Use of pulse pressure method for estimating total arterial compliance in vivo

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Stergiopulos, N., P. Segers, and N. Westerhof. Use of pulse pressure method for estimating total arterial compliance. Am. J. Physiol. 276 (Heart Circ. Physiol. 45): H424–H428, 1999.—We determined total arterial compliance from pressure and flow in the ascending aorta of seven anesthetized dogs using the pulse pressure method (PPM) and the decay time method (DTM). Compliance was determined under control and during occlusion of the aorta at four different locations (iliac, renal, diaphragm, and proximal descending thoracic aorta). Compliance of PPM gave consistently lower values (0.893 ± 0.015) compared with the compliance of DTM (means ± SE; r = 0.989). The lower compliance estimates by the PPM can be attributed to the difference in mean pressures at which compliance is determined (mean pressure, 81.0 ± 3.6 mmHg; mean diastolic pressure, over which the DTM applies, 67.0 ± 3.6 mmHg). Total arterial compliance under control conditions was 0.169 ± 0.007 ml/mmHg. Compliance of the proximal aorta, obtained during occlusion of the proximal descending aorta, was 0.100 ± 0.007 ml/mmHg. Mean aortic pressure was 80.4 ± 3.6 mmHg during control and 102 ± 7.7 mmHg during proximal descending aortic occlusion. From these results and assuming that upper limbs and the head contribute as little as the lower limbs, we conclude that 60% of total arterial compliance resides in the proximal aorta. When we take into account the inverse relationship between pressure and compliance, the contribution of the proximal aorta to the total arterial compliance is even more significant.

METHODS

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The total arterial compliance of the systemic arterial tree is an important determinant of the cardiac load. Alterations in the total arterial compliance are linked to various physiological (aging) or pathological (hypertension) states, and thus the estimation of total arterial compliance has long interested clinicians and researchers of cardiovascular physiology (4, 6, 7, 9, 18–20).

The most widely used and accepted method is the diastolic decay time method (DTM), which, on the basis of the two-element windkessel model (4), fits a monoeponential curve to the time decay of the diastolic pressure (9, 10). To avoid problems that arise when aortic pressure does not decay monoexponentially, the area method was suggested (7). Under most conditions, the two methods yield identical results. Although the diastolic decay time and area methods are simple to use, they can only be applied when flow in diastole is zero, limiting them to measurements in the ascending aorta only. Another inconvenience of the diastolic decay and area methods is that they require knowledge of the diastolic aortic pressure waveform, which, at present, is difficult to obtain accurately noninvasively.

Recently, Stergiopulos et al. (11) proposed the pulse pressure method (PPM) for estimating arterial compliance. The PPM is based on simultaneous measurements of flow and pulse pressure (see detailed description of the method in METHODS), but it does not necessitate knowledge of the whole pressure pulse and it does not require zero flow in diastole. Therefore, it may be more adapted to in vivo applications. The PPM was tested for a variety of simulated physiological and pathological conditions using an extensive nonlinear distributed model of the arterial tree (13). Comparison with six other compliance estimation methods, including the decay time and area methods, showed that overall the PPM is the most accurate method (12). Good correlation, however, was found between the PPM estimates and the DTM estimates. We have never tested, however, the use of the PPM in the intact animal.

In the present study we evaluate the performance of the PPM using data from canine experiments under control conditions and under total aortic occlusion at different aortic locations. Because in the intact animal compliance is unknown in an absolute sense, we chose to compare the PPM with the DTM, the latter being generally accepted as a standard method for estimating total arterial compliance. The results will be used to: 1) assess the applicability of the PPM in vivo, and 2) evaluate the relative contribution of the proximal aorta on the total compliance of the arterial tree.

METHODS

Pulse pressure method. For an extensive description of the PPM, we refer the reader to the original article (11). In brief, the PPM, shown schematically in Fig. 1, is implemented as follows. From the ratio of mean pressure over mean flow we derive peripheral resistance. Then, using measured flow as input to the two-element windkessel, we fit the predicted pulse pressure to the actual pulse pressure by adjusting compliance. Compliance adjustment is done by a simple “trial and error” type of approach knowing, however, that lower compliance yields larger pulse pressures. From that, a few iterations always suffice. The value of the compliance (C) that
Table 1. Hemodynamic data in baseline conditions

<table>
<thead>
<tr>
<th>Dog</th>
<th>HR, beats/min</th>
<th>$P_m$, mmHg</th>
<th>CO, ml/min</th>
<th>$R$, ml/min/mmHg</th>
<th>$C_{PPM}$, ml/mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>153 ± 3.3</td>
<td>104 ± 2.9</td>
<td>20.2 ± 0.5</td>
<td>5.17 ± 0.21</td>
<td>0.197 ± 0.005</td>
</tr>
<tr>
<td>2</td>
<td>130 ± 6.5</td>
<td>56 ± 7.9</td>
<td>24.7 ± 0.7</td>
<td>2.27 ± 0.32</td>
<td>0.176 ± 0.009</td>
</tr>
<tr>
<td>3</td>
<td>100 ± 3.9</td>
<td>73.4 ± 3.8</td>
<td>13.8 ± 0.9</td>
<td>3.38 ± 0.15</td>
<td>0.149 ± 0.009</td>
</tr>
<tr>
<td>4</td>
<td>129 ± 6.0</td>
<td>67.8 ± 2.8</td>
<td>17.3 ± 0.5</td>
<td>3.92 ± 0.18</td>
<td>0.135 ± 0.004</td>
</tr>
<tr>
<td>5</td>
<td>149 ± 0.7</td>
<td>90.6 ± 1.5</td>
<td>18.4 ± 0.8</td>
<td>4.92 ± 0.77</td>
<td>0.157 ± 0.024</td>
</tr>
<tr>
<td>6</td>
<td>85.1 ± 4.1</td>
<td>85.7 ± 1.5</td>
<td>19.7 ± 0.8</td>
<td>4.40 ± 0.25</td>
<td>0.247 ± 0.005</td>
</tr>
<tr>
<td>7</td>
<td>115 ± 2.6</td>
<td>82.5 ± 1.8</td>
<td>15.0 ± 0.3</td>
<td>5.51 ± 0.82</td>
<td>0.112 ± 0.002</td>
</tr>
</tbody>
</table>

Data are means ± SE. HR, heart rate; $P_m$, mean pressure; CO, cardiac output; $R$, resistance; $C_{PPM}$, compliance of pulse pressure method.
60% of its baseline value, showing that the proximal aorta forms the major part of the total arterial compliance. Unpaired t-tests showed that total arterial compliance is significantly different between control and occlusion at C and D (P < 0.05) but not at A and B.

Paired t-tests showed that the total arterial compliance estimated by the PPM is always slightly but significantly lower than the compliance estimated by the DTM (P < 0.0001). The estimates for interventions A–D, however, were not significantly different (P > 0.05).

Part of the difference between the compliance estimates given by estimates given by the PPM and the DTM may be attributed to the pressure dependence of compliance. Figure 4, bottom, shows the mean values of $P_m$ and $P_{avg,diast}$ for control and aortic occlusions. Under control conditions $P_m$ was $80.9 \pm 3.32$ mmHg (means ± SE), and $P_m$ used in the DTM was $67.1 \pm 3.6$ mmHg. Unpaired t-tests show that $P_m$ was significantly augmented (P value < 0.05) with respect to control only for occlusion at B, C, and D. For occlusion at the iliacs (group A) $P_m$ decreased slightly ($78 \pm 6.8$ mmHg, Table 1), but the difference with control was not significant (P = 0.066). As expected, the difference between $P_m$ and $P_{avg,diast}$ increased as aortic occlusion sites moved closer to the heart due to a loss in compliance and increase in wave reflections.
was at the proximal descending aorta (site moved centrally (Table 1). When total occlusion
Compliance decreased progressively as the occlusion
unchanged (0.170
sion at the iliacs, total compliance remains essentially
unchanged (0.170
mM/Hg. With total occlusion at the iliacs, total compliance remains essentially
unchanged (0.170 ± 0.020), suggesting that the lower limbs contribute very little to the total compliance. Compliance decreased progressively as the occlusion site moved centrally (Table 1). When total occlusion was at the proximal descending aorta (group D), mean total arterial compliance was 0.100 ± 0.007 ml/mmHg.

DISCUSSION

We have used aortic pressure and flow data from in vivo experiments in anesthetized dogs to compare compliance estimates obtained by the PPM and the DTM. We found a good correlation between the PPM estimates and the DTM estimates. The PPM estimates compliance at mean pressure, whereas the DTM estimates compliance at the average pressure over the diastolic portion of the wave where the DTM is applied. Therefore, given the inverse relationship between pressure and compliance in the physiological pressure range (5), PPM yields lower compliance estimates in comparison to the DTM. If the pressure dependence of compliance is taken into account, the PPM and the DTM seem to yield coherent results following the same compliance-pressure curve (Fig. 5).

Although the PPM and the DTM may seem to be both equally applicable to in vivo situations, we foresee certain advantages in the use of the PPM compared with the DTM. First, the PPM yields the compliance at mean pressure, which is most relevant from a physiological standpoint. Mean compliance and peripheral resistance are the major determinants of heart load (3). Second, the PPM requires the knowledge of aortic pulse pressure only (not even the absolute values of systolic and diastolic pressures) compared with the whole pressure curve in diastole, which is necessary for the DTM. Calibrated aortic pressure is difficult to obtain noninvasively. We may speculate that aortic pulse pressure might be able to be predicted accurately by means of noninvasive measurements (brachial sphygmomanometry, tonometric measurements in the carotids, etc.), whereas the precise diastolic pressure decay in the aorta is difficult to obtain accurately. Third, aortic flow required by the PPM may be obtained noninvasively using magnetic resonance imaging or ultrasound. Fourth, the PPM does not require zero flow in the diastole, and therefore it applies equally well at other locations and in certain pathological cases such as aortic valve regurgitation. Fifth, there is certain ambiguity as to which part of the diastolic portion of the pressure wave should be used for the DTM. Preliminary analysis has shown that the compliance estimates by the DTM do depend on the choice of the diastolic part. There is no such ambiguity for the PPM which is very robust.

Application of the compliance estimation techniques in the clinical practice requires attention with respect to certain aspects of the arterial pulse. For example, the monoexponential character of the arterial pulse may be warranted only at the very late part of the diastolic wave. Wave reflections may play a role. It seems, however, that even in case of increased wave reflections in the aorta (as in the case of aortic occlusion), the PPM method yields valid compliance estimates. The most commonly used pressure measurement in the clinic is the brachial pulse pressure which, however, cannot be used as a surrogate to aortic pulse pressure. The

The pressure dependence of compliance can be evidenced under control conditions, where multiple independent measurements are available, in Fig. 5. We observe that when compliance estimates by the PPM and DTM are plotted against their corresponding pressures Pm and Pav,diast, they lie on a single curve. It is clear, however, that each dog has its own compliance-pressure curve. In this particular example, dogs 71 and 78 have similar compliance-pressure curves, whereas dog 79 operates on a totally different compliance-pressure curve. This makes interanimal comparison difficult.

Table 2. Hemodynamic parameters under control conditions and during interventions

<table>
<thead>
<tr>
<th>Type</th>
<th>n</th>
<th>Rm, mmHg·ml⁻¹·s⁻¹</th>
<th>Pm, mmHg</th>
<th>Cppm, ml/mmHg</th>
<th>Pav,diast, mmHg</th>
<th>Cdtm, ml/mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>6</td>
<td>4.0 ± 0.4</td>
<td>78 ± 6.7</td>
<td>0.170 ± 0.020</td>
<td>62 ± 8.1</td>
<td>0.172 ± 0.023</td>
</tr>
<tr>
<td>B</td>
<td>11</td>
<td>5.3 ± 0.5</td>
<td>96 ± 5.6</td>
<td>0.147 ± 0.013</td>
<td>77 ± 7.8</td>
<td>0.160 ± 0.017</td>
</tr>
<tr>
<td>C</td>
<td>6</td>
<td>6.0 ± 0.9</td>
<td>104 ± 10</td>
<td>0.119 ± 0.013</td>
<td>84 ± 11.1</td>
<td>0.127 ± 0.015</td>
</tr>
<tr>
<td>D</td>
<td>9</td>
<td>6.0 ± 0.8</td>
<td>102 ± 7.5</td>
<td>0.100 ± 0.007</td>
<td>78 ± 8.6</td>
<td>0.112 ± 0.013</td>
</tr>
<tr>
<td>X</td>
<td>46</td>
<td>4.2 ± 0.2</td>
<td>81 ± 3.3</td>
<td>0.169 ± 0.007</td>
<td>67 ± 3.6</td>
<td>0.186 ± 0.010</td>
</tr>
</tbody>
</table>

Data are means ± SE; n, number of dogs. Pav,diast, average pressure in diastolic portion of the wave.

Fig. 5. Evidence that PPM and DTM give compliance estimates belonging to same compliance-pressure curve. Depending on animal, compliance-pressure curves may be similar (dogs 71 and 78) or entirely different (dog 79).
arterial disease. Our results show that the proximal aorta (ascending aorta and aortic arch) forms the major determinant of the total arterial compliance. Although it is accepted that proximal aortic compliance forms a large part of total arterial compliance, quantitative data have, to the best of our knowledge, not been presented. When the dog aorta was occluded at the level of the proximal descending aorta (point D), compliance was reduced from 0.169 ml/mmHg under control conditions to 0.100 ml/mmHg, a 41% decrease in value. At the same time, mean pressure increased from 80.9 mmHg under control conditions to 102.3 mmHg under total occlusion at point D. Thus, at control pressure (80.9 mmHg), mean compliance with the aorta occluded at point D would have been even larger. A good estimate of mean compliance with occlusion at point D at Pm = 80.9 mmHg can be obtained from Fig. 4, A and B: CDTM = 0.112 for Pm,k = 77.9 mmHg and thus, by simple linear interpolation, C(P = 80.9 mmHg) = 0.110 ml/mmHg. This means that ~65% of total arterial compliance is located in the proximal aorta and the head and upper limb vessels.

We may thus conclude that total arterial compliance is mainly determined by the properties of the proximal aorta. This may have important clinical implications. In the human aortic pulse pressure appears to be well predicted by stroke volume over compliance (2), and thus compliance is the primary determinant of pulse pressure. Pulse pressure appears to be a primary risk factor for coronary disease and cardiovascular mortality (1, 8). In this context it seems much more important to determine proximal aortic compliance than local carotic, radial, or brachial compliance. We may thus hypothesize that aortic compliance should be one of the target quantities for risk assessment and prevention of arterial disease.

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Received 27 May 1998; accepted in final form 13 October 1998.

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