Height and Heart Disease

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Height has a relationship with a number of medical conditions, including heart disease. Atrial fibrillation has been observed to be more common in taller individuals. Marfan syndrome, with its high prevalence of mitral valve disease and abnormalities of the aorta, is associated with increased height. Mitral valve prolapse in patients without Marfan syndrome may be more common in taller people. Conversely, congestive heart failure, coronary artery disease, and possibly aortic valve calcification are less prevalent with increasing height. The relationship between height and health will be of increasing importance as the population grows taller.

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KEY WORDS

Heart disease • Height • Atrial fibrillation • Marfan syndrome • Mitral valve prolapse • Congestive heart failure • Coronary artery disease

The height of our population is increasing. According to the National Health Nutrition Examination Survey (NHANES), the mean adult height in the United States increased between the 1960s and 2002 by approximately 1 inch.[1] A study of young adult men in 15 Western European countries, from the 1870s to the 1970s, found that the average height increased by more than 1 cm per decade.[2] Tall stature has been reported to be related, either negatively or positively, with a number of cardiovascular disorders. This article reviews common heart problems found in adult patients and summarizes the relationship of these problems with height.

Methods

In order to look for associations, we performed literature searches combining key terms related to height with those of common cardiovascular problems, using Scopus, Google Scholar, and MEDLINE via PubMed. We describe the relationships of height to atrial fibrillation (AF), congestive heart failure, aorta and valve disease, and coronary artery disease (CAD).

Height and AF

Tall stature has been identified as a statistically significant risk factor for the development of AF in various populations and settings, including patients with lone AF and patients with underlying heart disease.

Patients with lone AF presenting to an emergency department in Spain were found to be taller than matched control subjects, with average heights of 168 ± 8 cm versus 165 ± 5 cm.[3] In Japan, screening of

5795 patients without heart disease, diabetes, or hypertension demonstrated a 12.2% overall risk of developing AF. From the lowest to the highest height tertiles, the relative risk for height was 2.07 (1.70-2.52).[4] In a Swedish Primary Prevention Study of 6903 men, 18.2% developed AF over a 34.3-year prospective follow-up period, with the highest quartile for height having double the risk.[5] Data from a random sample of 13,391 people living in Copenhagen demonstrated that the development of AF was independently associated with increased height. In men the odds ratio (OR) was 1.4 and in women the OR was 1.6.[6] A population-based control study in Washington State included 425 subjects with new-onset AF and 707 control subjects. In this study, height was strongly associated with AF, with an adjusted OR of 1.38, comparing the fourth quartile to the first.[7]

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Taller patients with left ventricular dysfunction also have a higher incidence of AF. In the National Registry to Advance Heart Health (ADVANCENT) study, a prospective observational study of 25,268 patients with systolic dysfunction, the prevalence of AF was 24% in the shortest quartile and 31.7% in the tallest. A 16-cm (6.2 in) increase in height translated to a 50% increase in AF. This relationship was seen in men and women, and was present in all patterns of AF.[8] In the Physicians' Health Study, which included 22,042 men (mostly white physicians), the risk of heart failure was found to be inversely associated with height; however, the risk of AF was higher (0.87% in the lowest height quartile and 2.09% in the highest). Interesting, white race was also found to be correlated with a higher AF incidence.[9] In most of these studies, increased weight or body mass index (BMI) was also statistically correlated with AF; however, height remained an independent risk factor.[4-8]

Left Atrial Size, AF, and Height

Relationships have been documented between left atrial size and AF, and between left atrial size and height. In the Framingham Heart Study, in a sample of 4957 subjects, 166 developed AF over 7.7 years. A 4.4-fold risk was seen in those with the highest quintile of left atrial dimension as compared with the lowest. There was also a positive relationship between left atrial size and height.[10] In another publication from the Framingham Heart Study group, a 38-year follow-up showed a 39% increase in the risk of AF with a 5-mm increase in left atrial size.[11]

Echocardiographic studies show a clear correlation between left atrial size and the propensity to develop AF.

A study including 1655 older men and women in Olmsted County, Minnesota, showed that a 30% larger left atrium by volume calculation was associated with a 43% greater risk of AF.[12] A later publication by Pritchett and colleagues[13] described a random sample of 2042 residents who had Doppler echocardiographic evaluation of left atrial size, and the measurements correlated with the prevalence of AF and with height.

Vaziri and colleagues looked at echocardiographic predictors of AF in 1924 subjects. Left atrial size was associated with the development of AF over the 7.2-year follow-up, with a hazard ratio of 1.39 for each 5-mm increase.[14] The Cardiovascular Health Study followed 5201 older adults. Echocardiographically determined left atrial size was independently and strongly associated with the incidence of AF over 3 years. The relative risk for AF related to left atrial size in all subjects was 1.74. Importantly, the left atrial size was assessed prospectively at baseline, suggesting that the enlarged left atrium was a cause rather than a consequence of AF.[15] In the Spanish study by Mont and associates,[3] echocardiograms performed 2 weeks following the return of sinus rhythm demonstrated that left atrial diameter was linearly associated with AF. A subset of the ADVANCENT registry, in which 362 patients had echocardiograms, showed a strong association between left atrial diameter and height, in both men and women ($P < .005$ and $P = .05$, respectively).[8]

Mechanisms

Echocardiographic studies show a clear correlation between left atrial size and the propensity to develop AF. Observations from nonhuman species reinforce these observations. Large animals with correspondingly large left atria have been observed to develop AF, whereas very small animals do not share this predisposition. AF is rarely observed in small animals such as rats, rabbits, cats, or dogs weighing < 20 kg, whereas it is well known to occur in large dogs and horses.[16,17] In a canine study, left atrial dimensions were larger in dogs that developed AF. In an atrial pacing study, a greater persistence of AF was seen in animals having large left atria compared with animals with smaller atria. It has been shown that experimentally induced AF ceases spontaneously in small

animals—presumably because their atria do not have a large enough critical mass to sustain the arrhythmia.[16] Another canine atrial model demonstrated that substrate size is a determinant of maintenance of fibrillatory activity.[18]

Aorta and Valvular Disease

Diseases of the aorta and of cardiac valves have been shown to have a correlation with patient height. Normal aortic root size should be defined relative to patient height. As part of the Framingham Heart Study, 4001 patients underwent M-mode echocardiography to determine aortic root size. A 10-cm increase in height was associated with a 0.24-mm and 0.38-mm increase in aortic root size for men and women, respectively.[19]

Although many genetic disorders are associated with tall or short stature, we limit our discussion to Marfan syndrome. Marfan syndrome is an autosomal dominant connective tissue disorder with a variety of cardiovascular manifestations, including aortic root dilation with a predisposition for dissection, mitral valve prolapse (MVP), and pulmonary artery dilation.[20] Patients with Marfan syndrome are taller than the general population, with an average height of 184.3 cm, whereas the average height of adult American patients as noted by NHANES data is 162.2 cm for women and 176.3 cm for men.[21,22] As with aortic diseases, patients with Marfan syndrome appear to have a higher prevalence of MVP—estimated to be nearly 30%. The prevalence of MVP in this population of generally taller patients is higher than the general population.[22]

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It is a common perception that tall people are more likely to have MVP, but evidence supporting this notion is mixed.[23] A cohort study of 4967 patients from the Framingham Study revealed a 5% prevalence of MVP with no statistical difference in height between those with and without the disorder. For any given weight, however, the patients with MVP were 1 cm taller than those without, suggesting different anthropomorphic builds.[24] A case-control study of women with MVP reiterated the concept that MVP is associated with distinctive anthropomorphic characteristics. Although women with MVP were not statistically taller, they did have narrower anteroposterior chest diameters and longer arm span-to-height ratios than those without MVP.[25] Examination of a cohort of 4136 patients in the Coronary Artery Risk Development in Young Adults Study (CARDIA) revealed that patients with MVP were, on average, 3 cm taller and had a lower BMI and leaner habitus than those without the findings.[26] Another study of offspring from the Framingham Heart Study found that MVP patients had a BMI average of nearly 25 kg/m^2—2 points lower than those without prolapse.[27] Additional studies suggest a similar conclusion that MVP patients are leaner, with lower BMIs than control groups.[28-30]

Limited data also suggest that height may inversely correlate with the development of aortic valve calcification. Echocardiographic data from the Cardiovascular Health Study demonstrated this finding in a group of patients over age 65. The authors speculate that such a trend may have a link to osteoporosis and loss of height, or that less shear stress related to the height of the vasculature may play a role.[31]

Heart Failure

Until recently, there had been few data to suggest a relationship between height and the development of heart failure. Recent research has shown a potential protective effect of tall stature.

In a cohort of 22,042 patients from the Physicians' Health Study, survey information obtained included self-reported height. Researchers also surveyed for the development of a heart failure diagnosis. Findings were statistically significant for a 24% reduction in the risk of heart failure when comparing the tallest quartile of patients with the shortest quartile, even when adjusted for AF, left ventricular hypertrophy, valvular heart disease, and coronary artery bypass surgery.[9]

The authors speculate on a variety of mechanisms for such a reduction, including the effects of gravity on the cardiovascular system as well as the inverse relationship between pulse pressure and height.[32,33] The effects of gravity are thought to reduce preload and peripheral vascular resistance via expansion of the vascular bed leading to decreased preload and increased cardiac output. Gravity may decrease the work of the heart, lessening the progression of ventricular hypertrophy. Similarly, a reduction in the pulse pressure with increased height may also lead to less hypertrophy as the arterial compliance increases with height.[9]

Coronary Disease

Numerous studies demonstrate the apparent inverse relationship between height and the risk of CAD. The majority of these studies utilize large cohort and case-control

populations to extrapolate this relationship. Although height is not a modifiable risk factor, it might be useful in estimating cardiovascular risk.

A single-center study of 1046 men demonstrated a greater propensity for patients < 64.7 inches in height to have stenoses > 50% in the proximal and mid segments of the coronary arteries. Shorter patients were also more likely to have three-vessel disease.[34] The Physicians' Health Study followed 22,071 physicians (all men) aged 40 to 84 years over a 5-year period. The tallest men (≥ 73 in or 185.4 cm) were at a 35% lower risk of myocardial infarction (MI) when compared with the shortest subjects (< 67 in or 170.2 cm) even after adjusting for various risk factors. A 2% to 3% decline in risk of MI was noted for each inch of additional height.[35] A cohort of 17,139 British men from the Whitehall Study was followed over 33 years. Data demonstrated an inverse relationship between height and mortality from cardiovascular disease, with a hazard ratio of 0.89 per 15-cm increase in height.[36] Furthermore, a population of 1393 World War II Army soldiers (all men) with known CAD was retrospectively evaluated for risk factors associated with angina pectoris, coronary insufficiency, MI, and death from heart disease. When compared with age-matched control subjects, height was found to be negatively associated with all evaluated endpoints.[37]

Many of the aforementioned data are driven by studies performed in white men; however, most studies in women show similar findings. One hospital-based study from the 1980s studied 910 women with a first nonfatal MI in comparison to 1140 control subjects. Women 69 inches tall had a relative risk estimate for having MI of 0.5 (95% confidence interval [CI], 0.2-0.8). For women shorter than 59 inches, that index did not reach statistical significance at 1.5 (95% CI, 0.9-2.6). Results were sustained when corrected for age, BMI, and educational status.[38] A second cross-sectional analysis of 4286 British women reported that, for every standard deviation of height (6.4 cm) above the mean, the OR of having coronary disease was 0.79 when corrected for age, forced expiratory volume in 1 second (FEV_1), and smoking status.[39]

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A cohort of the large Nurses' Health Study further supports the association in women. When compared with women < 61 inches in height, the OR for developing nonfatal or fatal MI, angina, or the need for coronary revascularization was 0.73 (95% CI, 0.65-0.83) for women ≥ 67 inches in height.[40] Moreover, an examination of Swedish women with known coronary events suggested that women < 160 cm in height had a 2.1-fold increased rate of developing adverse cardiac events, including recurrent infarction, revascularization, and death, when compared with women > 165 cm in height.[41]

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A 2010 meta-analysis examined 22 studies encompassing subjects with diverse ages, ethnic groups, sex, and socioeconomic factors. The authors concluded that adults of short stature had a roughly 50% greater morbidity and mortality from CAD than those of tall stature.[42]

A retrospective study of both black men and women in a Chicago, Illinois, hospital examined 1682 patients who underwent coronary angiography over a 4-year period. The data demonstrated a significantly increased prevalence of CAD in men with shorter statures. This was true even after adjustment for other risk factors. However, the data were not significant for women. Of note, socioeconomic factors probably did not influence the findings because the study population in this inner-city hospital was relatively homogenous from a socioeconomic perspective.[43]

Nevertheless, there are conflicting data. The NHANES I Epidemiologic Follow-Up Study of the 1970s suggested no association between height and MI, coronary heart disease, overall mortality, and mortality from cardiovascular disease when samples were corrected for age and years of education.[44] Others argue that rather than having a detrimental impact, short stature benefits longevity.[45]

Mechanisms of Inverse Relationship Between Height and CAD

No single factor for why height and CAD have an inverse relationship has been identified. Rather, there are a number of possible explanations. These include anatomic, biochemical, and hormonal factors.

Factors of Birth. Size at birth may play a role in the development of CAD. A retrospective study of

4775 Scandinavian patients demonstrated an inverse relationship between birth length and CAD, particularly among men. This effect persisted even if the men grew to be taller adults.[46] A cohort study of Finnish women born in the 1920s suggests that CAD is associated with a low birth weight, but a greater risk is seen in those who were of shorter length at birth. The risk was more pronounced in those women who grew to be of normal height during adolescence, indicating a "catch up" growth effect, suggesting stunted uterine growth with subsequent recovery.[47]

Vessel Diameter. One explanation could be that shorter people have small diameter coronary arteries and thus have the potential for obstruction with less disease burden—a postulate with some evidence in terms of coronary artery bypass outcomes.[48] Unfortunately, strong data relating height to coronary diameter are lacking.[35,49,50]

Lung Volumes. The inverse relationship between CAD and height may be due to variation in lung volumes.[51,52] There is evidence for this relationship particularly among elderly women.[53] There is further suggestion that FEV_1 alone may have a bigger role in mortality from CAD than height, suggesting that height might not be the true risk factor.[44]

Lipids. Lipid profiles also appear to be related to height, at least in the adolescent population. A study of 1500 adolescents in Taiwan revealed that a shorter body height was associated with higher total cholesterol, low-density lipoprotein cholesterol, and apolipoprotein B concentrations.[54] A group of Japanese school-aged children examined over 3 years demonstrated a similar negative relationship between total cholesterol and height in both boys and girls. Those who grew taller had lower total cholesterol.[55] It is uncertain if this benefit is maintained into adulthood.

Growth Hormone. Growth hormone deficiency may lead to a shorter stature as well as increased risk for CAD.[56] A large, prospective, case-control study demonstrated an increased risk of developing CAD in subjects found to have low circulating levels of insulin-like growth factor 1 (IGF-1), whereas another study demonstrated an increased presence of coronary calcification on computed tomography imaging in patients with decreased IGF-1 levels.[57,58] Epidemiologic studies have shown similar results.[59] Moreover, growth hormone supplementation in children with Prader-Willi syndrome improves lipid profiles by decreasing low-density lipoprotein and increasing high-density lipoprotein cholesterol.[60]

Conclusions

Some cardiac disorders have been shown to be more common with increased height. Tall individuals have a higher incidence of developing AF, which seems to be related to the increased left atrial size seen in, but not limited to, tall people. Marfan syndrome, which includes increased height as one of its primary features, is associated with aorta and valve disease as part of the genetic syndrome. MVP may be more common in taller individuals, although this relationship is less definite in patients without Marfan syndrome.

The news is not all bad for tall people, however, as they appear to have a lower incidence of congestive heart failure, CAD, and aortic valve calcification. For congestive heart failure the mechanism may relate to effects of pulse pressure and gravity, with decreasing preload and pulse pressure as height increases. For CAD, size at birth, vessel diameter, lung volume, and hormonal issues have all been hypothesized to have an effect. It is particularly difficult to isolate the effects of height on CAD because of the presence of other risk factors. It should be noted that most of the studies had intrinsic limitations because patients were often of a similar socioeconomic class, taller patients were often more overweight, and, in some studies, height was self-reported.

Despite limitations in the data, the implications of these positive and negative associations of height with various forms of heart disease are significant. In addition to considering traditional risk factors, health care providers should also be aware of these correlations between tall stature and heart problems. With a greater awareness of the relationship between height and heart disease, and with the increasing height of our population, further studies should be designed to confirm these correlations and determine whether new relationships can be identified. ■

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MAIN POINTS

- Height has a relationship to a number of medical conditions, including heart disease. The relationship between height and health will be of increasing importance as the population grows taller.
- Atrial fibrillation (AF) has been observed to be more common in taller individuals, and is associated with increased left atrial size.
- Taller patients with left ventricular dysfunction also have a higher incidence of AF. In one prospective observational study of 25,268 patients with systolic dysfunction, the prevalence of AF was 24% in the shortest quartile and 31.7% in the tallest. A 16-cm (6.2 in) increase in height translated to a 50% increase in AF.
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