A Comparison Between Systolic Aortic Root Pressure and Finger Blood Pressure*

Eddy G. Philippe, MD; Jean-Louis Hébert, MD, PhD; Catherine Coirault, MD, PhD; Karen Zamani, MD; Yves Lecarpentier, MD, PhD; and Denis Chemla, MD, PhD

**Background:** Digital photoplethysmography is used to assess hemodynamic variability and baroreflex sensitivity. Numerous studies have critically evaluated the accuracy of the photoplethysmographic device against peripheral pressure. The aim of our study was to compare finger blood and aortic root pressure.

**Methods:** We prospectively compared simultaneous recordings of systolic pressure at the aortic root and finger level over three consecutive respiratory cycles in 15 patients (56±11 years) undergoing routine cardiac catheterization. Data were obtained at baseline, during deep breathing maneuver (0.1 Hz), and after left ventricular cineangiography.

**Results:** At baseline, systolic finger pressure overestimated systolic aortic pressure (145.2±22.5 vs 115.0±20.1 mm Hg; p<0.001). The pressure difference (30.2±17.0 mm Hg) was not influenced by systolic aortic pressure. There was no relationship between pressure difference and the main determinants of the pulse wave amplification phenomenon. There was a beat-to-beat relationship between finger and aortic pressure in 14 of 15 subjects (slope ranging from 0.37 to 1.70; ordinate: from −56 to +98 mm Hg). During the deep breathing maneuver and after left ventricular cineangiography, finger pressure still overestimated aortic pressure by 32.5±15.0 mm Hg and 38.3±13.9 mm Hg, respectively (each p<0.001). There was a beat-to-beat relationship between systolic aortic root pressure (IAoBP) and systolic finger (FBP) in 13 of 15 patients, with major scattering of both slopes and ordinates. Throughout the study, there was no predictable relationship between the level of IAoBP and pressure bias.

**Conclusions:** As expected, FBP was almost always higher than IAoBP. Importantly, the differences in systolic pressure did not correlate with known determinants of the pulse wave amplification phenomenon. The device must be used cautiously if one wants to noninvasively track spontaneous or induced changes in IAoBP. *(CHEST 1998; 113:1466-74)*

**Key words:** aorta; beat-to-beat analysis; finger BP; photoplethysmography; pulse wave amplification

**Abbreviations:** Cvar=coefficient of variation; FBP=systolic finger BP; IAoBP=systolic aortic root pressure; ΔIAoBP=systolic aortic root pressure variation; LV=left ventricular; LVET=left ventricular ejection time; LVETi=heart rate-corrected left ventricular ejection time; PWA=pulse wave amplification

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Photoplethysmographic devices are currently used to measure the finger BP by using the volume clamp method described by Penaz.1,2 In numerous studies, photoplethysmographic devices have exhibited a reasonably good ability to accurately track intra-arterial BP both at baseline and under dynamic conditions.3-7 Others have reported significant pressure bias between photoplethysmographic and intra-arterial recordings, such that these devices cannot be recommended to monitor peripheral pressure in anesthetized patients.8-11 However, numerous clinical and physiologic studies still use these devices to assess hemodynamic variability and baroreflex sensitivity.

Finger systolic BP (FBP) may not necessarily reflect systolic aortic root BP (IAoBP), given that there is a physiologic amplification of systolic pressure from the aortic root to the radial artery.12-15 Arterial pressure wave is determined by the amplitude and timing of both the incident wave (which travels forward from heart to periphery) and the
reflected wave (traveling backwards from peripheral reflecting sites to the heart).\textsuperscript{15-18} In young normo-
tensive adults, the reflected wave causes an increase in aortic pressure, occurring in early diastole; con-
versely, peripheral arteries are close to the reflecting site, such that the incident and reflected pressure waves produce an additive effect. Thus, the arterial systolic pressure wave is amplified from the aortic root to the radial artery, while mean arterial pressure is nearly constant or drops slightly (<5 mm Hg). The extent of this pulse wave amplification (PWA) de-

depends mainly on total arterial compliance of the systemic arterial tree and effective arterial length.\textsuperscript{15-22} PWA is also influenced by age, body length, BP, heart rate, left ventricular ejection time (LVET), and vasomotor tone.\textsuperscript{12-25}

The aim of our prospective, time-domain study was to document how FBP compares with IAoBP. To this end, digital photoplethysmography and intra-aortic catheter recordings were simultaneously obtained in unselected patients undergoing routine left heart catheterization. Patients were studied both at baseline and under dynamic conditions (deep-

breathing maneuver, after left ventricular [LV] cineangiography period). We tested the following hypotheses: (1) that FBP overestimated IAoBP, as expected given the PWA phenomenon; (2) that pressure offset was related to the known determinants of the PWA phenomenon; and (3) that FBP tracked IAoBP reliably, on a beat-to-beat, short-term basis.

\section*{Materials and Methods}

\subsection*{Patients}

Fifteen consecutive patients (11 men and 4 women) were enrolled in this study after informed consent had been obtained. Patients were scheduled for elective cardiac catheterization and coronary angiography. The study was approved by our institutional review committee. Patients with rhythm disturbances, valvular heart diseases, and Raynaud’s phenomenon were excluded from the study. The clinical characteristics of the study group were as follows (mean\(\pm SD\)): age, 56\(\pm\)11 years; body weight, 70\(\pm\)9 kg; body height, 1.68\(\pm\)0.05 m; body surface area, 1.77\(\pm\)0.12 m\(^2\). Two patients were free of cardiovascular drug therapy. Otherwise, patients were receiving \(\beta\)-adrenoceptor blocking agents (n=6), angiotensin-converting enzyme inhibitors (n=4), calcium channel blockers (n=3), nitrates (n=7), and diuretics (n=1). The underlying conditions were diabetes mellit-

\section*{Catheterization Technique}

Catheterization was performed using the femoral percutaneous Seldinger technique with a 6F sheath. A single lumen, 0.035-inch supertorque plus multipurpose 6F catheter (Cordis Corporation; Miami) was used. The sheath was introduced 10 to 20 mm under Poupart’s ligament, under local anesthesia (2% lidocaine). The catheter was advanced to the aortic root, 1 cm above the aortic cusps, for the intra-aortic BP measurements. The catheter was connected to a single-use pressure transducer (DPT 6003; ISSA Laboratory, Smiths Industries Medical System; Keene, NH) and the pressure transducer was, in turn, connected to a recorder (Honeywell VR12; Hagerstown, Md). We performed static cali-

bration of the pressure transducer, fixed at the right atrial level and adjusted to atmospheric zero pressure. The system was flushed continuously. The natural frequency (15 Hz) and damping coefficient (0.4) of the catheter-tubing-transducer system conformed with recommendations for adequate dynamic pressure response.\textsuperscript{26}

\subsection*{Finger BP Measurements}

Finger BP was measured with a monitor (Ohmeda Finapres 2300 NIBP; Denver). This monitor uses Marey’s principle of unloading of the arterial wall, implemented by the volume clamp method of Penaz.\textsuperscript{12} This method uses an air-filled finger cuff coupled with a photoelectric plethysmograph to measure arterial size. Cuff pressure is regulated by a fast-acting servo-control system and maintains the arteries in an unloaded state. The display screen shows the continuous wave form and indicates heart rate, systolic, diastolic, and mean arterial pressures. The measurement range of finger pressure is from 20 to 260 mm Hg (with 1 mm Hg resolution). The appropriate cuff (small, medium, and large) was wrapped to the middle phalanx of the middle finger with infrared diodes positioned on the sides of the phalange. The box containing the electropneumatic transducer was strapped to the back of the left hand. To prevent hydrostatic pressure differences, both the electropneumatic transducer box and the systemic pressure transducer were fixed at the right atrial level. In cases where the optic plethysmogram signal was damped, the cuff was repositioned after transitory deflation.

\subsection*{Respiratory Inductive Plethysmography}

Respiratory cycles were determined with a respiratory inductive plethysmograph (Respiritrace; Ambulatory Monitoring; Ards-

ley, NJ).\textsuperscript{27} Changes in rib cage circumference after the self-

conductance of a wire coil sewn onto an elastic belt, the changes in conductance being converted into proportional voltage changes. The elastic belt was fixed around the thorax and under the armpit and connected to an oscillator. This device allows continuous measurement of thoracic displacement, and thus, monitors respiratory cycle determination.

\subsection*{LV Cineangiography and Coronary Angiography}

Selective coronary angiographies and two consecutive, orthog-

\subsection*{Protocol and Data Analysis}

Patients were studied in the early morning, at least 12 h after

previous intake of current medications. Simultaneous recordings
of ECG, aortic root pressure, finger BP, and respiratory trace were A/D converted at a sample rate of 500 Hz and input to a personal computer (Toshiba 3,200 SX, Tokyo, Japan) with custom-made software. Data were obtained over three consecutive respiratory cycles (average number of heart beats analyzed=14±6). Data were obtained following three consecutive protocols: protocol 1—the data were first obtained at rest, before angiography, the patient being asked to breathe normally; protocol 2—thereafter, the measurements were made during deep-breathing maneuver (0.1 Hz); and protocol 3—immediately after completion of coronary angiography and LV cineangiography, the catheter was withdrawn from the left ventricle to the aortic root, and zero pressure level was checked. Thereafter, data were computed. Because all 15 patients completed the three protocols, a total of 45 protocols were analyzed.

We measured IAoBP and FBP. We calculated aortic and finger mean pressure (ie, the area under the pressure curve divided by the pulse interval) and aortic pulse pressure (ie, IAoBP minus diastolic pressure). We also measured LVET (ie, the time from the foot of the aortic pressure upstroke to the incisure), and heart rate-corrected LVET (LVETi), in accordance with standard formulas. Stroke volume was calculated as end-diastolic minus end-systolic volume. Aortic compliance was estimated by using the stroke volume over aortic pulse pressure ratio. We calculated absolute pressure bias between finger and aortic root (FBP-IAoBP, expressed in millimeters of mercury) and relative pressure bias (100×(FBP-IAoBP)/IAoBP, expressed in percent). We also calculated the coefficient of variation (Cvar) (ie, SD/mean ratio) for both FBP (Cvar FBP) and IAoBP (Cvar IAoBP) over three consecutive respiratory cycles. Systolic aortic pressure variation (ΔIAoBP) was defined as the difference between the maximal value of IAoBP and the minimal value of IAoBP over the three consecutive respiratory cycles.

**Statistical Analysis**

Data were averaged over three consecutive respiratory cycles and were expressed as mean±SD. In the overall population, comparisons between FBP and IAoBP were performed by using the paired Student t test. Linear regressions were obtained by using the least squares method. The agreement between FBP and IAoBP measurements was assessed (1) by calculating the 95% confidence interval for the (FBP-IAoBP) difference, and (2) by plotting the (FBP-IAoBP) difference against IAoBP. A p value <0.05 was considered statistically significant.

**RESULTS**

Standard hemodynamics of the study population are listed in Table 1.

**Comparison Between FBP and IAoBP (n=15)**

Individual IAoBP and systolic BP bias (FBP-IAoBP) are given in Table 2. During the control period at rest, IAoBP ranged from 90.9 to 160.3 mm Hg, and FBP ranged from 110.6 to 192.3 mm Hg. On average, FBP (145.2±22.5 mm Hg) significantly overestimated IAoBP (115.0±20.1 mm Hg) (p<0.001), with a 30.2±17.0 mm Hg systolic BP bias (95% confidence interval ranging from −3.1 to 63.5 mm Hg). Relative systolic pressure bias was 28±17% (95% confidence interval ranging from −6% to 62%). There was no significant relationship between IAoBP and systolic pressure bias (Fig 1, top) or relative pressure bias (Table 3). There was no significant relationship between either pressure bias or relative pressure bias on the one hand, and age, weight, height, heart rate, LVET, LVETi, stroke volume, ejection fraction, mean aortic pressure, pulse aortic pressure, and

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**Table 1—Standard Hemodynamic Results**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Heart Period, ms</th>
<th>LVETi, ms</th>
<th>EDV, mL</th>
<th>EF, %</th>
<th>SV, mL</th>
<th>MAoP, mm Hg</th>
<th>PAoP, mm Hg</th>
<th>Aortic Compliance, mL/mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>854</td>
<td>467</td>
<td>147</td>
<td>52</td>
<td>76</td>
<td>80.0</td>
<td>53.7</td>
<td>1.42</td>
</tr>
<tr>
<td>2</td>
<td>892</td>
<td>444</td>
<td>60</td>
<td>55</td>
<td>51</td>
<td>78.2</td>
<td>82.0</td>
<td>0.62</td>
</tr>
<tr>
<td>3</td>
<td>948</td>
<td>455</td>
<td>238</td>
<td>25</td>
<td>60</td>
<td>71.5</td>
<td>53.2</td>
<td>1.12</td>
</tr>
<tr>
<td>4</td>
<td>1,056</td>
<td>454</td>
<td>140</td>
<td>74</td>
<td>104</td>
<td>87.9</td>
<td>69.8</td>
<td>1.48</td>
</tr>
<tr>
<td>5</td>
<td>936</td>
<td>417</td>
<td>134</td>
<td>46</td>
<td>62</td>
<td>73.8</td>
<td>32.7</td>
<td>1.89</td>
</tr>
<tr>
<td>6</td>
<td>1,045</td>
<td>482</td>
<td>164</td>
<td>84</td>
<td>138</td>
<td>87.3</td>
<td>64.0</td>
<td>2.15</td>
</tr>
<tr>
<td>7</td>
<td>714</td>
<td>451</td>
<td>—</td>
<td>—</td>
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<td>445</td>
<td>185</td>
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<td>69.9</td>
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<td>126</td>
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<td>55.2</td>
<td>1.99</td>
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<td>—</td>
<td>—</td>
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<td>—</td>
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<td>851</td>
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<td>87</td>
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</tr>
<tr>
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<td>919</td>
<td>480</td>
<td>184</td>
<td>65</td>
<td>120</td>
<td>90.4</td>
<td>62.0</td>
<td>1.93</td>
</tr>
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<td>13</td>
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<td>502</td>
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<td>83</td>
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<td>64.5</td>
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<td>56.4</td>
<td>2.83</td>
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<td>15</td>
<td>910</td>
<td>477</td>
<td>172</td>
<td>68</td>
<td>117</td>
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<td>37.0</td>
<td>3.16</td>
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<td>Mean</td>
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<td>457</td>
<td>156</td>
<td>63</td>
<td>94</td>
<td>82.3</td>
<td>52.9</td>
<td>1.82</td>
</tr>
<tr>
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<td>122</td>
<td>23</td>
<td>44</td>
<td>20</td>
<td>34</td>
<td>7.5</td>
<td>16.7</td>
<td>0.79</td>
</tr>
</tbody>
</table>

*EDV=end-diastolic volume; EF=ejection fraction; SV=stroke volume; EDV, EF, and SV were determined by using LV cineangiography. MAoP=mean aortic pressure; PAoP=pulse aortic pressure averaged out over three consecutive respiratory cycles. Aortic compliance was calculated as the SV/PAoP ratio.
the stroke volume/pulse pressure ratio on the other (Table 3). At baseline, the device (Finapres) significantly overestimated diastolic aortic pressure (pressure bias=20.9±10.5 mm Hg) and mean aortic pressure (pressure bias=18.2±14.8) (each p<0.001).

During the deep-breathing maneuver, both I AoBP and FBP decreased (each p<0.001) while heart rate was not significantly modified (65±9 beats/min before vs 68±8 beats/min during the maneuver). The FBP overestimated I AoBP (p<0.001), with a 32.3±15.0 mm Hg pressure bias (33.1±17.2%).

After LV cineangiography, both I AoBP and FBP remained unchanged while heart rate increased (78±12 beats/min) (p<0.001). The FBP overestimated I AoBP (p<0.001), with a 38.3±13.9 mm Hg pressure bias (36.6±17.1%). The pressure bias was of similar magnitude at rest, during deep-breathing maneuver and after LV cineangiography. Both during the deep-breathing maneuver and after LV cineangiography, there was no significant relationship between pressure bias and I AoBP (Fig 1, center and bottom, respectively).

**Beat-to-Beat Relationship Between FBP and I AoBP Over Three Consecutive Respiratory Cycles**

In each patient during control period at rest, I AoBP variations ranged from 3 to 14 mm Hg (mean±SD=8±3 mm Hg). There was a significant, linear beat-to-beat relationship between FBP and I AoBP in 14 of 15 subjects (r ranging from 0.59 to 0.98, p<0.05 to p<0.001). There was no significant relationship between I AoBP and FBP in one patient. The slope of the I AoBP vs FBP relationship ranged from 0.37 to 1.70 and the ordinate ranged from −56.2 to 98.3 mm Hg, thus indicating major scattering in the individual relationship between I AoBP and FBP. There was no relationship between the slope of the I AoBP vs FBP relationship and the known determinants of the PWA phenomenon. During deep breathing, there was a significant, linear beat-to-beat relationship between FBP and I AoBP in 13 of 15 subjects (r ranging from 0.50 to 0.99, p<0.05 to p<0.001). The slope of the I AoBP vs FBP relationship and the ordinate were also markedly scattered (ranging from 0.70 to 1.43 and from −42 to +74.7 mm Hg, respectively). After LV cineangiography, there was a significant, linear beat-to-beat relationship between FBP and I AoBP in 13 of 15 subjects (r ranging from 0.61 to 0.98, p<0.01 to p<0.001). The slope of the I AoBP vs FBP relationship and the ordinate were also markedly scattered (ranging from −3.5 to 2.36 and from −142.4 to +189 mm Hg, respectively).

In some patients, the I AoBP variation was <5 mm Hg, and this would reduce both the probability and the physiologic relevance of any correlation with FBP. Thus, we performed a subgroup analysis involving only those patients whose I AoBP variation was ≥5 mm Hg (Table 4). There was no significant relationship between the Cvar of I AoBP and Cvar FBP both at rest and during the

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**Table 2—Intra-Aortic Systolic BP (I AoBP) and BP Bias Between FBP and I AoBP at Baseline, During Deep-Breathing Maneuver, and After LV Cineangiography**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Rest I AoBP</th>
<th>FBP-I AoBP</th>
<th>Deep Breathing I AoBP</th>
<th>FBP-I AoBP</th>
<th>Postangiography I AoBP</th>
<th>FBP-I AoBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>112.3</td>
<td>1.7</td>
<td>108.5</td>
<td>5.4</td>
<td>115.7</td>
<td>39.8</td>
</tr>
<tr>
<td>2</td>
<td>160.3</td>
<td>32.0</td>
<td>129.1</td>
<td>31.9</td>
<td>81.7</td>
<td>44.8</td>
</tr>
<tr>
<td>3</td>
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</tr>
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</tr>
<tr>
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<td>107.4</td>
<td>30.0</td>
<td>161.1</td>
<td>14.4</td>
</tr>
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<tr>
<td>Mean</td>
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<tr>
<td>SD</td>
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<td>14.5</td>
<td>15.0</td>
<td>21.2</td>
<td>13.9</td>
</tr>
</tbody>
</table>

*For each patient, the values presented are mean values, the pressure data being averaged over three consecutive respiratory cycles.
Influence of IAoBP on Beat-to-Beat Pressure Bias

No significant relationship between beat-to-beat IAoBP and the pressure bias was found in 26 of 45 protocols, namely in 9 of 15 patients at baseline, 10 of 15 patients during the deep-breathing maneuver, and 7 of 15 patients after LV cineangiography (Fig 2). Otherwise, this relationship was either negative (16 of 45 protocols) or positive (3 of 45 protocols). Overall, these results suggested that there was no simple, predictable relationship between the level of IAoBP and pressure bias.

**Discussion**

As expected, FBP significantly overestimated IAoBP in patients undergoing left heart catheterization. However, contrary to expectations, pressure bias was unpredictable, as it was not related to known determinants of the PWA phenomenon. Although beat-to-beat FBP tracked IAoBP in 14 of 15 patients, the beat-to-beat offset was random, with major scattering of the IAoBP vs FBP relationship. Similar results were observed during the deep-breathing maneuver and after LV cineangiography. Thus, the device must be used cautiously if one wants to noninvasively track spontaneous or induced changes in aortic systolic BP.

**Overestimation of IAoBP by FBP at Rest: the Potential Role of the PWA Phenomenon**

The FBP overestimated IAoBP at rest (mean bias ± SD = 30.2 ± 17.0 mm Hg). The role of the PWA phenomenon\[12-25\] needs to be discussed. On average, the amount of systolic BP amplification between the aorta and radial artery is about 20 mm Hg in healthy subjects\[13,14,19\] and 10 mm Hg in patients with congestive heart failure.\[20\] The PWA phenomenon is more marked in clinical conditions associated with reduced wave reflections\[12-16,19,20,35\] (eg, exercise, Valsalva maneuver). Conversely, in cases of increased pulse wave velocity due to arterial stiffening, the reflected wave reaches the aortic root earlier and boosts pressure during the late systolic period, thus

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**Table 3—Correlation Matrix for BP Bias (FBP-IAoBP) and for Relative Pressure Bias (FBP-IAoBP)/IAoBP at Rest**

<table>
<thead>
<tr>
<th></th>
<th>FBP-IAoBP</th>
<th>(FBP-IAoBP)/IAoBP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>−0.39</td>
<td>−0.48</td>
</tr>
<tr>
<td>Body height</td>
<td>+0.30</td>
<td>+0.35</td>
</tr>
<tr>
<td>Heart period</td>
<td>−0.19</td>
<td>−0.18</td>
</tr>
<tr>
<td>EF</td>
<td>−0.22</td>
<td>−0.30</td>
</tr>
<tr>
<td>LVETi</td>
<td>−0.19</td>
<td>−0.31</td>
</tr>
<tr>
<td>SV</td>
<td>−0.16</td>
<td>−0.18</td>
</tr>
<tr>
<td>Mean aortic BP</td>
<td>−0.25</td>
<td>−0.40</td>
</tr>
<tr>
<td>IAoBP</td>
<td>−0.27</td>
<td>−0.51</td>
</tr>
<tr>
<td>Aortic compliance</td>
<td>+0.10</td>
<td>+0.23</td>
</tr>
</tbody>
</table>

\(r^2\) values are listed. Each p = not significant. EF = ejection fraction; SV = stroke volume.

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**Figure 1.** Difference between FBP and simultaneous IAoBP at baseline (top), during the 0.1-Hz deep-breathing maneuver (center), and after LV cineangiography (bottom) in the 15 patients under study. The FBP-IAoBP difference was plotted against IAoBP. Mean values are presented with data averaged over three consecutive respiratory cycles. For the three tasks, the mean pressure difference (horizontal line) and +2 SD and −2 SD (dotted lines) are indicated.
cancelling out the PWA phenomenon\textsuperscript{15-18,23-25} (eg, in aged or hypertensive subjects). PWA has been found to be a function of the patterns of LV ejection and LVET.\textsuperscript{13,23-25} Recently, it has been suggested that changes in heart rate and LVET rather than changes in vascular tone and BP are the main determinants of PWA from brachial to finger arteries.\textsuperscript{36,37}

Contrary to expectations, there was no relationship between systolic BP bias and the main determinants of PWA (ie, patient’s age, body length, LVET, aortic pressure, stroke volume, ejection fraction, heart rate, and the stroke volume/pulse aortic pressure ratio). As a result, FBP did not furnish an accurate estimation of systolic BP at the aortic root level in patients undergoing heart catheterization, and this was insufficiently explained by the PWA phenomenon.

**Reliability of FBP Recording at Rest in Previous Studies**

One alternative hypothesis could be that systolic FBP was not an accurate reflection of peripheral arterial pressure in the patients we studied. Numerous studies have shown that the device (Finapres) follows intrabrachial and intraradial changes faithfully in both healthy and hypertensive subjects under steady-state conditions\textsuperscript{3-6} (Table 5). Imholz et al\textsuperscript{6} have found that the accuracy (mean bias) of systolic and diastolic finger BP is within the 5 mm Hg limit required by the Association for the Advancement of Medical Instrumentation,\textsuperscript{38} while systolic and diastolic finger BP precision (SD of mean bias) is insufficient (>8 mm Hg). Other studies suggest caution as to the accuracy of the device (Finapres) compared with either intrabrachial or intraradial pressures in hypertensive patients,\textsuperscript{39} in elderly patients,\textsuperscript{40} and in anesthetized patients.\textsuperscript{9,10} Very recently, it has been shown that an antiresonance waveform filter could remove waveform distortion from FBP tracings, making them more similar to intrabrachial recordings.\textsuperscript{41}

Thus, there is still an unresolved controversy as to whether the FBP gives an accurate reflection of peripheral arterial BP. This could be explained (1) by differences in study design and study population, and (2) by the necessary use of an intra-arterial catheter for upstream ipsilateral arteries that could induce a partial obstruction of the artery and could also cause a distal vasospasm.\textsuperscript{8} The latter methodologic concern did not apply in our study, where intra-arterial pressure was recorded at the aortic root level. Furthermore, Epstein et al\textsuperscript{10} have emphasized that there is no theoretical basis on which to predict the positive device bias (Finapres) that they reported for mean pressure. Consistently, we also found that the device significantly overestimated mean aortic pressure. Given that systolic pressure relates to both the steady and the pulsatile components of arterial pressure,\textsuperscript{15} systolic pressure bias could also be related to mean pressure bias due to a range of factors (eg, vasomotor tone of the exposed hand), hence affecting unloading pressure level.

**Unpredictable Offset of Beat-to-Beat Systolic FBP at Rest and During Dynamic Maneuvers**

Using a study design similar to ours in patients with coronary artery diseases, Hartikainen et al,\textsuperscript{7} reported a 22.7 mm Hg (FBP-IAoBP) pressure bias at rest, and our results are consistent with their findings. Interestingly, their Table 2 indicates a wide

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Table 4—Comparison Between Beat-to-Beat IAoBP and FBP Changes in the Patients With $\Delta$IAoBP $\geq$ 5 mm Hg$^*$

<table>
<thead>
<tr>
<th></th>
<th>$\Delta$IAoBP, mm Hg</th>
<th>$\Delta$IAoBP, mm Hg</th>
<th>CvarIAoBP, %</th>
<th>CvarFBP, %</th>
<th>r</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td>Mean ± SD</td>
<td></td>
</tr>
<tr>
<td>Rest</td>
<td>12</td>
<td>6-14</td>
<td>9 ± 3</td>
<td>2.7 ± 0.8</td>
<td>2.1 ± 0.7</td>
</tr>
<tr>
<td>Deep breathing</td>
<td>14</td>
<td>7-42</td>
<td>17 ± 10</td>
<td>4.6 ± 2.4</td>
<td>4.6 ± 2.1</td>
</tr>
<tr>
<td>Postangiography</td>
<td>10</td>
<td>6-33</td>
<td>15 ± 9</td>
<td>3.9 ± 2.3</td>
<td>2.4 ± 1.5</td>
</tr>
</tbody>
</table>

$^*$r=linear correlation coefficient of the CvarFBP vs CvarIAoBP relationship. $\Delta$IAoBP=Difference between the maximal value of IAoBP and the minimal value of IAoBP over three consecutive respiratory cycles; n=number of patients with $\Delta$IAoBP $\geq$ 5 mm Hg.

$^1p<0.05$ vs CvarIAoBP.

$^1p<0.01$; only statistical significance is shown.

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**Figure 2.** A typical example where no significant relationship between beat-to-beat IAoBP and the FBP-IAoBP difference was observed. This was found in 26 of 45 protocols.
range of pressure bias (from −40.0 to 53.0 mm Hg), thus also suggesting an unpredictable offset of systolic FBP at rest.7 Goldman et al42 have studied the physiologic, inspiratory fall of IAoBP and FBP in six patients exhibiting normal coronary angiography and LV function. At rest, they reported a wide 95% confidence interval for the difference between FBP inspiratory fall and IAoBP inspiratory fall, and this is consistent with a random, beat-to-beat pressure bias of the device (Finapres) over time.

It is generally recognized that the device (Finapres) faithfully tracks the peripheral, intraarterial BP changes during dynamic maneuvers, although large offsets may occur in some patients.3,5,6 However, during the strain phase of the Valsalva maneuver, FBP has been reported to either overestimating5,13 or as underestimating39 intrabrachial/radial BP, and differences in the characteristics of the measuring systems could explain such discrepant results.39 FBP overestimates peripheral BP during both exercise39,43 and phentylephrine vasoconstriction,44 while FBP underestimates peripheral BP after nitrates.39 Furthermore, brachial/radial BP monitoring is a poor guide to intra-aortic changes during these maneuvers, mainly due to changes in both reflection patterns and the PWA phenomenon.13,14,17

When isolated changes in either IAoBP or heart rate were considered during deep breathing and after LV cineangiography, the poor accuracy of FBP was confirmed. We also reported large differences in the trends of change between FBP and IAoBP on a beat-to-beat basis. Consistent with our results, Wilkes et al11 have reported that in 40% of cases, the variations in finger and in radial pressures are out of phase during spinal anesthesia. As far as baroreflex sensitivity is concerned, conflicting results have been published. Although they have reported a small but significant difference between invasive (intra-aortic) and noninvasive (FBP) baroreflex sensitivities, Hartikainen et al7 encourage the use of FBP. Conversely, Gizdulich et al11 have observed large differences in the baroreflex sensitivities obtained from unfiltered FBP and intrabrachial artery pressure during graded phentylephrine infusion in healthy volunteers. This could be explained by changes in PWA and the unpredictable, beat-to-beat pressure bias observed with FBP (Fig 2).

**Limitations**

The limitations of our study need to be discussed. First, the results apply strictly to the population under study, ie, unselected, middle-aged adult patients scheduled for left heart catheterization. We cannot exclude the possibility that FBP is a more accurate reflection of IAoBP in other populations. Second, beat-to-beat tracking of IAoBP by FBP was studied in the time domain only. Thus, our results cannot be extended to the frequency domain. Third, fluid-filled catheters, in contrast to transducer-tipped catheters, always have the potential to overestimate or underestimate IAoBP if not ideal in their dynamic response. Microscopic air in the flush system can introduce significant errors. However, in our study, the system was flushed continuously, and its characteristics conformed with recommendations for adequate dynamic pressure response.26 Further studies are needed to confirm the present results using high-fidelity, transducer-tipped catheters.

**Implications and Conclusion**

On average, FBP overestimated IAoBP by 30 mm Hg. The wide 95% confidence interval for the bias in our study is doubtless of clinical significance. Furthermore, FBP offset IAoBP in an unpredictable manner. As pressure bias was not related to the main determinants of the PWA phenomenon, it was not possible to propose an empiric formula so as to correct FBP for physiologic pressure amplification. In populations similar to ours, we believe that this calls into question the use of FBP as a substitute for IAoBP if one wants to calculate LV wall stress, LV elastance, or arterial elastance. The poor accuracy of the device was confirmed during the deep-breathing maneuver and after LV angiography. Furthermore,
on a beat-to-beat basis, the FBP vs IAoBP relationships were markedly scattered in our patients. As a result, beat-to-beat pressure bias was also unpredictable. Thus, in populations similar to ours, the device must be used cautiously if one wants to noninvasively track spontaneous or induced changes in aortic systolic BP.

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