ORIGINAL ARTICLE

# Central Hemodynamics Could Explain the Inverse Association Between Height and Cardiovascular Mortality

Jake C. Reeve,[1] Walter P. Abhayaratna,[2] Justin E. Davies,[3] and James E. Sharman[1]

**BACKGROUND**
Mechanisms underlying the inverse relationship between height and cardiovascular mortality are unknown but could relate to central hemodynamics. We sought to determine the relation of height to central and peripheral hemodynamics, as well as clinical characteristics.

**METHODS**
The study population was comprised of 1,152 randomly selected community-dwelling adults (aged 67.7 ± 12.3 years; 48% men). Brachial blood pressure (BP) was recorded by sphygmomanometry; central BP and aortic pulse wave velocity were estimated by applanation tonometry. Stepwise multiple regression analysis was used to determine associations between height and central and peripheral hemodynamics.

**RESULTS**
Height was not significantly associated with aortic pulse wave velocity in men or women. The relationship with height and brachial systolic BP was borderline in women ($\beta = -0.115$; $P = 0.051$) but not significant in men ($\beta = -0.096$; $P = 0.09$). Conversely, central systolic BP, estimated by transfer function ($\beta = -0.139$ for men [$\beta_M$]; $\beta = -0.172$ for women [$\beta_W$]) or radial second systolic peak ($\beta_M = -0.239$; $\beta_W = -0.281$), augmentation index at 75 bpm ($\beta_M = -0.189$; $\beta_W = -0.224$), and aortic pulse wave timing ($\beta_M = 0.224$; $\beta_W = 0.262$) were independently associated with height in both sexes ($P < 0.003$ for all). Both men and women of greater than median height were less likely to have coronary artery disease ($P < 0.05$), to have systemic hypertension ($P < 0.01$), or to be taking vasoactive medication ($P < 0.001$) compared with participants of less than median height.

**CONCLUSIONS**
Even after correcting for conventional cardiovascular risk factors, taller individuals have more favorable central hemodynamics and reduced evidence of coronary artery disease compared with shorter individuals. These findings may help explain the decreased cardiovascular risk associated with being taller and also have important clinical consequences regarding therapy.

*Keywords:* aortic pulse wave velocity; arterial stiffness; blood pressure; central blood pressure; hypertension.

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The inverse relationship between height and cardiovascular mortality was first reported in 1951 by Gertler and Garn,[1] but pathophysiological mechanisms underlying this association have yet to be elucidated. Numerous studies have confirmed these observations persist after accounting for potentially confounding variables, including socioeconomic status, race, age, smoking history, history of cardiovascular disease or coronary artery disease, serum cholesterol and glucose levels, presence of type 2 diabetes, body mass index, or brachial blood pressure (BP).[2–9] When considering BP as a prognostic marker related to height, it is necessary to appreciate that, because of pressure amplification, major discrepancies may exist between central (aortic) and brachial systolic BP values. Although central systolic BP is usually lower than brachial systolic BP, this magnitude may vary from approximately 2 mm Hg to approximately 30 mm Hg, even between individuals with similar brachial systolic BP, and can occur irrespective of age or cardiovascular disease risk.[10,11] Seminal work by Weber *et al.* showed that central BP was an independent marker of premature coronary artery disease.[12] Further, this group showed that elevated central BP, as determined by augmentation index, was a strong, independent marker of severe cardiovascular events in patients undergoing percutaneous coronary interventions, over and above brachial BP.[13] Moreover, recent meta-analyses reported central BP indices (including augmentation index and aortic stiffness) predicted cardiovascular events and all-cause mortality, independent of brachial BP.[14,15] Accordingly, an inaccurate understanding of the contribution of height to BP and cardiovascular risk could occur if only brachial BP is considered.

Augmentation index can be recorded noninvasively by radial applanation tonometry and is considered a combined marker of left ventricular afterload and systemic arterial stiffness. Several reports have shown height to be inversely associated with augmentation index.[16–19] However, some reports were of small sample size or included participants with established disease (i.e., hypertension, end-stage renal disease, or coronary artery disease), and none

Correspondence: James E. Sharman (james.sharman@menzies.utas.edu.au).

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**[1]Menzies Research Institute Tasmania, University of Tasmania, Hobart, Australia; [2]Clinical Trials Unit, Canberra Hospital, Canberra, Australia; [3]International Centre for Circulatory Health, Imperial College Healthcare NHS Trust, St Mary's Hospital Campus, London, United Kingdom.**

of these studies have been conducted in the community setting using randomly selected populations. All of these factors could potentially distort the true relationship between height and augmentation index. Interestingly, a recent study found that height modified the prognostic value of aortic stiffness (aortic pulse wave velocity) in patients with end-stage renal disease.[20] In this study, taller participants had a reduced risk for cardiovascular mortality compared with shorter subjects.[20] Although increased aortic stiffness is a strong prognostic marker for cardiovascular mortality,[15] the study by Nemeth and colleagues[20] was the first to consider the role of height in the statistical analysis. Taken altogether, the above data suggest that taller individuals have improved central hemodynamics, which could mitigate cardiovascular risk, irrespective of brachial BP. The aim of this study was to determine associations between height and central and peripheral hemodynamics (including aortic stiffness) for the first time in a large population-based sample. We hypothesized that increased height would be associated with lower central BP, augmentation index, and aortic stiffness, independent of brachial BP. Second, we expected taller people to have less prevalence of coronary artery disease.

## METHODS

### Study participants

Participants (n = 1,413) were randomly selected from the Canberra electoral role and invited by letter to participate in the study.[21] There was an 84% response rate (n = 1,187). Participants were excluded from analysis if they presented with irregularities that may have resulted in errors in central BP estimation. These included arrhythmia or tachycardia (resting heart rate >100 bpm; n = 20) or poor quality arterial pressure waveforms (n = 7). Eight participants were also excluded because height was not recorded. A total of 1,152 participants were included for analysis.

### Study protocol

Participants attended the Canberra Hospital for anthropomorphic measurements and assessment of clinical history, brachial BP, central BP, and aortic stiffness. The study complied with the Declaration of Helsinki and was approved by local human research ethics committees; participants provided written informed consent.

### Anthropometry

Height and weight were measured in light clothing with shoes removed. Height was measured with each participant's head in the Frankfurt plane to the nearest 0.5 cm using a Seca 220 stadiometer (Vogel & Halke, Hamburg, Germany). Body mass index was calculated as weight in kilograms divided by height in meters squared.

### Clinical history

A self-administered questionnaire was used to gather information on medical history, including current medication use. A history of coronary artery disease was self-reported by participants and confirmed from medical records. Hypertension was defined as brachial BP ≥140/90 mm Hg or the use of antihypertensive medication. Presence of type 2 diabetes was defined as a fasting serum glucose ≥7 mmol/L (≥126 mg/dL) or the use of insulin or oral hypoglycemic agents. Hyperlipidemia was defined by self-report as receiving treatment to lower blood cholesterol or eligibility to receive treatment according to the Pharmaceutical Benefits Scheme of Australia. Smoking history was self-defined as current smoker (including those in the process of quitting), past smoker, or nonhabitual smoker.

### Brachial BP

Brachial systolic and diastolic BPs were determined by mercury sphygmomanometry from the first and fifth Korotkoff phases, respectively. After 10 minutes of rest in the seated position, 2 readings were taken on the right arm with 5 minutes between each recording. A third reading was taken 10 minutes after this, and an average of the 3 readings was used for analysis. Brachial pulse pressure was defined as the difference between systolic and diastolic BP.

### Central BP

Immediately after the brachial BP readings, radial applanation tonometry was undertaken to synthesize an ascending aortic pressure waveform and central BP indices using a validated[22] and reproducible[23] generalized transfer function (SphygmoCor 8.1; AtCor Medical, Sydney, Australia). The radial waveform was calibrated using the average brachial BP values obtained as described above. In addition to the transfer function, central systolic BP was also determined from the second systolic peak on the radial waveform (radial PP2).[24] Several variables were generated from the central pressure waveform (see Figure 1 for examples). Augmentation pressure was the difference between the first and second systolic peaks, with augmentation index expressed as a percentage of the pulse pressure. Because augmentation is dependent on heart rate, this variable was normalized to 75 bpm. Aortic pulse wave timing ($T_R$) was calculated as the time from the systolic upstroke to the inflection point (Pi) after the first systolic peak (P1) on the central waveform.[25] Central pulse pressure was defined as the difference between central systolic and diastolic BPs. Pulse pressure amplification was defined as the ratio between brachial and central pulse pressure. Systolic amplification was defined as brachial systolic BP minus central systolic BP.

### Aortic stiffness

Aortic pulse wave velocity was measured by electrocardiogram-gated sequential applanation tonometry of the carotid and femoral arteries (SphygmoCor 8.1; AtCor Medical) as recommended.[26] Aortic path length was defined as the distance from the supra-sternal notch, via the umbilicus, to the femoral measurement site, minus the distance from the carotid measurement site to the supra-sternal notch.

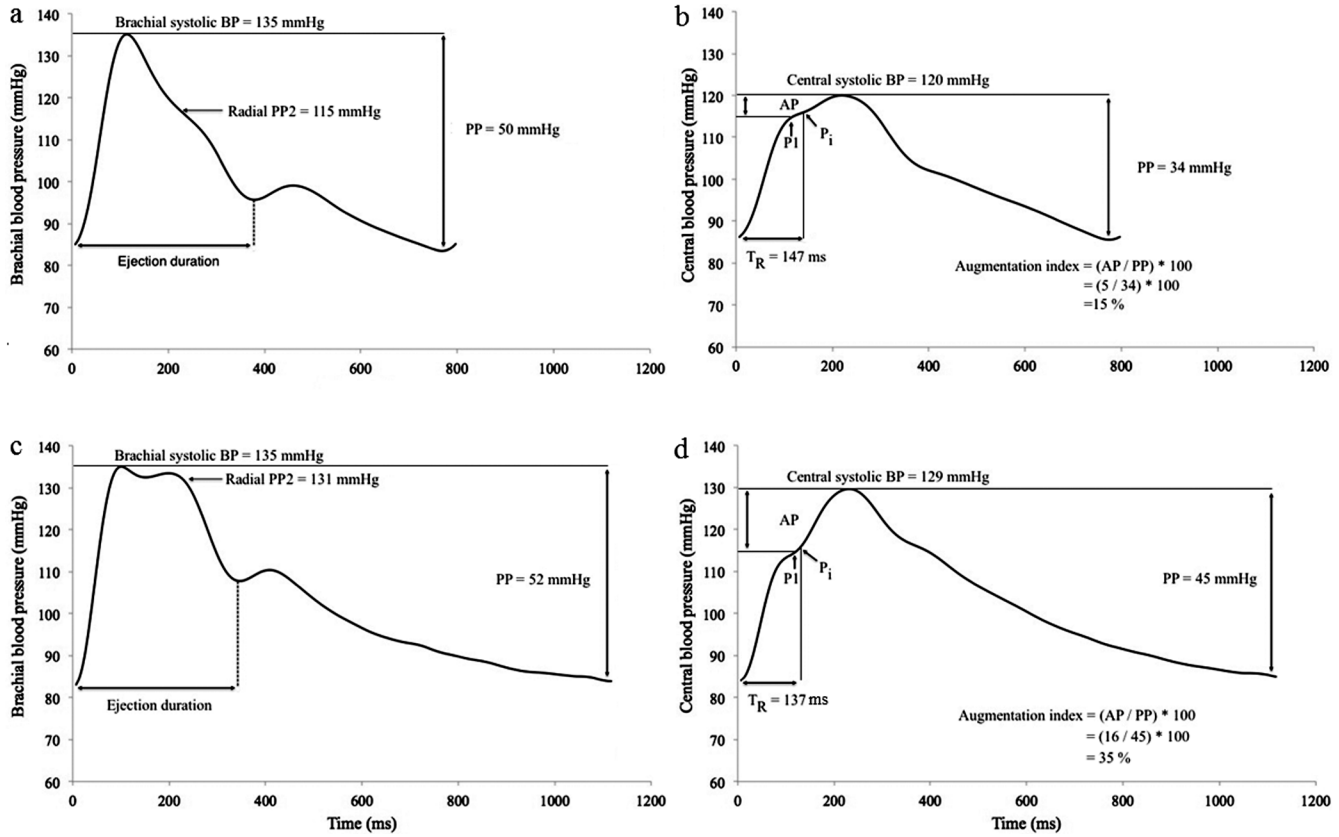


**Figure 1.** Peripheral and generated central waveforms from 2 female participants of similar brachial blood pressure (BP) and differing heights. (**a** and **b**) Brachial and central BP for a participant aged 49 years and 173 cm in height. (**c** and **d**) Brachial and central BP for a participant aged 49 years and 160 cm in height. Abbreviations: AP, augmentation pressure; BP, blood pressure; P1, central first systolic peak; Pi, inflection point; PP, pulse pressure; PP2, radial second systolic peak; $T_R$, aortic pulse wave timing.

### Statistical analysis

Analysis was performed with SPSS version 14.0 (SPSS, Chicago, IL), with $P < 0.05$ considered to be significant. Data were assessed for normality and transformed where necessary. Differences between groups were determined with Student independent *t* tests, and univariable associations were assessed by Pearsons product moment correlations. Differences between the slopes of associations between height and key variables were assessed by *Z* statistics. Multiregression analysis was undertaken with the stepwise forward enter method with collinearity diagnostics. Independent variables were included in each model when univariable associations with each dependent variable were $P < 0.20$, and all models were corrected for traditional cardiovascular risk factors as detailed in the Results. Logistic regression was undertaken to assess the relationship between height and coronary artery disease status. All results were stratified according to sex, and analyses were conducted with subjects dichotomized by sex into greater than and less than median height values.

## RESULTS

### Participant characteristics

Ninety-seven percent of the population was white, and participant characteristics are presented in Table 1. The median height was 174 cm for men and 161 cm for women. Compared with men of less than median height, taller males were younger, heavier, and less likely to have hypertension or a history of coronary artery disease. The significantly lower prevalence of coronary artery disease in taller men remained after correction for hypertension, vasoactive medication, smoking status, and hyperlipidemia and diabetes status or therapy (odds ratio (OR = 0.48; 95% confidence interval (CI) = 0.28–0.87; $P = 0.006$). Taller men were also less likely to be taking vasoactive medications, statins, and oral hypoglycemic medications. No other significant differences were observed. Similar results were apparent in the female cohort, with the exception of hypercholesterolemia and medication use. Taller women were less likely to have hypercholesterolemia and to use diuretics than shorter women. The lower prevalence of coronary artery disease in taller women also remained significant after correction for the same factors as per the analysis in men (OR = 0.43; 95% CI = 0.19–0.96; $P = 0.04$).

### Hemodynamic characteristics and height

Table 2 shows the hemodynamic characteristics of the study participants according to height. Compared with men of less than median height, the taller men had

**Table 1.** Subject characteristics according to height status

| Variable | Men | | | Women | | |
|---|---|---|---|---|---|---|
| | Less than median height (n = 277) | Greater than median Height (n = 272) | *P* value | Less than median height (n = 299) | Greater than median height (n = 304) | *P* value |
| Age, y (range) | 71 (48–93) | 64 (48–93) | <0.001 | 72 (48–93) | 63 (48–89) | <0.001 |
| Weight, kg, mean (SD) | 78 (12) | 90 (14) | <0.001 | 67 (13) | 76 (15) | <0.001 |
| Height, cm, mean (SD) | 168 (4.4) | 180 (4.2) | <0.001 | 156 (3.9) | 167 (5) | <0.001 |
| Body mass index, kg/m$^2$, mean (SD) | 27.6 (3.9) | 27.8 (4.0) | 0.43 | 27.8 (5.2) | 27.3 (5.4) | 0.33 |
| Systemic hypertension, no. (%) | 148 (53) | 110 (40) | 0.002 | 167 (56) | 135 (44) | 0.005 |
| Diabetes mellitus, no. (%) | 36 (13) | 24 (8.8) | 0.12 | 33 (11) | 21 (6.9) | 0.08 |
| Hypercholesterolemia, no. (%) | 122 (44) | 103 (38) | 0.14 | 138 (46) | 109 (36) | 0.01 |
| Coronary artery disease, no. (%)[a] | 53 (19) | 25 (9.2) | 0.001 | 23 (7.7) | 9 (3.0) | 0.01 |
| Vasoactive medication, no. (%) | 154 (56) | 103 (38) | <0.001 | 165 (55) | 106 (35) | <0.001 |
| ACE/ARB | 128 (46) | 84 (31) | <0.001 | 123 (41) | 87 (29) | 0.001 |
| Calcium blocker | 38 (14) | 21 (7.7) | 0.02 | 35 (12) | 22 (7.2) | 0.06 |
| Beta-blocker | 43 (16) | 24 (8.8) | 0.02 | 42 (14) | 27 (8.9) | 0.046 |
| Nitrate | 6 (2.2) | 2 (0.7) | 0.16 | 7 (2.3) | 2 (.7) | 0.09 |
| Diuretic | 44 (16) | 30 (11) | 0.10 | 75 (25) | 48 (16) | 0.005 |
| Alpha-blocker | 9 (3.3) | 6 (2.2) | 0.45 | 3 (1.0) | 0 (0) | 0.08 |
| Statin, no. (%) | 109 (39) | 68 (25) | <0.001 | 117 (39) | 60 (20) | <0.001 |
| Insulin, no. (%) | 7 (2.5) | 2 (0.7) | 0.10 | 3 (1.0) | 3 (1.0) | 0.98 |
| Oral hypoglycemic agents, no. (%) | 29 (10) | 15 (5.5) | 0.03 | 22 (7.4) | 10 (3.3) | 0.03 |
| Never smoker, no. (%) | 113 (41) | 131 (48) | 0.08 | 193 (65) | 190 (63) | 0.60 |

Abbreviations: ACE, angiotensin-converting enzyme inhibitors; ARB, angiotensin receptor blockers.
[a]Remained significant in both men and women after adjustment for hypertension, vasoactive medication, smoking status, hyperlipidemia, and diabetic status or therapy.

significantly lower brachial systolic BP, brachial pulse pressure, central systolic BP (by generalized transfer function or radial PP2), central pulse pressure, augmentation pressure, augmentation index, augmentation index at 75 bpm, and aortic pulse wave velocity. On the other hand, taller men had significantly higher brachial diastolic BP, central diastolic BP, pulse pressure amplification, and $T_R$. No significant differences were observed in heart rate, mean arterial pressure, central first systolic peak, or systolic BP amplification. Similar findings were evident between women of greater than and less than median height, with the exception of heart rate and central first systolic peak, which were slightly (but significantly) lower in taller women. Furthermore, mean arterial pressure was lower (of borderline significance) in taller women. Example pressure waveforms typifying the height differences between female participants are depicted in Figure 1.

### Univariable relationships between height and central and peripheral hemodynamics

Table 3 and Figure 2 present correlations between height and key variables stratified by sex. When $T_R$ was adjusted for aortic pulse wave velocity, there was still a significant relationship with height in both men ($r = 0.26$; $P < 0.001$) and women ($r = 0.25$; $P < 0.001$). For the entire study population (including men and women), the slope of the association between height and central systolic BP (by PP2) was significantly stronger than the slope of the association between height and brachial systolic BP ($Z = 2.50$; $P = 0.01$). Height was a weak, albeit statistically significant, correlate of aortic pulse wave velocity.

### Multiple regression analyses

Multiple regression models were undertaken to determine the relation between height (as the first independent variable in the model) and the following dependent variables: brachial systolic BP, central systolic BP, radial PP2, $T_R$, augmentation index at 75 bpm, and aortic pulse wave velocity. Each model included the following independent variables: age, body mass index, coronary artery disease (0 = no, 1 = yes), smoking history (0 = nonsmoker, 1 = ex-smoker, 2 = current smoker (or in the process of quitting)), type 2 diabetes (0 = no, 1 = yes), total cholesterol, medication use (angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, calcium blockers, beta-blockers, nitrates, diuretics, alpha-blockers, statins, insulin, and oral hypoglycemic agents), heart rate, aortic pulse wave velocity (except

**Table 2.** Participants' hemodynamic characteristics according to height

| Variable | Men | | | Women | | |
|---|---|---|---|---|---|---|
| | Less than median height (n = 277) | Greater than median height (n = 272) | *P* value | Less than median height (n = 299) | Greater than median height (n = 304) | *P* value |
| Heart rate, bpm | 62 (10) | 61 (9.8) | 0.51 | 65 (10) | 63 (9) | 0.03 |
| Brachial systolic blood pressure, mm Hg | 150 (21) | 143 (19) | <0.001 | 153 (23) | 142 (19) | <0.001 |
| Brachial diastolic blood pressure, mm Hg | 84 (11) | 86 (10) | 0.02 | 80 (10) | 83 (9) | 0.001 |
| Brachial pulse pressure, mm Hg | 66 (19) | 57 (16) | <0.001 | 72 (22) | 59 (18) | <0.001 |
| Central systolic blood pressure, mm Hg | 140 (22) | 132 (18) | <0.001 | 144 (22) | 134 (18) | <0.001 |
| Central diastolic blood pressure, mm Hg | 85 (11) | 87 (10) | 0.02 | 82 (10) | 84 (9) | 0.003 |
| Central pulse pressure, mm Hg | 55 (18) | 45 (16) | <0.001 | 62 (21) | 50 (17) | <0.001 |
| Mean arterial pressure, mm Hg | 106 (12) | 105 (11) | 0.42 | 105 (12) | 103 (10) | 0.051 |
| Central first systolic peak, mm Hg | 122 (14) | 120 (13) | 0.27 | 120 (14) | 118 (13) | 0.04 |
| Radial second systolic peak, mm Hg | 141 (24) | 132 (20) | <0.001 | 146 (24) | 136 (20) | <0.001 |
| Augmentation pressure, mm Hg | 17 (9.5) | 13 (8.6) | <0.001 | 23 (10) | 18 (9) | <0.001 |
| Augmentation index, % | 30 (9) | 25 (10) | <0.001 | 37 (8) | 34 (8) | <0.001 |
| Augmentation index at 75 bpm, % | 24 (8) | 19 (10) | <0.001 | 32 (7) | 28 (7) | <0.001 |
| Aortic pulse wave timing, ms | 139 (12) | 145 (12) | <0.001 | 131 (10) | 136 (10) | <0.001 |
| Aortic pulse wave velocity, m/s | 10.4 (2.9) | 9.6 (2.6) | 0.002 | 10.0 (2.7) | 9.2 (2.4) | <0.001 |
| Pulse pressure amplification, ratio | 1.23 (0.13) | 1.29 (0.16) | <0.001 | 1.17 (0.09) | 1.9 (0.10) | 0.007 |
| Systolic blood pressure amplification, mm Hg | 10.2 (4.8) | 10.8 (4.7) | 0.13 | 8.5 (3.9) | 8.1 (3.9) | 0.15 |

Data are mean (SD).

**Table 3.** Univariable Pearson values for correlations of height and key variables stratified by sex

| Variable | Men (n = 549) | | Women (n = 603) | |
|---|---|---|---|---|
| | *r* | *P* value | *r* | *P* value |
| Age | −0.38 | <0.001 | −0.42 | <0.001 |
| Brachial systolic blood pressure | −0.19 | <0.001 | −0.28 | <0.001 |
| Brachial diastolic blood pressure | 0.13 | 0.003 | 0.13 | 0.005 |
| Brachial pulse pressure | −0.28 | <0.001 | −0.34 | <0.001 |
| Brachial mean pressure | −0.05 | 0.27 | −0.15 | 0.0003 |
| Central first systolic peak | −0.09 | 0.04 | −0.18 | <0.001 |
| Radial second systolic peak | −0.23 | <0.001 | −0.30 | <0.001 |
| Heart rate | −0.09 | 0.04 | −0.11 | 0.006 |
| Augmentation pressure | −0.30 | <0.001 | −0.34 | <0.001 |
| Augmentation index | −0.29 | <0.001 | −0.24 | <0.001 |
| Augmentation index at 75 bpm | −0.34 | <0.001 | −0.33 | <0.001 |
| Aortic pulse wave timing | 0.33 | <0.001 | 0.30 | <0.001 |
| Central systolic blood pressure | −0.20 | <0.001 | −0.28 | <0.001 |
| Central diastolic blood pressure | 0.12 | 0.005 | 0.10 | 0.01 |
| Central pulse pressure | −0.30 | <0.001 | −0.34 | <0.001 |
| Aortic pulse wave velocity | −0.18 | <0.001 | −0.18 | <0.001 |

Data adjusted for age and heart rate.

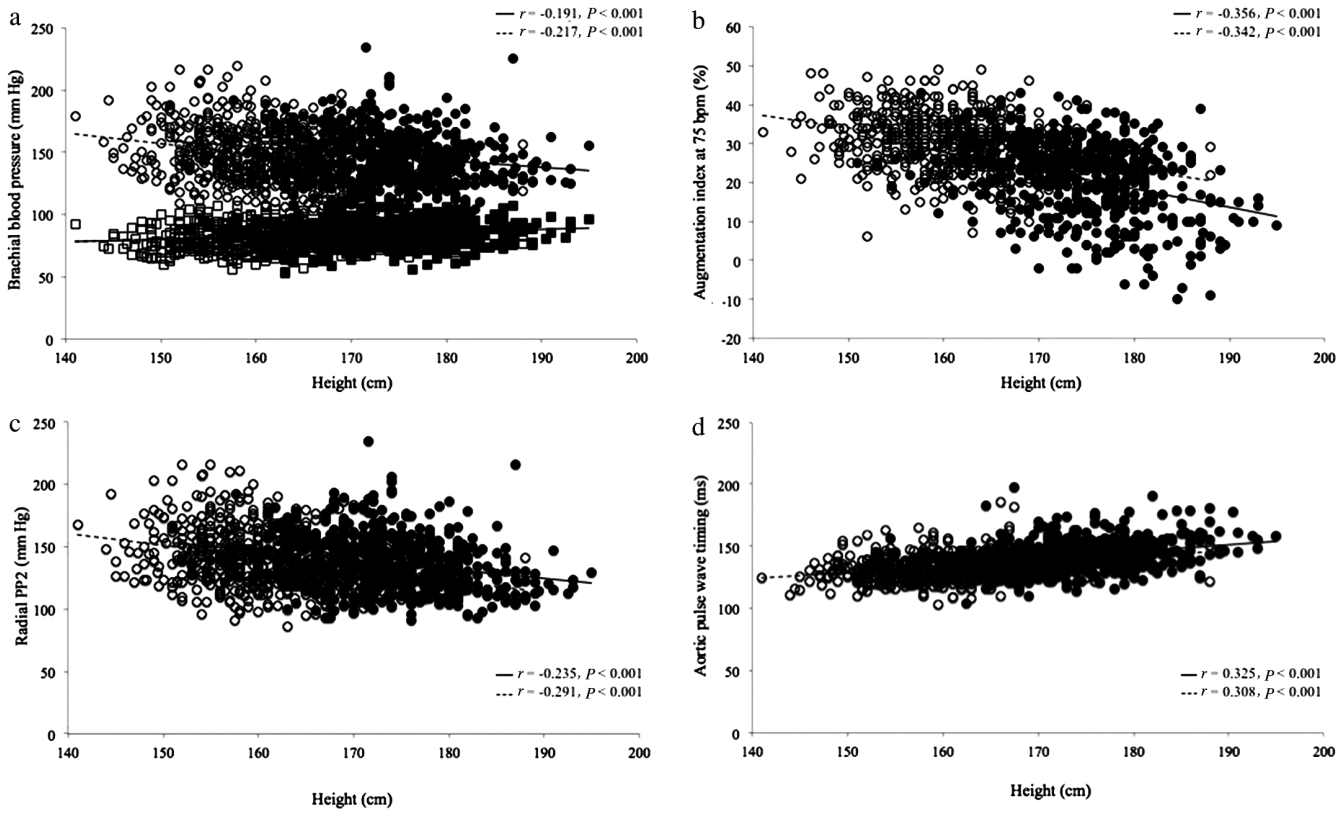


**Figure 2.** Relationships with height and key hemodynamic variables. (**a**) Circles represent systolic blood pressure, with open circles representing women and the closed circles representing men. Squares represent diastolic blood pressure, with open squares representing women and the closed squares representing men. (**b**–**d**) The open circles and squares represent women, and the solid circles and squares represent men for the designated variables. In all parts, solid lines indicate the regression trend lines for men, and the dashed line indicates regression trend lines for women.

for when this was the dependent variable), and mean arterial pressure. The results are summarized in Table 4. There was no significant relationship between height and peripheral systolic BP or aortic pulse wave velocity in men or women. However, in both sexes, significant independent associations were observed between height and central systolic BP (estimated from either the generalized transfer function or radial PP2), $T_R$, and augmentation index at 75 bpm.

## DISCUSSION

To our knowledge, this is the first study to report on the relationship of height and central and peripheral hemodynamics in a large population-based sample. Although this study does not exclude the possibility of a relation between height and peripheral BP, the main finding was that the relation of height with central BP was stronger than the relationship of height with brachial BP. Indeed, height was independently related to central systolic BP, augmentation index, and $T_R$ but not to brachial systolic BP or aortic stiffness. Height was also found to be inversely associated with the prevalence of traditional cardiovascular risk factors such as hypertension and established coronary artery disease. The observation that taller persons have more favorable central hemodynamics in this study may contribute to the inverse association between height and cardiovascular mortality that has been previously documented.

Although studies have examined the relationship between height and cardiovascular mortality,[2–9] few have considered the influence of brachial BP or history of hypertension in their multivariable models.[6–9] In each case, inclusion of these variables in the models did not significantly alter the magnitude of the association between height and mortality. A possible reason for this is that brachial BP does not necessarily give an accurate indication of the BP to which the organs are exposed.[10,11] It is now well appreciated that central BP is a more important determinant of risk related to BP than brachial BP. Thus, our observations of independent associations between height and central BP (i.e., systolic BP and augmentation index), but not brachial BP, are of relevance to the conclusions of previous studies that suggest that BP does not mediate the relation between height and cardiovascular mortality.[6–9] Indeed, only accounting for brachial BP is likely to overlook the prominent contribution of central BP in the association between height and cardiovascular mortality. Beyond the relation with cardiovascular death, central BP could also have relevance to other conditions, such as preeclampsia, in which shorter statue is associated with higher risk.[27]

Independent of mean arterial pressure and other cardiovascular risk factors, augmentation index normalized to 75

**Table 4.** Multiple regression analysis for sex-specific associations with height as an independent variable (n = 549 men; n = 603 women)

| Dependent variable | Sex | Beta coefficient | *P* value | Adjusted $R^2$ |
|---|---|---|---|---|
| Brachial systolic blood pressure, mm Hg | Women | −0.115 | 0.051 | 0.84 |
| | Men | −0.096 | 0.09 | 0.82 |
| Central systolic blood pressure, mm Hg | Women | −0.172 | <0.001 | 0.88 |
| | Men | −0.139 | 0.002 | 0.89 |
| Radial second systolic peak, mm Hg | Women | −0.281 | <0.001 | 0.88 |
| | Men | −0.239 | <0.001 | 0.88 |
| Aortic pulse wave timing, ms | Women | 0.262 | <0.001 | 0.19 |
| | Men | 0.224 | <0.001 | 0.38 |
| Augmentation index at 75 bpm, % | Women | −0.224 | <0.001 | 0.33 |
| | Men | −0.189 | <0.001 | 0.52 |
| Aortic pulse wave velocity, m/s | Women | 0.025 | 0.07 | 0.34 |
| | Men | 0.014 | 0.15 | 0.39 |

bpm had the strongest correlation with height of all indices. Given that augmentation index has been shown by meta-analysis to predict mortality above and beyond brachial BP,[14] we speculate that raised augmentation index associated with being shorter could be a strong explanatory factor in those studies showing inverse relationships between height and cardiovascular mortality. The lack of association between height and aortic pulse wave velocity when assessed by multiple regression analysis implies a lesser role of measures of central artery stiffness than measures of central BP on cardiovascular outcomes related to height, although this hypothesis needs to be tested prospectively. Notably, $T_R$ had markedly different associations with height compared with aortic pulse wave velocity. This observation supports the recommendation that $T_R$ should not be used as a surrogate of aortic stiffness, as suggested by Westerhof *et al.*[28]

According to the principal textbook of arterial hemodynamics,[29] the increase in augmentation index associated with being shorter would mainly be explained by increased wave reflection due to decreased distance to major reflection sites. Thus, with each cardiac ejection there would be a reduction in the round-trip travel time of aortic incident and reflected waves, such that reflected waves arrive earlier in systole and augment central systolic BP (and augmented pressure). This theory is supported by our observations that $T_R$ was significantly reduced in shorter men and women, as well as the significant positive relationship between height and $T_R$, even after adjustment for aortic pulse wave velocity. On the other hand, the shorter $T_R$ in shorter people could equally be explained by increased impedance to aortic outflow (i.e., impaired aortic reservoir characteristics) and mismatched ventricular–vascular interaction relative to taller people.[30] This is a more likely physiological explanation that is supported by several recent investigations on aortic reservoir function, wave intensity analysis, and aortic pulse wave timing,[30–36] which cast doubt on wave reflection (particularly from single dominant sites)[37] as being a dominant factor contributing to central arterial waveform morphology. Because neither wave reflection nor aortic reservoir characteristics have been directly measured in this study, further work will be needed to understand the true physiology underlying the height and central hemodynamic relationships.

This is a cross-sectional study and therefore causality cannot be inferred. Furthermore, the study population did not have significant ethnic or socioeconomic diversity, which may limit generalizability of the findings to races other than white or populations with a larger socioeconomic gradient. The noninvasive measures of central hemodynamics could have introduced a source of error due to calibration with brachial BP. Aside from this, should there have been a higher level of brachial-to-radial systolic BP amplification in taller individuals compared with shorter individuals, systematic underestimation of central SBP could have occurred in taller people. This said, the magnitude of upper limb pressure amplification has been shown to be independent of height, age, body mass index, and the level of mean arterial pressure.[38] Furthermore, invasive techniques are unsuitable for population-based studies, and calculation of augmentation index would not be affected by these potential calibration issues. Lastly, the older participants in this study could have been exposed to childhood factors detrimental to height development and cardiovascular health related to central hemodynamics. However, although shorter participants were significantly older than the taller participants, analyses were corrected for the potential influence of age.

This study found that height was inversely and independently related to central hemodynamics after correction for traditional cardiovascular risk factors. Overall, taller people had more favorable central hemodynamics, and this was observed regardless of age or sex and despite significantly less use of antihypertensive medication. These findings suggest that the inverse relationship between height and cardiovascular mortality could be a consequence of raised central BP and impaired ventricular–vascular interaction. Our observation that taller participants have improved central hemodynamics, together with a reduced prevalence of both hypertension and antihypertensive medication use, suggests a potential usefulness for height as a factor in cardiovascular risk estimation, hypertension diagnosis, and consequent treatment.

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## DISCLOSURE

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