Influence of Body Height on Pulsatile Arterial Hemodynamic Data

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Objectives. This study sought to present evidence that short stature is a hemodynamic liability, which could explain in part the inverse relation between body height and cardiovascular risk.

Background. Other explanations for the association of short stature with increased cardiovascular risk include advancing age, reduced pulmonary function, genetic factors, poor childhood nutrition and small-caliber coronary arteries. This study adds another factor—the physiologic effects of reduced body height on the arterial tree, which increase left ventricular work and jeopardize myocardial perfusion.

Methods. Four hundred two subjects were studied: 149 with end-stage renal disease and 253 with normal renal function. Measurements included blood pressure, body height, cardiac cycle length, carotid to femoral artery pulse wave velocity, carotid artery pulse waves (by applanation tonometry) and the arrival time of reflected waves. Calculations included the carotid augmentation index, carotid artery compliance and the diastolic to systolic pressure–time ratio (an index of myocardial supply and demand).

Results. On linear and stepwise multiple regression, body height correlated with all variables except mean blood pressure.

Conclusions. The early systolic arrival of reflected waves in short people in this group acts to stiffen the aorta and increase the pulsatile effort of the left ventricle, even at the same mean blood pressures. Short stature also induces a faster heart rate, which increases cardiac minute work and shorten diastole. Stiffening lowers the aortic diastolic pressure and, coupled with a shortened diastole, could adversely influence myocardial supply. Although indirect, this evidence supports a physiologic hypothesis for the body height–cardiovascular risk association.

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A relation between body height and the risk of cardiovascular disease, specifically coronary heart disease (CHD), was reported as early as 1951 by Gertler et al. (1), and has been repeatedly observed in the last 15 years (2–15). The observation has been reported from many countries including England and Wales (6,7,14), Norway (16), Finland (17), Sweden (5,10) and the United States (2,4,8,11–13,15), and persists even after adjustment for known CHD risk factors. Gender has not been an issue, because the phenomenon occurs in both women and men, although not always in the same study (8,12,15). Disclaimers have arisen, not about the presence of the observation, but rather about its explanation.

Aging (16) and abnormal lung function (13,18,19) have been proposed as factors that influence the statistical association between short stature and CHD. However, when these factors are accounted for within the statistical models, the association persists, albeit weaker (8,11,14,16). The role of environmental factors, such as poor socioeconomic conditions and low birth weight, have been frequently reported in the United Kingdom (6,7,9,20,21), but have not been confirmed in the United States, where the relation has been found in women of all levels of education (8) and in the relatively homogeneous populations of nurses and physicians (11,15). Explanations based on the pathophyslogic mechanisms of cardiovascular diseases may be more direct and therefore more persuasive as explanations for the relation between CHD and short stature. Although there are no known direct genetic links connecting body height to CHD, short stature and a predisposition for CHD are both known to “run in families” (10,13). This could be explained in part by an analysis of the Coronary Artery Surgery Study (CASS) registry data, which showed that among patients who underwent coronary artery bypass graft surgery, the coronary arteries were smaller and the operative mortality higher in short people (4). Similar conclusions were reached in patients undergoing percutaneous transluminal coronary angioplasty (22).

In the published data, there is information to suggest a physiologic explanation for this phenomenon. Comparative physiologists have observed a consistent relation between body length, arterial path length and heart rate in animals (23,24). Although well described among species, this relation is not documented within the same species. If true in humans, short stature and arterial length would shorten the cardiac cycle length and increase ventricular minute work. Furthermore, a

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short arterial tree brings arterial pulse wave reflection sites closer to the heart. Reflected pressure waves then return to the aorta in systole, rather than diastole, and amplify the primary wave. This would increase the pulsatile work of the left ventricle and make the ventricle potentially more vulnerable to any form of heart disease, especially CHD.

The association between cardiovascular risk and short stature has been supported in patients with end-stage renal disease (ESRD) studied in our laboratory (25–27). In this group, a strong inverse correlation has been observed between body height and arterial wave reflections (25–27). Because patients with ESRD are known to have stiff arterial trees (28) and reduced body height as a consequence of kidney disease, our working hypothesis is that this pattern may be a representation of the same physiologic mechanism present in comparative physiologic studies and also suggested by the preliminary study of a small group of 85 normotensive and hypertensive subjects (27). Thus, the purpose of the present study was to correlate body height with several measures of pulsatile arterial hemodynamic data in a large group of normal and hypertensive subjects, with and without the presence of ESRD. Establishing these correlations should allow us to add ventricular-vascular mismatch as a physiologic mechanism to the other factors already presented as explanations for the body height–cardiovascular risk phenomenon.

**Methods**

**Subjects.** Measurements were made in 402 subjects: 149 with ESRD on dialysis and 253 with normal renal function. Those with normal renal function were asymptomatic hospital employees with no known disease or patients with minor, noncirculatory disturbances who were matched in terms of age and gender with the ESRD group. Because patients with ESRD may have normal or elevated blood pressures, 60 patients with uncomplicated essential hypertension were included in the normal renal function group so that the blood pressure of both groups would also be matched. In all hypertensive patients, with or without ESRD, all beta-blocking drugs and calcium channel antagonists were discontinued 4 weeks before the study to avoid any confounding effects these drugs might have on heart rate relations. Measurements in patients with ESRD were made just before their mid-week dialysis (25–27). The augmentation index was measured in 333 subjects: 195 with ESRD and 138 with normal renal function. Measurements of carotid artery compliance were carried out in 155 subjects: 102 with ESRD and 53 with normal renal function. All subjects in both groups were also evaluated for height, heart period and brachial artery pressures. Patients with known CHD, valvular heart disease, cerebral vascular disease, peripheral vascular disease, congestive heart failure or diabetes mellitus were excluded from both study groups. The studies were approved by the institution’s Review Committee, and each subject gave written informed consent.

**Blood pressure and cardiac cycle length.** Brachial artery pressures were measured with a cuff and mercury sphygmomanometer after 15 min of recumbency. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded at the first and fifth Korotkoff phases. The average RR interval during three respiratory cycles on the body surface electrocardiogram (ECG) was calculated for the heart period.

**Common carotid artery (CCA) pressure and waveform.**

The CCA waveform was recorded noninvasively with a pencil-type probe incorporating a high fidelity strain gauge transducer (SPT-301, Millar Instruments) on a Gould 8188 recorder (Gould Electronique, Ballainvilliers, France) at a paper speed of 100 mm/s. A detailed description of this system has been published previously (29–31). The tonometer is internally calibrated using a Millar preamplifier (TCB-500).

SBP and pulse pressure (PP) may increase from the central to peripheral arteries, while the reductions in diastolic or mean blood pressure (MBP) from the ascending aorta to the radial artery does not exceed 2 to 3 mm Hg (32–34). Therefore, the CCA pressure wave was calibrated assuming that brachial and carotid artery DBPs and MBPs were equal. MBP on the CCA pressure wave was identified from the area of the CCA pressure wave in the corresponding heart period, and set equal to brachial MBP. CCA pressure amplitude (PP) was then computed from the brachial artery DBP and the position of MBP on the CCA pressure wave (29–31,35,36).

The CCA pressure wave was analyzed according to Muro et al. (37) (Fig. 1). The height of the late systolic peak (Ppk) above the inflection point (Pi) is \( \Delta P \), where \( \Delta P = P_{pk} - P_i \). The \( \Delta P \) to PP ratio defines the augmentation index, which represents the effect of arterial wave reflections on blood pressure in the central arteries. The \( \Delta t_p \) represents the travel time of the pulse wave to peripheral reflecting sites and back.

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**Abbreviations and Acronyms**

CCA = common carotid artery  
CHD = coronary heart disease  
DBP = diastolic blood pressure  
DPTI/SPTI = diastolic/systolic pressure–time index  
ESRD = end-stage renal disease  
MBP = mean blood pressure  
PP = pulse pressure  
PWV = pulse wave velocity  
SBP = systolic blood pressure  
\( \Delta t_p \) = travel time of reflected wave

**Figure 1.** Contour of the carotid pressure waveform recorded at the speed of 100 mm/s in a hypertensive patient. PP = pulse pressure; \( P_{pk} \) = mid to late systolic peak; \( P_i \) = early inflection point; \( \Delta t_p \) = travel time of the reflected wave.
server correlation for repeated measurements of carotid
to the area under the diastolic
trace, with zero as baseline (38–40). These areas, measured
be computer, were averaged for 10 beats. Reproducibility has
been previously reported (25–27,40).

Carotid-femoral artery pulse wave velocity (PWV).
Carotid-femoral artery PWV was determined using the foot-
to-floor method (30,41). Transcutaneous Doppler flow velocity
recordings were carried out simultaneously at the base of the
neck over the CCA and the femoral artery in the groin with an
8-MHz Doppler unit (SEGA M842, Société d’Electronique
Générale et Appliquée, Paris, France) and a Gould 8188
recorder. The time delay (t) was measured between the feet of
the flow waves recorded at these points. The distance (D)
traveled by the pulse wave was measured over the body surface
as the distance between the two recording sites minus that
from the suprasternal notch to the carotid artery recording site.
PWW was calculated as PWV = D/t. The reproducibility of
the measurement for aortic PWV, expressed as the percent mean
(SD) value, was 5.3 ± 1.2% (25–28). The distance between
the suprasternal notch and the femoral artery recording site
was also used as an approximation of aortic length.

Statistics. Data are presented as mean values ± SD. The Student t
-test was used to compare patients with ESRD with
those with normal renal function. Simple linear regression
analysis correlated body height with other variables individu-
ally. Stepwise multiple regression analysis assessed the percent
variation in individual measurements explained by body height
and other interrelated factors.

Results

Table 1 compares the mean values of the group with ESRD
with those of the group without renal disease. As described in

### Table 1. Baseline Data

<table>
<thead>
<tr>
<th></th>
<th>Normal Renal Function (n = 195)</th>
<th>ESRD (n = 138)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>48.9 ± 20 (17 to 98)</td>
<td>52.9 ± 16.9 (13 to 87)</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>68.8 ± 13 (33 to 122)</td>
<td>61 ± 13 (39 to 98)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>168.5 ± 11 (138 to 197)</td>
<td>163 ± 19 (138 to 198)</td>
<td>&lt;0.025</td>
</tr>
<tr>
<td>Gender ratio (M/F)</td>
<td>1.40</td>
<td>1.43</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic BP (mm Hg)</td>
<td>141 ± 25 (103 to 236)</td>
<td>149 ± 31 (86 to 231)</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic BP (mm Hg)</td>
<td>83 ± 14 (53 to 120)</td>
<td>82 ± 15 (51 to 129)</td>
<td>NS</td>
</tr>
<tr>
<td>Heart period (ms)</td>
<td>918 ± 149 (570 to 1,326)</td>
<td>859 ± 133 (600 to 1,283)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Aortic PWV (cm/s)</td>
<td>907 ± 223 (540 to 1,720)</td>
<td>1,117 ± 337 (561 to 2,294)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Δp (ms)</td>
<td>133 ± 30 (60 to 210)</td>
<td>107 ± 23 (60 to 164)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Augmentation index (%)</td>
<td>7.9 ± 16.4 (−29.7 to +44.1)</td>
<td>23.6 ± 15 (−24 to +55)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>CCA compliance (kPa⁻¹m²⁻10⁻⁷)</td>
<td>5.5 ± 2.3 (2 to 12)</td>
<td>4.8 ± 2.2 (1.35 to 12.4)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>DPTI/SPTI</td>
<td>1.78 ± 0.28 (1.14 to 2.41)</td>
<td>1.49 ± 0.32 (0.85 to 2.39)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are presented as mean value ± SD (range). BP = blood pressure; CCA = common carotid artery; DPTI =
diastolic pressure–time index; ESRD = end-stage renal disease; F = female; M = male; PWV = pulse wave velocity;
SPTI = systolic pressure–time index; Δp = time from onset of carotid pulse to inflection point.

CCA diameters. CCA diameters and wall motion were measured by a high resolution B-mode (7.5-MHz transducer)
echo tracking system (Wall-Track system), allowing assessment of arterial wall displacement during the cardiac cycle. A
complete detailed description of this system has been published previously (36). The radiofrequency signal over six
heart cycles is digitized and stored in a large-memory computer. Two sample volumes, selected under cursor control, are
positioned on the anterior and posterior walls. The vessel walls are continuously tracked by sample volumes according to
phase, and the displacement of the arterial walls is obtained by auto correlation processing of the Doppler signal. The accuracy
of the system is ±30 μm for CCA diastolic diameter (Dd) and <1 μm for the stroke change in CCA diameter (Ds − Dd,
where Ds is the systolic diameter). The repeatability coefficient of the measurements was ±0.273 mm for CCA diameter and
±0.025 mm for Ds − Dd. Diameters were measured on the right CCA, 2 cm below the bifurcation. CCA lumen cross-
sectional area (LCSA) was calculated as LCSA = π (CCA diameter)²/4. A localized echo structure encroaching into the
vessel lumen was considered to be a plaque if the CCA intima-media thickness was >50% thicker than neighboring sites (29).
Measurements of CCA diameter were always performed in plaque-free arterial segments.

CCA compliance. CCA compliance was determined from changes in CCA diameter between systole and diastole and the
simultaneously measured CCA PP (ΔP) according to the following formula: CCA compliance = (π Dd[Ds − Dd]/2) ΔP
(m²-kPa−1·10⁻⁷). The repeatability coefficient of the measurement was 0.52 m²-kPa−1·10⁻⁷.

Diastolic/systolic pressure–time index (DPTI/SPTI). This index is calculated as the ratio of the area under the diastolic
to the area under the systolic portion of the carotid tonometer tracing, with zero as baseline (38–40). These areas, measured
by computer, were averaged for 10 beats. Reproducibility has been previously reported (25–27,40).
the Methods section, the two groups are matched for age, gender and blood pressure, but differ significantly in all other measurements, including heart period.

Correlations were calculated for body height against all other variables for the overall group and for the subgroups separately (Table 2). For the overall group, there was no relation between body height and MBP ($r = 0.08$), but significant correlations were found between body height and $\Delta t_p$ ($r = 0.52$, $p < 0.001$), cardiac cycle length ($r = 0.30$, $p < 0.001$), CCA compliance ($r = 0.31$, $p < 0.002$) and age ($r = 0.23$, $p < 0.001$). Scatterplots for the relation between body height and $\Delta t_p$ and body height and heart period are shown in Figure 2. Although these correlations are highly significant, linear regression analysis alone fails to consider the interrelation among the variables. Therefore, stepwise multiple regression analysis was carried out to determine how much of the variation in each of these measurements was due to body height and other factors (Table 3). For example, when PWV and body height are considered as independent variables of $\Delta t_p$, body height explains 27% of the variation in $\Delta t_p$, whereas the two together explain 71%. This finding is probably explained by the interrelation between PWV and body height. Nonetheless, body height has a significant influence on the time elapsed between the onset of the pulse and the detected arrival of the reflected wave. With regard to the augmentation index and its partial coefficients, body height explained only a small part of the variance, whereas the total of height, age, PWV and brachial artery SBP accounted for 46% of the variation. When the variations in heart period were similarly analyzed, body height explained 9% of the variations, whereas the addition of PWV explained 7% more, for a total of 16%. Although the percent variations explained by body height are small, all are highly significant. For easy review, these data are shown in Table 3.

**Table 2. Linear Correlations With Body Height as Dependent Variable**

<table>
<thead>
<tr>
<th>Independent Variable</th>
<th>No. of Pts</th>
<th>$r$ Value</th>
<th>$p$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Entire Group</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\Delta t_p$ (ms)</td>
<td>333</td>
<td>0.52</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>Augmentation index (%)</td>
<td>333</td>
<td>0.42</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>DPTI/SPTI</td>
<td>157</td>
<td>0.33</td>
<td>$&lt; 0.003$</td>
</tr>
<tr>
<td>CCA compliance</td>
<td>157</td>
<td>0.31</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>Heart period (ms)</td>
<td>402</td>
<td>0.30</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>Age (years)</td>
<td>402</td>
<td>0.23</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>Mean BP (mm Hg)</td>
<td>402</td>
<td>0.08</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Normal Renal Function</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Augmentation index (%)</td>
<td>195</td>
<td>0.47</td>
<td>$&lt; 0.001$</td>
</tr>
<tr>
<td>DPTI/SPTI</td>
<td>53</td>
<td>0.33</td>
<td>$&lt; 0.02$</td>
</tr>
<tr>
<td><strong>End-Stage Renal Disease</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Augmentation index (%)</td>
<td>138</td>
<td>0.25</td>
<td>$&lt; 0.005$</td>
</tr>
<tr>
<td>DPTI/SPTI</td>
<td>102</td>
<td>0.21</td>
<td>$&lt; 0.05$</td>
</tr>
</tbody>
</table>

$\text{Pts} = \text{patients; other abbreviations as in Table 1.}$

Similarly, CCA compliance was 9.0% explained by body height, whereas the addition of age and SBP accounted for a total of 46% of the variation. When the variations in heart period were similarly analyzed, body height explained 9% of the variations, whereas the addition of PWV explained 7% more, for a total of 16%. Although the percent variations explained by body height are small, all are highly significant. For easy review, these data are shown in Table 3.

**Table 3. Stepwise Multiple Regression**

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Independent Variable</th>
<th>Sequential $r^2$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Delta t_p$ (ms)</td>
<td>PWV (m/s)</td>
<td>0.27</td>
</tr>
<tr>
<td></td>
<td>Body height (cm)</td>
<td>0.71</td>
</tr>
<tr>
<td>Augmentation index (%)</td>
<td>Age (yr)</td>
<td>0.35</td>
</tr>
<tr>
<td></td>
<td>Body height (cm)</td>
<td>0.43</td>
</tr>
<tr>
<td></td>
<td>PWV (m/s)</td>
<td>0.53</td>
</tr>
<tr>
<td></td>
<td>BA SBP (mm Hg)</td>
<td>0.61</td>
</tr>
<tr>
<td>DPTI/SPTI</td>
<td>Body height (cm)</td>
<td>0.11</td>
</tr>
<tr>
<td></td>
<td>Age (yr)</td>
<td>0.21</td>
</tr>
<tr>
<td></td>
<td>BA SBP (mm Hg)</td>
<td>0.31</td>
</tr>
<tr>
<td>CCA compliance (kPa$^{-1}$m$^2$10$^{-7}$)</td>
<td>Heart period (ms)</td>
<td>0.58</td>
</tr>
<tr>
<td></td>
<td>Body height (cm)</td>
<td>0.09</td>
</tr>
<tr>
<td></td>
<td>Age (yr)</td>
<td>0.31</td>
</tr>
<tr>
<td></td>
<td>BA SBP (mm Hg)</td>
<td>0.46</td>
</tr>
<tr>
<td>Heart period (ms)</td>
<td>Body height (cm)</td>
<td>0.09</td>
</tr>
<tr>
<td></td>
<td>PWV (m/s)</td>
<td>0.16</td>
</tr>
</tbody>
</table>

$\text{BA = brachial artery; SBP = systolic blood pressure; other abbreviations as in Table 1.}$
A number of relations with body height were significantly different in the groups with and without renal disease (Table 2). The correlation coefficient for body height and augmentation index for the entire group was 0.42 ($p < 0.001$), but for the ESRD group, it was 0.25 ($p = 0.004$) and for the nonrenal disease group 0.47 ($p < 0.001$). The slopes of these regression lines were significantly different ($p < 0.01$). Similarly, the body height to DPTI/SPTI correlation for the overall group was 0.33, but it was 0.21 ($p = 0.036$) for the ESRD group and 0.33 ($p = 0.016$) for the nonrenal group.

The distance between the suprasternal notch and the femoral artery recording site offered an estimate of aortic length. This aortic length measurement correlated strongly with body height ($r = 0.93, p = 0.001$). However, aortic length correlations with augmentation index, $\Delta p$, heart rate and DPTI/SPTI were only equal to or less than those observed using body height. Therefore, aortic length values were not analyzed further.

**Discussion**

The major findings of this study are that short stature is associated with faster heart rates, shortened return times for reflected waves and increased augmentation of the primary systolic pulse, but is independent of MBP. These positive factors each operate to increase the stroke and minute work of the left ventricle, while reducing the diastolic time and pressure available for coronary filling. Although indirect, this evidence supports a physiologic explanation for the increased risk of cardiovascular disease in short people.

**Body height and reflected waves.** In this study, carotid pulse waves were used as a substitute for aortic pulses, a substitution that has been validated previously (42). Using carotid artery pulses as a surrogate (42), reflected waves to the aorta can be detected and semiquantified by use of the tonometric technique. These waves, initially described and classified by Murgo et al. (37), are easily identified and their arrival timed $\Delta t_{pr}$ from the onset of the primary pulse. The absolute magnitude of reflected waves cannot be measured, but the augmentation index offers a means of estimating their relative amplitude. Clinical credence has been lent to the augmentation index because it has been previously found to relate to left ventricular mass (25). This is probably due to an increase in left ventricular pulsatile work, a longer left ventricular ejection time and the need for the left ventricle to respond to an inappropriately timed late systolic increase in pressure (43)—all of which are liabilities in patients with CHD.

Arterial wave reflection sites are numerous and diffuse, but have been approximately localized, for the lower part of the body, to a region extending from the renal arteries to the aortic bifurcation (37,44). Because people of short stature necessarily have correspondingly short arterial trees, their reflecting sites are closer to the heart. The effect of reduced path length has also been demonstrated in young children (45) whose low body height and short arterial tree cause their aortic pulses to resemble those of the elderly. In contrast, aging in adults produces arterial tortuosity, increasing path length, but aging also stiffens the aorta and increases PWV, which returns the reflected wave to the aorta in systole. In short people even with normal wave velocities, the reflected waves return to the aorta earlier in systole, mimicking the effects of aging and requiring more pulsatile effort from the left ventricle. The importance of reflected waves in short people can be inferred from the strong relation of arrival times of the reflected waves ($\Delta t_{pr}$) and of the augmentation index to body height ($r = 0.52$ and 0.42, respectively). Interestingly, the increase in left ventricular pulsatile work imposed by these reflected waves can occur when MBP and systemic vascular resistance are both normal.

**Body height and heart rate.** Body height is a physiologic variable in another way. Comparative physiologists have observed a consistent relation between body length and heart rate in animals of different sizes (23). Because the wave velocities are similar in most animals, the heart rate–body length relation has been explained by the biologic need to have the reflected waves return to the aorta in diastole rather than in systole, to minimize the cardiac work of pulsations. An alternative explanation has been provided by Westerhof (24), who argued that the heart rate in animals of various sizes best relates to the rate of diastolic decay (time constant) of the arterial pulse. This is so because small arterial trees have small capacitances and require accordingly small stroke volumes to maintain the equivalent arterial pressures found in the animal kingdom. Cardiac cycle length must therefore be short to provide brief diastolic periods and prevent diastolic pressures from falling too low to maintain coronary perfusion. Fast heart rates in short animals meet these needs. Although the body length–heart rate relation is well described among different species, it has been only rarely applied in the same species (46). The principle also holds in humans in the present series, where, despite multiple factors influencing heart rate in both groups, body height showed an $r$ value of 0.32 when correlated with heart rate. Even when age and PWV were included in multiple stepwise regression analysis, body height remained a small but significant determinant of heart rate. An increased heart rate alone is another way in which reduced body height increases cardiac work.

**Mechanism of increased cardiovascular risk.** The combination of augmented systolic waves, which increase ventricular systolic work, and faster heart rates, which shorten diastole, operates to increase the need and decrease the opportunity for myocardial perfusion. This scenario has been well summarized by the DPTI/SPTI index (38,39,43,47). Although this index does not provide a complete description of myocardial vulnerability (39), it does offer a means of approximating ventricular requirements and the potential for receiving it (in the absence of coronary obstruction) in a single value. In the present study, body height correlated significantly with this index, reflecting again the relations among stature, systolic pressure augmentation and heart rate.

Carotid compliance, as expected, also correlated with body height in both groups, indicating increased stiffness in people of short stature. The correlation coefficient was lower than that...
observed with other variables, owing to the concomitant influence of age and SBP as shown by stepwise multiple regression analysis.

The correlations among body height and the circulatory variables already described showed no differences between women and men. This finding implies that the relations are physiologic and not gender induced. If true, the intrinsic shorter stature of women should make them, other factors being equal, more vulnerable to CHD than men. This was found to be the case for women and their risk of myocardial infarction in the Framingham study (12).

The findings not only apply to men and women, but also to normal subjects, hypertensive patients and patients with ESRD. Although the correlations cannot be strictly applied to other patient groups, the principles which underlie the findings are likely to apply widely.

Conclusions. These data show body height to be a determinant of the timing and magnitude of reflected arterial pulses. These reflections alter the shape of the aortic pulse, which increases left ventricular pulsatile work and left ventricular mass and alters the myocardial need and the potential supply relations. The risks imposed by these changes are even greater in patients with ESRD, who have an increased predisposition for CHD when compared with subjects with normal renal function. The influence of body height on the risks of CHD, due to reflected waves, are exerted in many ways: by increasing heart rate, by stiffening the aorta in late systole and by reducing aortic diastolic pressure and duration. The correlations between body height and the various variables which relate to ventricular vulnerability are small but significant. This is not surprising, because these factors are interrelated, as illustrated by the stepwise multiple regression calculations. Accounting for these interrelations demonstrates how body height operates as a risk factor. Multiple regression analysis weakens the correlations between body height alone and individual variables, but does not eliminate them. This suggests that body height itself survives as a risk factor for CHD, perhaps not a powerful one, but a risk factor nonetheless.

References


