Population-based studies that assess risk related to high blood pressure (BP) demonstrate strong associations between brachial BP (measured by cuff at the upper-arm) and cardiovascular morbidity and mortality. While mean and diastolic BP (DBP) are relatively constant throughout the arterial tree, it is well recognized that systolic BP (SBP) is higher in the more muscular peripheral arteries (e.g., brachial artery) than in the elastic central arteries (e.g., aortic, carotid). The magnitude of this pressure amplification varies between individuals and can result in markedly different aortic (central) SBP between individuals despite sharing similar peripheral (brachial) SBP. Importantly, central BP may be a stronger predictor of cardiovascular risk and mortality than brachial BP. Furthermore, antihypertensive agents may have a different effect on central BP when compared with brachial BP, and consideration of central BP (in addition to brachial BP) when assessing BP control may improve treatment decisions. Thus, measurement of central BP could aid in the diagnosis, risk stratification, and management of patients with hypertension.

Invasive assessment of central BP is impractical for widespread use, and this has led to development of various noninvasive methods. The reference standard is generally accepted to be radial applanation tonometry, and several of these devices are available on the market for estimating central BP indices.

To mitigate these limitations, a number of upper-arm cuff-based methods to estimate central BP have been developed. These include XCEL, Atcor Medical, Mobil-o-graph, I.E.M. GmbH, Vicorder, and Skidmore Medical, to name a few that have potential for incorporation into routine clinical practice. One of the earliest cuff-based devices (Pulsecor, which uses a suprasystolic algorithm) shows good agreement with arterial tonometry–derived central BP and has undergone spread use, and this has led to development of various noninvasive methods. The reference standard is generally accepted to be radial applanation tonometry, and several of these devices are available on the market for estimating central BP indices.

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evaluation by comparison with invasive aortic BP measures under resting conditions. However, these studies were in relatively small populations and did not examine accuracy after induction of a hemodynamic perturbation, which is an important consideration when assessing device performance. Here, we evaluated the accuracy of the Pulsecor device in estimating central BP (CBP) by comparison with invasive measures of aortic BP (CBP) under basal conditions and after an acute perturbation induced by intravenous nitroglycerin (GTN).

METHODS

Study participants

The study population was drawn from patients scheduled for routine elective coronary angiography at the Royal Hobart Hospital, Hobart, Australia. A total of 52 participants were recruited and included in the study regardless of coronary artery disease (CAD) severity. Of those participants, 1 became unstable after commencement of angiography, and it was deemed inappropriate to continue with research measures; 2 were in atrial fibrillation that precluded accurate BP measurements; and 2 were found to have severe aortic stenosis and were subsequently excluded. This left 47 participants and a total of 94 measures recorded (47 at baseline and 47 following administration of GTN). The University of Tasmania Human Research Ethics Committee approved the study, and all participants signed written informed consent.

Study protocol

Participants were prepared for angiography following standard protocols, and the Pulsecor (R7.0; Pulsecor, Auckland, New Zealand) BP cuff was applied to the left upper arm (over the brachial artery) to measure brachial BP and calculate CBP. Simultaneously with measurement of CBP, CBP was obtained from a fluid-filled catheter positioned in the ascending aorta. Measurements were completed under baseline (resting) conditions and repeated 1 minute following intravenous GTN (100–200 μg). Analgesia and/or anxiolytic medication was used prior to recording measures according to the clinical indication. All measures were completed prior to imaging the coronary arteries as per standard angiogram procedure. Anthropometric and medical history data were retrieved from patient records. Participants were not instructed to abstain from taking antihypertensive medication prior to research measures.

Noninvasive BP measurement

Brachial BP and CBP were recorded using the noninvasive vascular monitor (Pulsecor R7.0), which has been approved by the US Food and Drug Administration. The device works by initially recording brachial SBP and DBP via oscillometry (Welch Allyn, Skaneateles Falls, NY). The cuff is then reinflated to approximately 30 mm Hg greater than the recorded SBP (suprasystolic pressure) and holds this inflation for 10 seconds. During the period of held inflation, suprasystolic BP waves are recorded with the cuff, and central BP is estimated via a time-domain approach that assesses the relationship between the total oscillatory pressure in the aorta and the total oscillatory pressure under the occluded brachial artery. Each participant was asked to remain still and quiet during inflation, measurement, and deflation periods. Results were stored in the Pulsecor device software (VasomonR, Auckland, New Zealand). The quality of each recording was assessed visually and using the signal-to-noise ratio generated by the device. A signal-to-noise ratio of ≥10 was considered satisfactory for inclusion in analysis. CBP was measured at baseline and 1 minute following a hemodynamic shift induced by GTN.

Invasive BP measurement

CBP was directly measured via fluid-filled catheter, with the type of catheter varying depending on the arterial access site (either via the radial or femoral artery), but included those routinely used in coronary angiography, for example, 6F Judkins Left (Cordis, NJ), multipurpose (Cordis), or TIG (Terumo, NJ). For all measures, the catheter was placed in the proximal ascending aorta approximately 1 cm above the aortic valve, which was assessed visually via fluoroscopy. Prior to each measurement, catheters were flushed and the BP trace visually inspected for quality. During all recordings, transducers were maintained at heart level. The CBP signal was acquired at a sample rate of 1,000 Hz and recorded using PC acquisition software (LabChart 7; AD Instruments, Bella Vista, Australia) via analogue-to-digital signal converter (PowerLab ML870 8/30; AD Instruments). CBP measurement was continuously maintained over the period of research measures (approximately 5 minutes) with electronic marker points inserted in the digital recording to denote the CBP measurement times (specifically the period of held cuff inflation and brachial artery occlusion at suprasystolic BP). The CBP signal was recorded in volts and converted to mm Hg offline using a 2-point calibration method. To obtain the calibration values for CBP, the catheter laboratory system (Mennen, Trevose, PA) was zeroed (to obtain the first calibration point; a 0 mm Hg value); then, on the first CBP waveform following the zero line, the peak BP value (corresponding to CBP) was quantified using the system’s built-in BP value detection (to obtain the second calibration point).

Comparisons and calibration methods

To evaluate the accuracy of the Pulsecor R7.0, we performed a direct comparison of the mean values of CBP and DBP with CBP and DBP (both at baseline and post-GTN). To determine CBP values, we averaged CBP values, and we captured over the corresponding period in which CBP values were determined using the Pulsecor device (this was determined over the 10- to 15-second period of brachial artery occlusion at suprasystolic pressure). This analysis technique conforms
had a history of hypertension, hypercholesterolemia, and diabetes, and one quarter of the study population had no evidence of significant CAD. Once the measurements of insufficient quality were excluded (assessed visually or signal-to-noise ratio <10), the final number of measurements included in analysis was 84 (40 at baseline, 44 following administration of GTN).

Baseline comparison between CBP_{invasive} and CBP_{estimated}

A comparison of CBP_{invasive} and CBP_{estimated} values under baseline conditions is outlined in Table 2. CBP_{estimated} SBP was lower than CBP_{invasive} SBP (Figure 1). There was a wide spread of SBP values in the Bland–Altman analysis (Figure 2); however, no evidence of systematic bias (Pearson r = −0.04; P = 0.80). The CBP_{estimated} method showed a tendency to overestimate the DBP compared with the CBP_{invasive} method of assessment (Figure 3), and there was also evidence of a trend toward systematic bias found in the Bland–Altman analysis (Pearson r = −0.30; P = 0.06; Figure 4). There were also no significant differences in the mean difference of CBP_{estimated} and CBP_{invasive} SBP and DBP between those with no CAD or single-vessel disease (n = 21) and those with double-vessel or triple-vessel disease (n = 20) at baseline (P = 0.568 and P = 0.508, respectively).

Post-GTN comparison between CBP_{invasive} and CBP_{estimated}

Administration of GTN significantly increased heart rate from baseline (from 75 ± 11 to 79 ± 11 bpm; P < 0.01). CBP_{estimated} and CBP_{invasive} BP measures following administration of GTN are outlined in Table 2. CBP_{estimated} SBP tended to be lower than CBP_{invasive} SBP (Figure 1) but with wide variability and no evidence of systematic bias (Pearson r = −0.04; P = 0.79; Figure 2). CBP_{estimated} DBP post-GTN overestimated CBP_{invasive} (Figure 3), with wide standard deviation, large mean difference, and significant systematic bias (Pearson r = −0.55; P < 0.001; Figure 4). The change in BP from baseline to post-GTN is outlined in Table 2. The change in CBP_{estimated} SBP closely agreed with that of CBP_{invasive} SBP again with greater error in agreement evident when the change in DBP was compared. There were also no significant differences in the mean difference of CBP_{estimated} and CBP_{invasive} SBP and DBP between those with no CAD or single-vessel disease (n = 23) and those with double-vessel or triple-vessel disease (n = 21) post-GTN (P = 0.229 and P = 0.230, respectively).

Comparison of SBP measures following recalibration of CBP_{estimated} with CBP_{invasive} MAP and diastolic BP

CBP_{estimated} SBP was recalibrated using the invasive measurements of MAP and DBP; the results are presented in Table 3. For all measurements (baseline and post-GTN), the recalibrated CBP_{estimated} SBP was lower than the CBP_{invasive} SBP. There was strong agreement between methods at baseline, but some variation in agreement post-GTN remained (see Figures 5 and 6).
Table 2. Comparison between central blood pressure (CBP)\textsubscript{invasive} and CBP\textsubscript{estimated} systolic blood pressure and diastolic blood pressure at baseline and post-glyceryl trinitrate

<table>
<thead>
<tr>
<th>Variable, correlation, and significance</th>
<th>Baseline (n = 40)</th>
<th>Post-GTN (n = 44)</th>
<th>Post GTN–baseline (n = 38)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CBP\textsubscript{invasive} SBP, mm Hg</td>
<td>131±20</td>
<td>113±17</td>
<td>−17±12</td>
</tr>
<tr>
<td>CBP\textsubscript{estimated} SBP, mm Hg</td>
<td>124±19</td>
<td>108±16</td>
<td>−16±13</td>
</tr>
<tr>
<td>CBP\textsubscript{estimated} SBP – CBP\textsubscript{invasive} SBP, mm Hg</td>
<td>−7±9</td>
<td>−6±9</td>
<td>−2±7</td>
</tr>
<tr>
<td>ICC</td>
<td>0.86</td>
<td>0.90</td>
<td>0.82</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CBP\textsubscript{invasive} DBP, mm Hg</td>
<td>67±11</td>
<td>63±13</td>
<td>−3±5</td>
</tr>
<tr>
<td>CBP\textsubscript{estimated} DBP, mm Hg</td>
<td>77±10</td>
<td>68±9</td>
<td>−8±5</td>
</tr>
<tr>
<td>CBP\textsubscript{estimated} DBP – CBP\textsubscript{invasive} DBP, mm Hg</td>
<td>10±6</td>
<td>5±7</td>
<td>−5±7</td>
</tr>
<tr>
<td>ICC</td>
<td>0.84</td>
<td>0.84</td>
<td>0.12</td>
</tr>
<tr>
<td>P value</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>0.135</td>
</tr>
</tbody>
</table>

Data are mean ± standard deviation. CBP\textsubscript{estimated} is calibrated using noninvasive brachial oscillometric BP (as is the inbuilt algorithm).

Abbreviations: CBP, central blood pressure; DBP, diastolic blood pressure; GTN, glyceryl trinitrate; ICC, intraclass correlation coefficients (2-way mixed with absolute agreement); SBP, systolic blood pressure.

**DISCUSSION**

In this study, we sought to evaluate the performance of an upper-arm oscillometric cuff method in estimating central BP. Our results indicate large variability between CBP\textsubscript{invasive} and CBP\textsubscript{estimated} SBP and DBP values when CBP\textsubscript{estimated} is calibrated with oscillometric cuff brachial BP. Agreement was improved when CBP\textsubscript{estimated} was recalibrated with invasive MAP and DBP, thereby providing evidence to support the accuracy of the algorithm to derive central SBP from a brachial suprasystolic waveform under baseline (normal resting state) conditions. Further refinement of the cuff-based technique to estimate central BP appears necessary because of error attributable to noninvasive brachial BP calibration.

Risk related to BP is traditionally determined via measurement of brachial BP. However, the prognostic and clinical importance of central BP beyond conventional brachial BP has recently been demonstrated.\textsuperscript{3,4,6–8,22} This has led to international consensus regarding the potential clinical importance of central BP,\textsuperscript{10} and threshold values denoting increased risk related to raised central BP have recently been determined.\textsuperscript{23} Arterial tonometry of the carotid artery or synthesis of the central BP wave via a radial-to-aortic transfer function (Sphygmocor) is considered the noninvasive gold standard measure of central BP.\textsuperscript{10} Nonetheless, assessment of central BP via this method has remained confined to research settings because of the requirement for specialized equipment, technical expertise, and extra time for accurate data acquisition.
The Pulsecor device is a new generation of monitor designed to circumvent the need for special training and equipment by using an automated oscillometric cuff to synthesize a central BP waveform from the occluded brachial artery at suprasystolic pressure. In the population-based Southall and Brent Revisited Study, 1,107 consecutive Pulsecor R6.5 measurements of central BP were compared with those derived from radial arterial tonometry (Sphygmocor) and relatively close concordance between methods was demonstrated (mean difference central SBP of 3 ± 6 mm Hg; intraclass correlation coefficient = 0.91). Our group also found Pulsecor R6.5–derived central SBP to be comparable with Sphygmocor-derived central SBP in different clinically applicable postures (i.e., supine, seated, and standing), 19 and the Pulsecor R6.5 device has good reproducibility over short 18, 19 and longer-term follow-up. 18

In the original study in which the performance of the Pulsecor R6.5 device was tested, 16 participants had simultaneous measures of Pulsecor central BP and invasive central BP recorded. 16 Although mean differences between invasive and noninvasive estimates of central SBP (−1.0 ± 14.7 mm Hg) and central DBP (3.9 ± 8.6 mm Hg) were small, there was wide variability (large SDs) and measurements were only compared under baseline conditions. 16 Pulsecor R6.5 performance was further evaluated in another study in which mean values of central BP were again highly comparable to catheter measurements of aortic (central) BP. 20 However, systematic bias (and wide variation) was evident, whereby noninvasive central SBP measures showed trends.

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Abbreviation: SD, standard deviation.

no evidence of a linear trend (systematic bias) at baseline or post-GTN. There was a small underestimation of systolic blood pressure (SBP) and DBP, measured at baseline (open circles) and acutely post-administration of glyceryl trinitrate (GTN; closed circles). There was no evidence of a linear trend (systematic bias) at baseline or post-GTN.

Figure 6. Bland–Altman plot of the mean values and differences between central blood pressure (CBP) invasive systolic blood pressure (SBP) and CBP estimated SBP when recalibrated with invasive mean arterial pressure and DBP, measured at baseline (open circles) and acutely post-administration of glyceryl trinitrate (GTN; closed circles). There was no evidence of a linear trend (systematic bias) at baseline or post-GTN. Abbreviation: SD, standard deviation.

to underestimate invasive central SBP with increasing pressure. More recently, a subgroup of participants from the study by Park et al. had Pulsecor R6.5 central SBP and DBP compared with ascending aortic SBP acquired by high-fidelity pressure wires. Limits of agreement met the AAMI standard (recorded mean difference in central SBP of $\pm 5\pm 8$ mm Hg); however, this analysis was limited by small sample size ($n = 6$).

Our study represents the largest study to date to evaluate the accuracy of the Pulsecor device compared with invasive BP measures and expands on previous studies by providing information on Pulsecor R7.0 performance following hemodynamic perturbation. This is an important addition because it highlights potential utility of the device for responding to clinically relevant situations in which arterial BP may acutely change, such as the response to standing from the sitting position. When we used noninvasive upper-arm cuff BP to calibrate CBP invasive, our results were consistent with those of previous studies, in that CBP invasive SBP was underestimated by CBP estimated SBP, and CBP invasive, DBP was overestimated by CBP estimated DBP. Agreement between CBP estimated SBP and CBP invasive SBP and DBP was not improved/changed when measures were performed post-GTN, although the reduction in CBP estimated SBP was similar in magnitude to the CBP invasive SBP reduction from baseline to post-GTN.

Although cuff-based devices may have superior clinical applicability for deriving central BP compared with applanation tonometry (because of their ease of use), caution regarding clinical uptake is still advised due to the potential error caused by noninvasive brachial BP calibration. In our study, agreement between CBP invasive and CBP estimated (baseline and following GTN) fell outside the standard required by the AAMI SP10 protocol (agreement required $\pm 5\pm 8$ mm Hg), when calibrated by the oscillometric brachial SBP and DBP. The Pulsecor device has an inbuilt oscillometric BP module that meets the AAMI SP10 specifications for measurement of brachial BP and achieves a British Hypertension Society A rating for this purpose (absolute difference between standard and test device $\leq 5$ mm Hg on $\geq 60$% of comparisons). However, in our study, when CBP estimated SBP was recalibrated using invasively measured MAP and DBP, the mean difference was substantially improved at baseline. This is consistent with the findings of Lin et al. who showed the algorithm to derive central SBP with invasive calibration to be valid under baseline conditions. Interestingly, invasive recalibration of CBP estimated improved agreement between CBP estimated SBP and CBP invasive SBP under resting conditions but not after GTN infusion. Administration of GTN acutely increased heart rate in our study, and it is possible that the accuracy of generalized transfer functions to derive central SBP may be compromised at higher heart rates. Indeed, Smulyan et al. showed that invasively calibrated, transfer function–derived central SBP underestimated invasive aortic SBP at higher heart rates (e.g., $>75$ bpm) induced by dobutamine infusion. Although one study showed similar underestimation of derived central SBP at higher heart rates induced with exercise, greater discrepancies between transfer function–derived central SBP and invasive aortic SBP do not appear to occur until exercise heart rate exceeds approximately 100 bpm. Since post-GTN heart rate was only $79\pm 11$ bpm in our study, the significant underestimation of central SBP post-GTN could be due to drug-related hemodynamic effects separate from the increase in heart rate; however, this remains to be tested. Nonetheless, our results do imply the difference in agreement between CBP estimated and CBP invasive values is, in large part, due to error attributable to noninvasive brachial BP calibration.

Noninvasive calibration is a previously reported limitation of cuff-based central BP devices, as upper-arm estimates of brachial SBP tend to underestimate true brachial SBP, resulting in underestimation of synthesized central SBP. While some have suggested calibration error can be improved by using noninvasive brachial MAP and DBP to calibrate the BP waveform (rather than SBP and DBP), Pulsecor R6.5–derived central SBP and mean difference (compared with tonometry-derived central SBP) does not appear to be improved via this method. Moreover, errors in central SBP derivation (via transfer function) are known to be equivalent to errors associated with brachial cuff sphygmomanometry. Thus, noninvasive calibration remains the “Achilles-heel” in noninvasive central BP estimation, and improved calibration techniques (or perhaps algorithms that account for calibration errors specific to the individual device) are required to improve the overall accuracy of central BP estimation.

Limitations

Our study was performed in a population of older individuals with indications for coronary angiography. Some participants had CAD and most had a number of comorbidities typical of this patient population. Although this allowed us to determine device performance in a clinically relevant population, our results may not be applicable to younger, healthier individuals or other patient groups.
CONCLUSIONS

This study demonstrated that the Pulsecor algorithm to noninvasively derive central SBP has reasonable accuracy when calibrated with invasive MAP and DBP under baseline "rest" conditions. However, GTN-induced higher heart rates and noninvasive calibration using upper-arm cuff BP produce errors in correct determination of central BP (resulting in underestimation of central SBP and overestimation of central DBP). Further refinement of the algorithm should improve clinical applicability of the device.

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DISCLOSURE

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REFERENCES


