 μg·kg⁻¹·min⁻¹ IV for a range of
infusion times of 5 to 20 seconds). The measured sequence of 10 to 20 cardiac beats was averaged and yielded one representative aortic (Pao, meas) and radial artery pressure wave. The generalized time-domain aortic-radial pressure transfer function was further applied on radial artery pressure to yield a reconstructed aortic pressure wave (Pao, rec). AIx was derived from measured (AIx meas) and reconstructed (AIx rec) aortic pressure wave, and 4 Goldwyn-Watt model parameters were determined on the radial artery pressure wave.

Data Analysis
Data are presented as mean±SD. Linear regression analysis (SigmaStat; Jandel Corp) was applied to assess the correlation between AIx meas and AIx rec and C2. We further studied baseline versus NTG data by using paired t-tests.

Results
Radial Artery Pressure Wave Analysis: Parameter Study
The results of the parameter study are shown in Figure 2. C1 and R mainly affect the time constant of the exponential decaying pressure, without much effect on the oscillatory character of the pressure wave. For the same end-systolic pressure, a higher C1 and R give higher end-diastolic pressures. The oscillatory nature of the diastolic pressure wave is affected by C2 and L. The peak of the diastolic pressure wave is shifted to the right for higher values of C2, but its maximum value is hardly changed. The inertial term has most effect on diastolic pressure wave morphology. The higher L, the more the diastolic wave is damped. For low L (<0.05 mm Hg/ml/s2), multiple oscillations are observed during diastole. Overall, the oscillatory character increases with higher C2 and lower values of L.

Augmentation Index Versus C2
Calculated Radial Pressure Corresponding to C- and A-Type Aortic Pressure
The digitized aortic and corresponding reconstructed radial artery pressure waves are given in Figure 3. The radial artery pressure curve corresponding to the C-type pressure has a somewhat higher oscillatory character. For both curves, expression of Equation 2 yielded a close fit to the diastolic portion of the radial artery pressure wave (r2>0.99). The resulting parameter values are given in Table 1. R and L are lower for the C-type subject, whereas C1 and C2 are markedly higher.

In Vivo Study
Hemodynamic data, AIx, and the 4-element Windkessel parameters are given in Table 2. NTG lowers blood pressure and slightly increases heart rate. NTG lowers R and increases C1, whereas there was no effect on C2 or L. NTG reduces AIx meas as well as AIx rec. At baseline, both AIx meas and AIx rec are correlated with age (r=0.40; P<0.01 and r=0.43; P<0.01), height (r=−0.38; P<0.05 and r=−0.49; P<0.001), and C2 (r=−0.34; P<0.05 and r=−0.37; P<0.05). C2 correlates with BSA (r=0.39; P<0.01) and cardiac output (r=0.46; P<0.01), as expected, but also with heart rate (r=−0.33; P<0.05). After NTG, only AIx meas correlates with C2 (r=−0.32; P<0.05). AIx rec correlates with age (r=0.46; P<0.05) and heart rate (r=−0.36; P<0.05). C2 correlates only with heart rate (r=−0.39; P<0.05). Figure 4 shows the correlation between both AIx meas and AIx rec and C2 for baseline and NTG. Pooling baseline and NTG, the...
correlation coefficients between $C_2$ and AIx$_\text{meas}$ and AIx$_\text{rec}$ become $-0.36$ ($P<0.001$) and $-0.30$ ($P=0.005$), respectively.

AIx$_\text{rec}$ underestimates AIx$_\text{meas}$ by $0.03\pm0.16$ (0.04±0.16 at baseline and 0.02±0.15 after NTG). The correlation between both is $0.66$ ($P<0.001$) at baseline, $0.46$ ($P<0.01$) after NTG, and $0.64$ ($P<0.001$) for all data (Figure 4).

**Discussion**

The radial artery pressure wave contour analysis method, as proposed by Cohn and coworkers, offers some interesting and potentially useful features. The method can be applied to the radial artery pressure, which is easily measured noninvasively by tonometry. The theory, as originally proposed by Goldwyn and Watt, is based on a relatively simple modified Windkessel model, consisting of 4 elements with a clear physiological meaning: total vascular resistance (R), a large proximal compliant chamber representing proximal large arteries ($C_1$), a small distal compliant chamber, representing elastic properties of the smaller arteries ($C_2$), and an inertial element in between, representing the inertia of the blood in the vessels (L). Moreover, in general, expression of Equation 2 yields excellent fittings of the diastolic portion of the radial artery pressure wave. Clinical studies have further shown that $C_2$ is reduced in aging, hypertension, or diabetes. $C_2$ is therefore expected that L varies inversely with body mass and BSA. Indeed, the analysis did uncover a negative though nonsignificant correlation ($r=-0.23, P=0.13$ and $r=-0.19, P=0.22$ for body mass and BSA, respectively). L also varied over a relatively wide range between 0.016 and 0.22 mm Hg/(mL/s$^2$). This would imply that within this population, cross-sectional area and vessel diameter in individuals may differ by a factor 13.8 in area and 3.7 in diameter, respectively. This variation is rather high to represent true, physical arterial differences.

It is also important to realize that the 4-element model parameters directly depend on the value of cardiac output that is used for the calculation of total peripheral resistance. The model is generally applied by Equation 6, using an empirical relation for cardiac output (expression of Equation 6), as described in the HDI/Pulsewave software (operating manual C-VPI Model CR-2000, Rev. November 18, 1998; page 1 to 7). In this study, cardiac output was measured with thermoligation at baseline in 24 patients. The correlation between measured and calculated cardiac output was rather low but significant ($r=0.45$, $P<0.05$), with the empirical relation overestimating measured cardiac output (5.56±0.86 versus 5.02±1.33 L/min; $P<0.05$). Bland-Altman analysis showed that the mean difference between the two values was 0.54±1.21 L/min. Taking the 95% confidence intervals, the individual difference between actual and estimated cardiac output may vary from $-1.9$ L/min to $3.0$ L/min, for an average value of $\approx 5$ L/min. The empiric relation may thus theoretically, yield model parameters that are independent of the measuring location. It has been shown in dogs, with invasive measurements, that this is not always the case. $C_2$ has been called “distal,” “oscillatory,” or “reflective” compliance, which illustrates that there is no straightforward physical interpretation of some of the model elements. It is impossible to attribute $C_2$ to a well-defined arterial territory. It is also somewhat confusing to use the terminology “reflective” compliance for an index that is based on the diastolic portion of the pressure wave (thus ignoring systole), knowing that the effect of wave reflection on the pressure wave contour is most present during systole. Another concern is the inertial element in the model. It is clear from the parameter study (Figure 2) that both $C_2$ and L have a major impact on the (radial) artery pressure wave contour. The higher $C_2$ and the lower L, the higher the oscillatory nature of the diastolic part of the pressure wave. Thus, when fitting a measured wave contour, both parameters may not be independent. We found a significant correlation between $C_2$ and 1/L ($r=0.36; P<0.05$). L represents the inertia of blood and depends on blood density and vessel cross-sectional area (L$\approx$pl/A). It is therefore expected that L varies inversely with body mass and BSA. Indeed, the analysis did uncover a negative though nonsignificant correlation ($r=-0.23, P=0.13$ and $r=-0.19, P=0.22$ for body mass and BSA, respectively). L also varied over a relatively wide range between 0.016 and 0.22 mm Hg/(mL/s$^2$). This would imply that within this population, cross-sectional area and vessel diameter in individuals may differ by a factor 13.8 in area and 3.7 in diameter, respectively. This variation is rather high to represent true, physical arterial differences.

**TABLE 1. Hemodynamic Data and AIx**

<table>
<thead>
<tr>
<th>Type</th>
<th>Age</th>
<th>SBP</th>
<th>DBP</th>
<th>MAP</th>
<th>CO</th>
<th>HR</th>
<th>AIx</th>
<th>$R$</th>
<th>$C_1$</th>
<th>$C_2$</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>32</td>
<td>111</td>
<td>71</td>
<td>90</td>
<td>6.8</td>
<td>68</td>
<td>0.19</td>
<td>0.79</td>
<td>1.77</td>
<td>0.056</td>
<td>0.026</td>
</tr>
<tr>
<td></td>
<td>29</td>
<td>100</td>
<td>70</td>
<td>87</td>
<td>8.2</td>
<td>80</td>
<td>-0.13</td>
<td>0.64</td>
<td>2.51</td>
<td>0.145</td>
<td>0.011</td>
</tr>
</tbody>
</table>

SBP indicates systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; CO, cardiac output; and HR, heart rate.

As reported by Muro et al (subjects A-5 and C-2) and 4-element WindKessel parameters derived on calculated radial aorta pressure waves (see also Figure 3). See Table 2 for dimensions.

**TABLE 2. Hemodynamic Data and 4-Element WindKessel Parameters**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Baseline (n=45)</th>
<th>NTG (n=40)</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP, mm Hg</td>
<td>89±13</td>
<td>75±12</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>119±18</td>
<td>97±13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>68±13</td>
<td>60±13</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CO, L/min</td>
<td>5.3±1.0</td>
<td>5.5±0.9</td>
<td>0.91</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>72±17</td>
<td>74±17</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>$A_{\text{max}}$</td>
<td>0.20±0.21</td>
<td>0.06±0.17</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>$A_{\text{min}}$</td>
<td>0.16±0.16</td>
<td>0.04±0.12</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>R, mm Hg · mL$^{-1}$ · s$^{-1}$</td>
<td>1.05±0.30</td>
<td>0.84±0.22</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>$C_1$, mL/mm Hg</td>
<td>1.88±0.93</td>
<td>2.71±1.37</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>$C_2$, mL/mm Hg</td>
<td>0.071±0.042</td>
<td>0.080±0.036</td>
<td>0.10</td>
</tr>
<tr>
<td>L, mm Hg · mL$^{-2}$ · s$^{-2}$</td>
<td>0.066±0.045</td>
<td>0.068±0.050</td>
<td>0.88</td>
</tr>
</tbody>
</table>

SBP indicates systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; CO, cardiac output; and HR, heart rate.

Derived from invasively measured aorta and radial artery pressure waves during baseline conditions and after NTG ($P$ values, baseline vs NTG).
introduce errors of \(\approx 50\%\) of the mean in R and, consequently, in \(C_1\), \(C_2\), and \(L\). On average, R is underestimated in our population by \(\approx 10\%\). Consequently (Equations 3 to 5), \(C_1\) and \(C_2\) will be overestimated (10%), whereas L is underestimated (10%). In the subgroup of 24 patients, \(C_2\) is 0.070\(\pm\)0.042 mL/mm Hg with the use of measured CO versus 0.076\(\pm\)0.040 mL/mm Hg with the use of estimated CO, and both values are highly correlated \((r=0.91, P<0.0001)\). The correlation between AIx meas and \(C_2\) in this group of 24 subjects improved with the use of measured CO \((r=-0.49, P=0.015 \text{ versus } r=-0.39, P=0.06)\). For AIx rec, the correlation coefficient increased from 0.22 to 0.30.

The augmentation index is measured directly on the aortic pressure wave. It is an index based on the hemodynamic principle of pressure wave propagation and reflection and quantifies the relative contribution of the reflected wave to the total pressure. With carotid tonometry or carotid wall distension used as a surrogate for aortic pressure, noninvasive assessment is possible. It has been shown that AIx changes with age\(^2\) and in hypertension.\(^{13,14}\) Alternatively, one may also estimate the augmentation index by using aortic pressure curves that are reconstructed from noninvasive radial artery pressure and a generalized pressure transfer function. It was shown, for instance, that AIx derived from the reconstructed curves is higher in type-1 diabetic than in control subjects.\(^{13}\) Chen et al\(^{10}\) reported that AIx, when calculated from reconstructed curves, was significantly lower than when calculated directly from the measured aortic pressure wave (0.19\(\pm\)0.12 versus 0.26\(\pm\)0.10, \(P<0.05\)) but no correlation coefficients were given in this study. More recently, Segers et al\(^{16}\) reported a correlation coefficient of 0.66 between AIx derived from measured and reconstructed pressure curves, with a small nonsignificant underestimation \((-0.03)\) with the use of the reconstructed curve. In this study, at baseline, we found a correlation coefficient of 0.66 between AIx meas and AIx rec, with a significant underestimation of AIx meas. The correlation was weaker during NTG administration, suggesting an increased variability by the application of the generalized pressure transfer function in these conditions. Overall, the correlation between AIx meas and AIx rec is acceptable, but the Bland-Altman plot in Figure 4 shows that large individual differences may exist between AIx derived from measured or reconstructed aortic pressure.

Both \(C_2\) and AIx quantify, in some way, the radial and aortic pressure wave contour and can be said to be associated with similar cardiovascular risk factors. We have shown that assuming a generalized pressure transfer function, C-type aortic waves correspond to radial artery pressure waves with a higher oscillatory character (ie, higher \(C_2\)) than A-type waves and vice versa. We further found a significant correlation between \(C_2\) and AIx, independent of AIx being derived from a directly measured or reconstructed aortic pressure. This suggests that the contour of the radial artery pressure wave is, at least in part, determined by the same factors affecting the central aortic pressure wave. Nevertheless, though correlations between \(C_2\) and AIx were significant \((P<0.001)\), they remain rather low \((r=0.36)\). We believe that at least 2 factors may have played a role. First, the relation between radial artery and aortic pressure is not constant; there is considerable scatter in individual transfer functions.\(^{8,10,11,16}\) Second, \(C_2\) and L both reflect the oscillatory nature of the pressure wave. Their cross-correlation may affect the individual correlation between \(C_2\) and AIx.

If \(C_2\) and AIx both reflect changes in the arterial pressure waveform with potential prognostic information on cardiovascular risk, the most sensitive and easily applicable index is to be preferred. The effect of NTG was most clear for AIx, its value lowering from 0.20 to 0.07 \((P<0.01)\), that is, almost a 3-fold change. \(C_2\) increased by 13% after administration of NTG, but the difference was not significant, possibly because of the large variability in \(C_2\) (Table 2). By contrast, large-artery compliance, \(C_1\), increased significantly from 1.88 to 2.71 mL/mm Hg (an increase of 44%). This variability was somewhat reduced after normalization of \(C_2\) with respect to BSA or body mass index (BMI), and the differences between baseline and NTG became somewhat clearer \((C_2/\text{BSA}: 0.036\pm0.020\)
versus 0.041±0.017 mL · mm Hg⁻¹ · m⁻², P=0.07; C₂/BMI: 0.00245±0.00136 versus 0.00282±0.00118 mL/mm Hg/[kg/m²], P<0.05). The correlation between normalized C₂ and AIx did not improve.

Conclusions
We have shown that the 4-element Windkessel parameter C₂, used to quantify the oscillatory character of the radial artery pressure wave, is related to the augmentation index, characterizing the central aorta wave shape and quantifying wave reflection. C₂ therefore reflects, at least in part, hemodynamic changes affecting central aortic pressure. Nevertheless, given the model assumptions and computational steps associated with calculating C₂, AIx may be a more appropriate parameter to use in the clinical setting as it is determined directly from the pressure wave contour.

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References
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