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Models of brachial to finger pulse wave distortion and pressure decrement

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Abstract

Objective: To model the pulse wave distortion and pressure decrement occurring between brachial and finger arteries. Distortion reversion and decrement correction were also our aims. Methods: Brachial artery pressure was recorded intra-arterially and finger pressure was recorded non-invasively by the Finapres technique in 53 adult human subjects. Mean pressure was subtracted from each pressure waveform and Fourier analysis applied to the pulsations. A distortion model was estimated for each subject and averaged over the group. The average inverse model was applied to the full finger pressure waveform. The pressure decrement was modelled by multiple regression on finger systolic and diastolic levels. Results: Waveform distortion could be described by a general, frequency dependent model having a resonance at 7.3 Hz. The general inverse model has an anti-resonance at this frequency. It converts finger to brachial pulsations thereby reducing average waveform distortion from 9.7 (s.d. 3.2) mmHg per sample for the finger pulse to 3.7 (1.7) mmHg for the converted pulse. Systolic and diastolic level differences between finger and brachial arterial pressures changed from -4 (1.5) and -8 (11) to +8 (14) and +8 (12) mmHg, respectively, after inverse modelling, with pulse pressures correct on average. The pressure decrement model reduced both the mean and the standard deviation of systolic and diastolic level differences to 0 (13) and 0 (8) mmHg. Diastolic differences were thus reduced most. Conclusion: Brachial to finger pulse wave distortion due to wave reflection in arteries is almost identical in all subjects and can be modelled by a single resonance. The pressure decrement due to flow in arteries is greatest for high pulse pressures superimposed on low means.

Keywords: Finapres; Brachial-to-finger modelling; Waveform distortion; Blood pressure; Human

1. Introduction

The brachial artery is often the clinical site for intra-arterial and for non-invasive Riva-Rocci cuff blood pressure measurements. For several years it has been possible for continuous finger arterial pressure waveforms to be recorded non-invasively. This has been used to follow changes in blood pressure in response to various stressors [6-8,17,24,26] and during the course of the 24-h day [9] in persons in whom insertion of an arterial cannula is not accepted as ethical. However, for physiologic reasons (pulse wave reflection and pressure gradient due to flow), pressure pulsations in the finger differ in shape from and are depressed in level compared to those recorded in the brachial artery.

Therefore, since non-invasive arterial blood pressure has become feasible [18,25], publications have appeared comparing finger arterial systolic, diastolic and mean pressure levels to levels recorded intra-arterially in the brachial or radial artery. Earlier studies concentrated on level comparisons sampled at various periodic intervals [13,22,24], while later studies compared beat-to-beat values [2,7,8,20]. As a next step we decided to study the differences in the waveforms recorded in the brachial artery and at the finger, with the aim to find a distortion model and then to inverse-model the finger to the brachial pressure pulsations.
A previous study described pressure pulse propagation in the arterial system from the aorta to the radial artery and stated that application of a generalized filter could remove waveform distortion [10]. Since finger diastolic and mean pressure are clearly below intra-arterial [7], due to a pressure gradient in the arteries of the arm and hand and decrements that appeared to depend on the activity level of the subject [9] we suspected that waveform distortion to the finger might also depend on pressure gradient, mean pressure, heart rate and subject age.

2. Methods

2.1. Subjects

Pressure recordings were available from 4 previous studies [2,6,14,20], chosen for our purpose because they cover a wide range of ages, blood pressures and conditions of the circulation. Study [14] provided pressures of 18 (suspectedly borderline) hypertensive patients aged 25–65 years. Eleven of the patients were on anti-hypertensive medication. Study [6] provided the pressures of 7 healthy resting volunteer subjects, aged 22–40 years. Studies [2] and [20] provided pressures of 28 subjects aged 52–83 years, 15 of whom were healthy elderly subjects, 13 suffered from therapy-resistant hypertension and 7 additionally from arteriosclerotic vascular disease. The original studies were approved by the respective ethical committees. Informed consent was obtained from all volunteer subjects and all patients prior to entering the protocol.

2.2. Pressure measurements

The non-dominant arm was used for cannulation. After local anaesthesia with a 1% lidocaine solution, a Travenol Quick Cath, N1113, 20 gauge, 11 cm long Teflon cannula was inserted into the brachial artery. The cannula was connected through a short section of tubing to a fluid-filled pressure transducer installed with a continuous flush system and strapped to the mid-upper arm at heart level. The resonance frequency of these systems was checked with a well-maintained mercury manometer. Pressure channel sensitivity (static accuracy) was checked against a Finapres. Differences were always less than 2 mmHg over the 0–300 mmHg range.

TNO Finapres Model 5 devices and finger cuffs were used for the finger pressure recordings. A properly sized cuff (see Finapres manual) was wrapped around the middle or index finger mid-phalanx of the ipsilateral hand, and supported at heart level. Using the ipsilateral hand may cause errors if the more proximal cannula partly blocks the radial artery [12]. However, in the studies whose results we used the much larger brachial artery was cannulated. No signs of damped brachial waves [19], or signs of blocking such as slow rising finger pressure pulses, could be detected.

2.3. Signal processing

The brachial and finger pressure signals were fed through identical Krohn-Hite Model 3750 40 dB/decade low pass filters at 30 Hz −3 dB cut-off. A computer system digitized the simultaneous pressure signals with a resolution of 0.1 mmHg at a rate of 200 Hz. Next, the records were digitally filtered with a Hamming (−1, −5, −5, +20, +70, +98, +70, +20, −5, −5, −1) low pass filter, which is −3 dB at 35 Hz. Every other sample was then deleted to have signals sampled at 100 Hz.

Epochs of approximately 10 s duration were selected from the recordings having an integer number of beats and an almost flat baseline. Selected epochs began and ended just before the beginning of a brachial upstroke, lasted on average 9 (s.d. 1.5) s, and contained n = 10 (s.d. 2), range 4–16 beats. Any slight remaining pressure differences between beginning and end of the records were linearly subtracted. This gave signal epochs ready for discrete Fourier analysis without further windowing.

2.4. Model identification

After subtraction of the mean pressure to obtain just the pulses Fourier analysis was used to establish each individual's and the geometric average 'forward' (brachial to finger artery) complex frequency transfer function. We used MATLAB (The Math Works, Inc., South Natick, MA, USA) for this purpose. The amplitude transfer functions were suggestive of the existence of a resonance near 7 or 8 Hz and subsequent return to unity transfer at higher frequencies.

A model transfer function having such response is:

$$H(f) - K \frac{(1 + df/f_0)^2}{1 + 2if/f_0 - (f/f_0)^2}$$

(1)

with i the imaginary unit. This model is equivalent to a gain (K), a second-order aperiodic high emphasis section at frequency $f_0$, followed by a second-order underdamped low pass section at resonance frequency $f_0$, with damping $D < 1$. The transfer function was forced to unity transfer at high frequencies by linking $f_0$ and $f_i$ by the equation:

$$f_0 = f_i \sqrt{K}$$

(2)
This avoided overtight fitting of the model to the high-frequency section of the response where coherence between brachial and finger pressure spectral components is low. Using MATLAB, the complex model was least-squares-fitted to each subject's complex transfer function. The fit program was free to change the relative delay of the two epochs per subject to obtain a best fit. Thus 3 model parameters were estimated: gain $K$, resonance frequency $f_r$, and damping $D$.

The model parameters obtained for each subject were entered into a table together with mean finger pressure, heart rate and age, and multiple regressions computed to verify interdependencies. A general model for the group was computed by taking the average of each model parameter. The result is the general forward model.

2.5. Inverse modelling

An inverse model amplifies the signal at frequencies where the forward model attenuates and vice versa. Each individual's own inverse model and the general inverse model for the group were applied to the finger pulses. The original finger pulse and the inverse-modelled finger pulse were compared to the corresponding brachial pulse and the standard deviation of the differences (SDD) obtained for the optimal delay. This provided a measure for the precision of brachial pulse approximation.

2.6. Level correction

Equally, the full finger pressure (without mean pressure subtracted) was inverse-modelled with the general inverse model to approach full brachial artery pressure. The remaining differences in systolic, diastolic and mean levels between finger and brachial, and between the inverse modelled finger and brachial pressures were computed. This provided a measure of the accuracy of the pressure level recovery by the inverse model. For the entire group we then checked if the level differences depended on measures obtainable non-invasively, such as finger pressure levels, heart rate, or age, by computing multiple regressions. When significant, we corrected the inverse modelled finger pressure levels by the value of the regression equation and recomputed level differences mean and standard deviation.

2.7. Random subgroups

Using the entire group for model analysis and inverse modelling provides for the best signal to noise ratio but does not allow the verification of selection effects. Therefore, 5 different random selections of 23–29 subjects and their complementary groups of 30–24 subjects were constructed. General distortion models for each of 10 subgroups were computed and compared to the general model for the entire group. Furthermore, the level differences multiple regressions for the 10 subgroups were computed and applied as a correction to their complementary group and the results compared.

3. Results

Some typical blood pressure records are shown in Fig. 1. Finger diastolic and mean levels are generally below intrabrachial; systolic levels tend to be at or above intrabrachial pressures. At the finger, young subjects (top traces) tend to show a strong initial systolic peak and a deeper falling dicrotic notch. Oscillations are more pronounced in the finger than in the brachial pressure. Older subjects (middle traces) tend to show two systolic peaks of approximately equal height. Baseline changes in intrabrachial pressure are tracked well by Finapres (bottom traces).

3.1. Waveform distortion model

Group average results of the spectral analyses are shown in Fig. 2. The amplitude spectrum for brachial pressure (upper panel) is high near the fundamental frequency of approximately 1 Hz. Towards higher frequencies the amplitude decreases quickly to below 0.1 mmHg at 10 Hz, or by a factor of 50 per decade of frequency.

Coherence between the brachial and finger pulsation spectra (middle panel) is near 1 up to about 10 Hz. Above 10 Hz coherence drops gradually to remain above 0.5–50 Hz.

The geometric average forward amplitude transfer as a
Fig. 2. The upper panel shows the group average brachial pressure amplitude spectra and standard error bars (mmHg). The middle panel shows the average coherence between the brachial and finger pressure spectra and standard error bars. The bottom panel presents the measured geometric-average amplitude transfer function (circles) and the average model transfer function (drawn curve). The dots in vertical arrangement indicate the position of the individual model transfer functions. Note that some of the scales are logarithmic. Measured data start at a frequency of 0.5 Hz which was present in all subjects. Model data start at 0.1 Hz.

function of frequency (bottom panel) is shown together with the average forward model transfer function. Both transfer functions follow the same trace over the entire frequency range. Low frequencies are attenuated in the finger. Frequencies near the resonance peak at 7 Hz are amplified. The transfer function crosses unity level near 2 Hz. At high frequencies the model transfer function returns to unity slightly quicker than the computed forward transfer function since coherence is low at these frequencies and a tight fit not attempted. Individual transfer functions differ from the average, as can be observed in the figure, but not wildly so, as can be judged from the model statistics in Table 1. The model gain $K$ has 10% standard deviation, the resonance frequency $f_r$ has 18%, and the damping coefficient $D$ has 45% standard deviation.

Various random selections of subjects from the pool of 53 and the remaining complementary groups each had model parameter averages nearly equal to those in Table 1 and no random selection showed significant differences ($P > 0.05$). No evidence of selection effects, therefore, is apparent.

### 3.2. Dependencies of model coefficients

Of the subject data, pulse pressure regressed significantly on age (correlation coefficient $r = 0.62; P < 0.001$). Of the model coefficients, $K$ did not regress on any other parameter. Resonance frequency, $f_r$, and $D$ regressed significantly on mean finger pulse pressure. Taking the regression into account the standard deviation of $f_r$ decreased slightly from 18 to 16%. The standard deviation of $D$ did not decrease appreciably after regression correction.

### 3.3. Inverse distortion model

The degree of pulse wave distortion removal can be judged from Fig. 3. The standard deviation of the differences (SDD) is reduced most by the individual inverse model (Table 1). Application of the general inverse model, since it is only correct on average, delivers less exact results. Still, most of the waveform distortion is removed by the general inverse model. Histograms of the SDD are shown in Fig. 4.

The general inverse model when applied to the full data of individual patients.
Fig. 4. Histograms of the standard deviation of the differences (SDD) with the brachial pulsations of, from above, the original finger pressure, and the inverse-modelled finger pressure for the individual and the group average models in the 53 subjects of the study.

finger pressure waveform does the same conversion of the pulses, but in addition mean pressure is amplified by the model's inverse $K$-factor, which moves the pressure curve upwards. The systolic, diastolic, mean and pulse pressures in the brachial artery, at the finger, and at the finger after inverse modelling are shown in the first 3 panels of Table 2. Since mean pressure in the finger, on average, is 90% of intrabrachial, but the inverse $K$-factor amplifies mean pressure by 1.19, the inverse-modelled mean pressure is higher than brachial, on average by 7%.

Table 2
Pressure levels and level differences (mmHg)

<table>
<thead>
<tr>
<th>Pressure Level</th>
<th>Mean</th>
<th>s.d.</th>
<th>Range</th>
<th>Mean</th>
<th>s.d.</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brachial</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>169</td>
<td>33</td>
<td>95–245</td>
<td>169</td>
<td>33</td>
<td>95–245</td>
</tr>
<tr>
<td>Diastolic</td>
<td>89</td>
<td>17</td>
<td>46–127</td>
<td>89</td>
<td>17</td>
<td>46–127</td>
</tr>
<tr>
<td>Mean</td>
<td>120</td>
<td>22</td>
<td>62–174</td>
<td>120</td>
<td>22</td>
<td>62–174</td>
</tr>
<tr>
<td>Pulse</td>
<td>80</td>
<td>23</td>
<td>41–145</td>
<td>80</td>
<td>23</td>
<td>41–145</td>
</tr>
<tr>
<td>Original finger</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
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<td>30</td>
<td>89–241</td>
<td>165</td>
<td>30</td>
<td>89–241</td>
</tr>
<tr>
<td>Diastolic</td>
<td>81</td>
<td>17</td>
<td>40–128</td>
<td>81</td>
<td>17</td>
<td>40–128</td>
</tr>
<tr>
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<td>53–104</td>
<td>107</td>
<td>21</td>
<td>53–104</td>
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<tr>
<td>Pulse</td>
<td>84</td>
<td>24</td>
<td>49–178</td>
<td>84</td>
<td>24</td>
<td>49–178</td>
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<tr>
<td>Inverse-modelled</td>
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<td></td>
<td></td>
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<tr>
<td>Systolic</td>
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<td>34</td>
<td>93–252</td>
<td>177</td>
<td>34</td>
<td>93–252</td>
</tr>
<tr>
<td>Diastolic</td>
<td>97</td>
<td>21</td>
<td>48–155</td>
<td>97</td>
<td>21</td>
<td>48–155</td>
</tr>
<tr>
<td>Mean</td>
<td>128</td>
<td>25</td>
<td>63–194</td>
<td>128</td>
<td>25</td>
<td>63–194</td>
</tr>
<tr>
<td>Pulse</td>
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<td>25</td>
<td>44–166</td>
<td>80</td>
<td>25</td>
<td>44–166</td>
</tr>
<tr>
<td>&amp; Level corrected</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>169</td>
<td>33</td>
<td>96–261</td>
<td>169</td>
<td>33</td>
<td>96–261</td>
</tr>
<tr>
<td>Diastolic</td>
<td>89</td>
<td>14</td>
<td>52–128</td>
<td>89</td>
<td>14</td>
<td>52–128</td>
</tr>
<tr>
<td>Mean</td>
<td>119</td>
<td>21</td>
<td>66–168</td>
<td>119</td>
<td>21</td>
<td>66–168</td>
</tr>
<tr>
<td>Pulse</td>
<td>80</td>
<td>25</td>
<td>44–166</td>
<td>80</td>
<td>25</td>
<td>44–166</td>
</tr>
</tbody>
</table>

3.4. Level correction

After application of the general inverse model pulse pressures were correct on average. The remaining differences of diastolic pressure with brachial ($\Delta p_d$) (Table 2, third panel: Inv–Bra) regressed significantly on inverse-modelled finger systolic ($p_s$) and diastolic ($p_d$) pressure ($r = 0.73$, $P < 0.001$), and slightly less well on mean and pulse pressure. The differences in mean pressure also

Fig. 5. Demonstration of the process of inverse modelling and level correction on a waveform of a 53-year-old subject. The original finger pressure (top panel) is distorted with respect to brachial (thin curve), and lower in level. After general inverse modelling (middle panel) the waveforms are nearly identical, but the level is still different. Level correction (bottom panel) shifts the waveform down by 7 mmHg in this case, after which it is nearly correct. The delay between waveforms was not modelled and, thus, not corrected, explaining the unchanged delays in the finger pressure derived waveforms.

Fig. 6. Systolic ($\Delta S$) and diastolic ($\Delta D$) level differences between brachial and finger (left panels) and between brachial and inverse modelled and corrected (right panels) finger pressures. The large squares indicate 95% confidence intervals, the centered solid squares the group average values.
regressed on systolic and diastolic pressure \( (r = 0.66, \ P < 0.001) \) although less closely. The differences in systolic pressure and pulse pressure did not correlate well. A level correction was applied to the inverse-modelled finger pressures using the regression model for the diastolic differences on inverse-modelled systolic and diastolic pressures:

\[
\Delta p_D = -13.3 - 0.194 p_s + 0.574 p_D
\]  

(3)

Subtracting \( \Delta p_D \) for each subject, from the systolic, diastolic and mean levels reduced both the mean and the standard deviation of the differences (Table 2, bottom panel).

The two-step process of inverse modelling and level correction is demonstrated in Fig. 5. Bland-Altman plots of the differences in systolic and diastolic pressure before and after correction are presented in Fig. 6. Improvement is greatest for diastolic pressure.

The 10 random selections from the 53 subjects on average provided very similar level correction effects (systolic 0.2 (s.d. 2), diastolic 0.4 (2), mean –0.2 (2) mmHg) and reductions in standard deviation (systolic to 13, diastolic to 8, mean to 9 mmHg) on their complementary selections, suggesting the absence of selection effects.

4. Discussion

We found models for the distortion and level decrement of the pulse wave between the brachial artery and the finger. Similar studies [10,16,23] have appeared before, but the present one is the first on brachial to finger pressure and the first also to consider level depression. A two-step approach was used. First we modelled brachial to finger pulse waveform distortion by a frequency-dependent equation (Eqs. (1) and (2)), then we modelled the remaining level differences between the two sites with a multiple regression equation (Eq. (3)). Correction was done in the same two-step procedure. Major reductions in distortion and level decrement were obtained this way.

Finger pressure waveforms and levels are less familiar to clinicians than brachial or radial pressures. Radial artery pressures are used principally in surgery and intensive care situations. Here, monitoring changes in blood pressure levels is often more important than absolute accuracy. Brachial artery pressures are used frequently for diagnosis in the practice of internal medicine and cardiology. The accuracy of systolic and diastolic levels is of primary importance. To correct the differences of finger with intra-brachial pressures, both the pulse pressure and the mean pressure levels must be correct, which requires application of both our modelled effects.

Some authors mention the possibility of obtaining an estimate of arterial compliance from inspection of the aortic pressure wave, in particular the late systolic augmentation of the pulsation [16]. Some authors stress that such changes are age-related [11]. The present inverse model (from finger to brachial artery) does not allow for such inspection of waveforms with clarity but would require extension of the inverse model to aortic pressures.

4.1. Waveform distortion

Finger pressure is quite similar to radial artery pulsations [21,23], whereas brachial pulsations appear as less resonant (Fig. 1). Using Fourier transformation, we determined individual linear forward transfer functions describing the waveform distortion that occurs when pulses travel in the arterial system between brachial and finger artery. Regardless of the individual differences in pulse shape, one major resonance near 7 Hz stood out (Fig. 2). Since the model is linear, and since the finger pulses after individual inverse modelling approach the brachial pulse so closely (Table 1 and Figs. 3 and 4), this shows that pressure pulse propagation in the arm arterial system can be understood as a linear process. This confirms in humans earlier findings in animals [15], even for pulse pressures as high as 145 mmHg in the brachial artery (Table 2).

The resonance frequency depends most on the length of the arterial section considered. Karamanoglu et al. [10] measured between aorta and radial artery, and detected a resonance near 4 Hz. We measured the shorter section between brachial artery and finger, and found a resonance near 7 Hz. Triedman and Saul [23] measured the even shorter section between radial artery and finger in children, and found a resonance near 10 Hz. Considering the arterial section as an organ pipe closed at the distal end (with reflection coefficient almost equal to 1 [15]), the shortest pipe indeed produces the highest tone.

Surprisingly, even the general inverse model provided a good approximation of the brachial from the finger pulse, although individual resonance frequency and damping showed substantial scatter and regressed significantly on mean blood pressure. An explanation is that at low frequencies the individual forward transfer functions are similar. It is at the lower frequencies that the harmonics in the finger pressure spectrum are strongest (Fig. 2) and thus have most influence on the shape of the pulse. What happens at higher frequencies has a lesser effect.

Karamanoglu et al. [10] stated recently that a generalized transfer function can be used to estimate central from peripheral pressure under different conditions in adult humans. It is not obvious, a priori, that Karamanoglu’s conclusions remain valid for an acral site such as the finger since the small size of the peripheral arteries leading to the finger cause a pressure gradient due to flow, which dampens the pulse. We did find a significant pressure dependency of the model resonance frequency \( (f_r) \) and damping \( (D) \) coefficients. However, application of general inverse models with the model coefficients individually adapted applying the regression of each coefficient on mean pressure led to only insignificant individual and no changes on average in inverse modelling accuracy. This
supports Karmanoglu's conclusion for transmission of pulses as far peripherally as the finger arteries.

4.2. Pressure levels

Finger pressures are not only distorted, but diastolic and mean pressures are depressed in level compared to brachial artery pressure. A notable pressure gradient in the arterial system exists caused by flow in the smaller resistive arteries. On applying the inverse model such mean level depressions on average are overcompensated, indicating that mean pressure is less affected by the resistance to flow than is pulse pressure. The extra damping for pulsations at low frequencies could well be caused by the compliance of the small branches of the arterial system.

Thus, application of the general inverse model—although demonstrated earlier to improve tracking of blood pressure changes [5]—did not improve the accuracy of the estimation of systolic, diastolic and mean levels of brachial pressure by the finger even though pulse pressures were now correct on average (Table 2, bottom panels). The underestimation of brachial diastolic and mean levels by finger pressure turned into an overestimation of 8 mmHg for all levels. This overestimation could have been removed simply by subtracting 8 mmHg from all pressure levels but that would not have reduced the standard deviation of the differences. Correction by application of a multiple regression model, however, did reduce standard deviation. Diastolic pressure accuracy now meets AAMI criteria [1], but systolic does not. We applied the diastolic level correction since its regression equation showed the highest correlation and since diastolic levels showed the largest relative differences (8% for systolic versus 14% for diastolic, computed from Table 2) and needed correction most. After level correction relative differences distributed more equally at 8 and 9%, respectively.

This level correction shifts the waveform in an upwards direction when large pulsations are superimposed on a low diastolic pressure. Such large pulsations tend to indicate a large stroke volume and cardiac output, and a low diastolic pressure in addition indicates a low peripheral resistance and a high peripheral flow which would cause a high peripheral pressure decrement. The opposite situation of a small pulse superimposed on a high diastolic pressure we have often observed to occur in cold fingers under a supposedly high sympathetic tone and a generally low peripheral flow state [24]. Since a high sortie flow does not necessarily indicate a high peripheral flow in the arm and hand, the model is imperfect and the level shift correction helps but provides no perfect remedy.

We tested our general inverse model under experimental conditions of vasoconstriction and exercise to exertion that extrapolated to higher and lower heart rates and to higher blood pressures [5]. The inverse model not only produced more central blood pressure waveforms from non-invasive finger pressures, but changes in systolic blood pressure were followed with greater accuracy. To track systolic pressures in exercise stress testing and for the computation of baroreflex sensitivity, application of this model appeared essential [5].

Having pulse pressures correct on average after inverse modelling, it should suffice to calibrate just one of its levels (systolic, diastolic or mean) to the corresponding brachial level to have the entire waveform corrected. This was tried in another study [3]. Best results, clearly within AAMI criteria [1] for all levels, were obtained by adjusting inverse-modelled finger systolic pressure to a return-to-flow systolic level detected with a Finapres mounted distal to the upper arm cuff. But extra equipment is needed for this important correction.

5. Conclusion

A simple, single resonance model describes waveform distortion in the arm arterial system between the brachial artery and the finger. A general inverse model converts non-invasively recorded finger pressure waveforms to brachial pulsations with precision independent of resting pressure, heart rate or age. A regression-based level correction procedure models the dependence of the pressure gradient on flow to further reduce group mean differences and standard deviations without taking an extra measurement.

References


