

Height Loss in Older Men

Associations With Total Mortality and Incidence of Cardiovascular Disease

S. Goya Wannamethee, PhD; A. Gerald Shaper, FRCP; Lucy Lennon, MSc; Peter H. Whincup, FRCP, PhD

Background: Height declines with age, but the impact of height loss on health outcomes has been little studied. We examined the relationships between height loss over 20 years (starting at middle age) and subsequent total mortality and incidence of coronary heart disease and stroke in older men.

Methods: A prospective study was performed on 4213 men whose height was measured between the ages of 40 and 59 years and again 20 years later between the ages of 60 and 79 years. The men were then followed up for a mean period of 6 years, during which 760 deaths occurred.

Results: Height loss correlated significantly with initial age ($r=0.20$) and weight loss ($r=0.20$). Total mortality risk was higher in men with a height loss of 3 cm or more than in men with a height loss of less than 1 cm (age-adjusted relative risk [RR], 1.64; 95% confidence inter-

val [CI], 1.33-2.03). The excess deaths were largely attributable to cardiovascular and respiratory conditions and other causes but not to cancer. Adjustment for age, established cardiovascular risk factors, lung function, pre-existing cardiovascular disease, albumin concentration, self-reported poor or fair health, and weight loss had a modest impact on the increased risk of total mortality (RR, 1.45; 95% CI, 1.15-1.82). The risk of major coronary heart disease events was increased only in men with a height loss of 3 cm or more even after adjustment (adjusted RR, 1.42; 95% CI, 1.02-1.98; ≥ 3.0 cm vs < 3.0 cm); no association was seen with stroke risk.

Conclusion: Marked height loss (≥ 3 cm) in older men is independently associated with an increased risk of all-cause mortality and coronary heart disease.

Arch Intern Med. 2006;166:2546-2552

PEOPLE BECOME SHORTER AS they get older, with the average magnitude of decline in height being greater in women than in men.^{1,2} Height loss is related to aging changes in bone, muscles, and joints. While a minor degree of height loss is usual and unlikely to be associated with any health problems, significant height loss may indicate osteoporosis.³⁻⁶ The resulting height loss can affect the normal functioning of the respiratory and gastrointestinal systems,⁴ which in turn may lead to early satiety, poor nutritional status, and weight loss.⁴ Height loss also appears to be related to sarcopenia,⁷ which is defined as the loss of skeletal muscle mass and strength with aging and is associated with weight loss⁸⁻¹¹ and increased mortality.¹²⁻¹⁴ Furthermore, there is evidence to suggest that some pathophysiologic mechanisms, such as dyslipidemia, oxidative stress, inflammation, hyperhomocystinemia, and hypertension, may be involved in both osteoporosis and cardiovascular disease (CVD).^{15,16} Although age-related decline in height is a common observation, its possible associa-

tion with cardiovascular health outcomes and all-cause mortality in men has not been studied, to our knowledge. We examined the relationship between height loss over a 20-year period (starting at middle age) and outcome in terms of indicators of ill health, incidence of CVD (coronary heart disease [CHD] and stroke), and total mortality in elderly men.

METHODS

The British Regional Heart Study is a prospective study of CVD that involved 7735 men aged 40 to 59 years who were selected from the age-sex registers of 1 general practice in each of 24 British towns and who were screened between 1978 and 1980.¹⁷ From 1998 to 2000, after a 20-year interval, all surviving men, now aged 60 to 79 years, were invited for a follow-up examination. Ethics approval was provided by all relevant local research ethics committees. All men provided informed written consent to the investigation, which was carried out in accordance with the Declaration of Helsinki. All men completed a questionnaire (Q), providing details of their medical history and lifestyle behavior. The men were then asked

Author Affiliations:

Department of Primary Care and Population Sciences, Royal Free and University College Medical School (Drs Wannamethee and Shaper and Ms Lennon), and Division of Community Health Sciences, St George's, University of London (Dr Whincup), London, England.

to fast for a minimum of 6 hours, during which they were instructed to drink only water and to attend for measurement at a specified time between 8 AM and 6 PM. A fasting blood sample was collected (Monovette; Sarstedt, Nurnbrecht, Germany), with 4252 men (77% of survivors) attending for examination.

ANTHROPOMETRIC MEASURES

Height and weight were measured both at baseline screening (Q1) and at the 20-year reexamination (Q20), with the subjects in light clothing without shoes. On both occasions, height was measured to the last complete 0.1 cm using the supported stretch technique with the same stadiometer (Harpending; Holtrain Ltd, Crymmych, Wales); weight was measured to the last complete 0.1 kg using externally calibrated digital electronic scales (Soehnle, Murrhardt, Germany). Body mass index (BMI [weight in kilograms divided by height in meters squared]) was calculated for each man. Height could not be measured in 17 of the 4252 men who attended reexamination. We excluded a further 22 men whose height appeared to have increased by more than 5 cm, as this increase in height is biologically implausible and may reflect invalid readings. Valid measures on height change were thus available in 4213 men. Fat-free mass was calculated using bioelectric impedance analysis (Bodystat 500; Bodystat Ltd, Douglas, Isle of Man),¹⁸ applying the equation of Deurenberg et al,¹⁹ which has been validated in an elderly population. Measured weight loss was defined as a loss of 4% in weight.²⁰ The men were also asked whether they had lost weight in the 3 years before reexamination (1998-2000) and whether this weight loss was intentional or unintentional.

CARDIOVASCULAR RISK FACTORS

Details of measurement and classification methods for smoking status, physical activity, social class, alcohol intake, blood pressure, blood lipid levels, and lung function (forced expiratory volume in 1 second [FEV₁] and forced vital capacity [FVC] in this cohort have been described elsewhere^{18,21,22}; FEV₁ and FVC were height standardized to the average height [1.73 m] in the study).²³ Possible chronic obstructive pulmonary disease (COPD) was defined on the basis of an FEV₁-FVC ratio of less than 70%.²⁴

INDICATORS OF HEALTH STATUS AT REEXAMINATION

The men were asked to describe their present health status as excellent, good, fair, or poor. They were also asked whether a physician had ever told them that they had angina, myocardial infarction (heart attack or coronary thrombosis), stroke, diabetes, cancer, or a number of other cardiovascular conditions. Patient recall of a physician diagnosis of CVD has been shown to be a valid measure of recording diseases in this study population.^{25,26} The κ statistic comparing record review with patient recall of CHD was 0.82.²⁵ The men were asked about regular treatment and were required to bring their medication to the examination session. The medications were classified according to the British National Formulary codes.²⁷ The presence of musculoskeletal and joint diseases was inferred by the use of any drugs for the treatment of rheumatic diseases, neuromuscular disorder, and soft tissue inflammation (British National Formulary codes 10.1-10.3).

FOLLOW-UP

All subjects were followed up from the initial examination (1978-1980) to December 2005 for all-cause mortality and to December 2004 for cardiovascular morbidity; follow-up was com-

pleted for 99% of the cohort.²⁸ In the present analyses, all-cause mortality was based on follow-up from rescreening during 1998 to 2000, when the men were aged 60 to 79 years, with a mean follow-up period of 6 years (range, 5-7 years), and cardiovascular morbidity was based on a mean follow-up of 5 years (range, 4-6 years). Information on death was collected through the established "tagging" procedures provided by the National Health Service registers. Fatal stroke episodes were defined as those coded 430 to 438 on the death certificate according to the *International Classification of Diseases, Ninth Revision (ICD-9)*. Nonfatal stroke events were defined as those that produced a neurologic deficit that was present for more than 24 hours. Fatal CHD events were defined as death with CHD (ICD codes 410-414) as the underlying code. A nonfatal myocardial infarction was diagnosed according to World Health Organization criteria.²⁹ Cardiovascular deaths included all those with ICD codes 401 to 459. Evidence regarding nonfatal CHD and stroke was obtained from ongoing reports from general practitioners, from biennial reviews of the patients' practice records (including hospital and clinic correspondence) throughout the study period, and from repeated personal questionnaires that were distributed to surviving subjects after the initial examination.

STATISTICAL ANALYSIS

The Cox proportional hazards model was used to assess the multivariate-adjusted relative risk (RR) for each height loss group compared with the reference group (height loss <1 cm). In the adjustment, smoking (never or long-term ex-smokers [>15 years], recent ex-smokers [<15 years], and current smokers), social class (7 groups), physical activity (4 groups), alcohol intake (5 groups), preexisting CHD (yes/no), stroke (yes/no), diabetes (yes/no), BMI (<25.0 , 25.0-29.9, and ≥ 30.0), poor/fair health (yes/no), musculoskeletal problems (yes/no), and weight loss (yes/no) were fitted as categorical variables. The biologic factors (serum high-density lipoprotein cholesterol, serum total cholesterol, blood pressure, serum albumin, and FEV₁) were fitted as continuous variables. Tests for trend were carried out across the height-loss groups. Analysis of covariance was used to obtain age-adjusted mean levels of the biologic factors for the 4 height-loss groups. Direct standardization was used to obtain age-adjusted rates for the indicators of ill health. Logistic regression was used to obtain the age-adjusted odds ratio for prevalent disease.

RESULTS

In the 4213 men with valid measure of height change, the mean height loss from Q1 to Q20 was 1.67 cm (SD, 1.79 cm). **Figure 1** shows the distribution of height loss. Height loss was significantly correlated with age ($r=0.20$; $P<.001$). Mean height loss increased with increasing age from 1.2 to 1.6 cm to 2.0 and 2.3 cm in men initially aged 40 to 44 years, 45 to 49 years, 50 to 54 years, and 55 to 59 years, respectively. During the mean follow-up period of 5 years, there were 760 deaths from all causes.

HEIGHT LOSS AND RISK FACTORS

The men were divided into 4 groups on the basis of height change (<1.0 cm, 1.0-1.9 cm, 2.0-2.9 cm, and ≥ 3.0 cm). **Table 1** shows the mean anthropometric factors, lifestyle characteristics, and biologic risk factors by the 4 height-loss groups. Increasing height loss was inversely associated with adiposity measures (BMI and waist circumference) and fat-free mass (muscle mass) and was

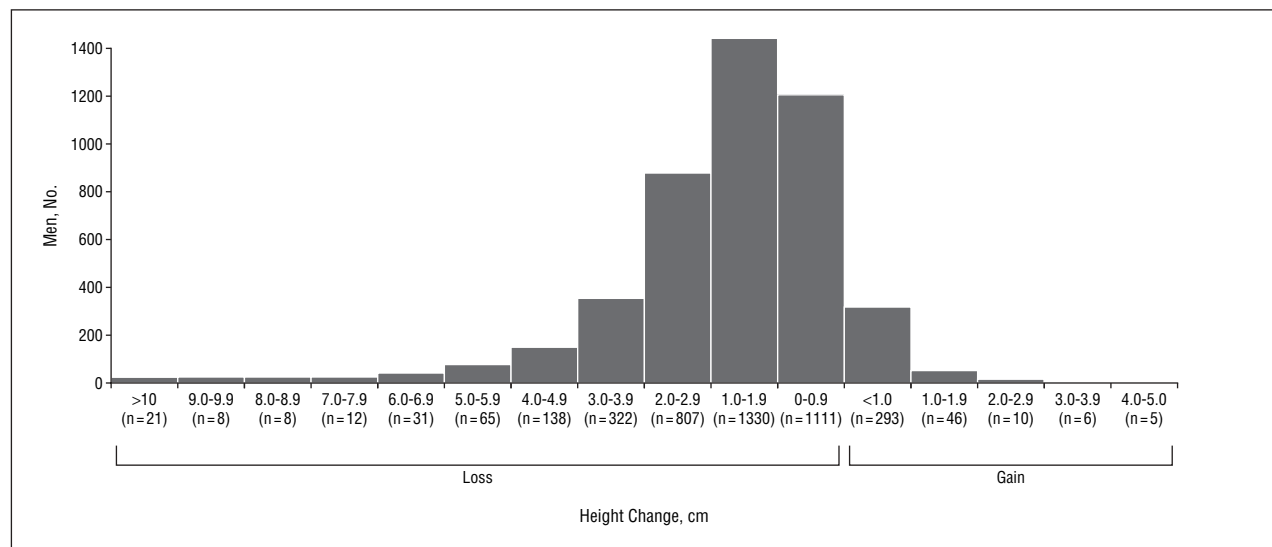


Figure 1. Distribution of height change.

Table 1. Height Loss and Anthropometric, Lifestyle, and Biologic Factors*

Variable	Height Loss, cm				P Value for Trend Across Groups
	0	1.0-1.9	2.0-2.9	≥3.0	
Men, No. (%)	1471 (34.0)	1330 (32.0)	807 (19.4)	605 (14.6)	
Age, y	67.0	68.7	70.0	71.1	<.001
Anthropometric factors					
Initial height, cm	173.4	174.1	174.3	174.5	<.001
Height Q20, cm	173.2	172.6	171.9	169.9	<.001
BMI Q20	27.4	26.8	26.6	26.6	<.001
WC Q20, cm	98.5	97.0	96.8	95.6	<.001
FFM Q20, kg	52.7	52.1	51.6	50.6	<.001
Weight change over 20 y, kg	+5.2	+3.2	+1.8	-0.2	<.001
Reported weight loss over previous 3 y, %	10.8	15.5	22.1	31.7	<.001
Proportion of reported unintentional weight loss, %	45.9	44.3	56.3	57.9	<.001
Lifestyle factors Q20, %					
Manual workers	54.7	54.8	50.1	53.4	.14
Current smokers	12.6	12.5	13.0	12.9	.72
Never smokers	28.5	30.5	29.0	27.4	.12
Inactive	31.9	33.4	36.4	42.8	<.001
Nondrinkers	20.9	20.4	22.9	29.2	<.001
Biologic risk factors Q20					
FEV ₁ , L	2.62	2.63	2.55	2.51	<.001
FVC, L	3.40	3.42	3.38	3.36	.24
FEV ₁ -FVC ratio, %	77.7	77.3	76.3	75.1	<.001
Cholesterol, mmol/L	6.0	6.1	6.0	5.8	<.001
Systolic blood pressure, mm Hg	148.8	149.4	149.8	148.2	.46
HDL cholesterol, mmol/L	1.29	1.32	1.35	1.37	<.001
Albumin, g/L	44.3	44.3	44.0	43.6	<.001

Abbreviations: BMI (calculated as weight in kilograms divided by height in meters squared); FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity; FFM, fat-free mass; HDL, high-density lipoprotein; Q20, at 20-year reexamination (1998-2000); WC, waist circumference.

Conventional unit conversion factors: To convert cholesterol and HDL cholesterol to milligrams per deciliter, divide by 0.0259.

*All numbers are expressed as means unless otherwise stated.

positively associated with weight loss, physical inactivity, and nondrinking status at the 20-year follow-up examination. No significant association was seen with smoking or social class. Height loss was inversely related to the FEV₁-FVC ratio (an indicator of chronic obstructive pulmonary function) and total serum cholesterol and serum albumin levels and was positively associated with high-density lipoprotein cholesterol levels.

HEIGHT LOSS AND INDICATORS OF HEALTH STATUS AT Q20

Age-adjusted prevalence of poor/fair health, mobility problems, musculoskeletal and joint problems, and COPD (as measured by the FEV₁-FVC ratio <70%) tended to increase with increasing height loss, with risk substantially increased in those with a height loss of 3.0 cm or more

Table 2. Height Loss and Age-Adjusted Prevalence of Indicators of Ill Health

Indicators of Ill Health	Height Loss, cm				≥3.0 vs <1.0, Adjusted OR (95% CI)
	<1.0	1.0-1.9	2.0-2.9	≥3.0	
Poor/fair health	25.1	23.3	25.8	31.1	1.34 (1.08-1.66)
Mobility problem	29.4	29.8	33.3	44.2	1.91 (1.56-2.34)
Musculoskeletal and joint problems	10.4	11.9	13.9	17.8	1.86 (1.41-2.45)
All CVD	21.0	22.6	22.8	25.3	1.02 (0.81-1.28)
MI	9.8	11.3	9.8	12.6	1.32 (0.97-1.78)
Angina	15.6	14.2	12.9	14.5	0.91 (0.69-1.19)
Stroke	5.2	5.6	5.6	6.2	1.22 (0.81-1.85)
Diabetes	8.3	6.4	4.8	6.0	0.70 (0.47-1.03)
COPD (FEV ₁ -FVC ratio <70%)	19.6	22.5	25.3	27.6	1.33 (1.06-1.68)
Cancer	5.3	6.8	5.1	6.4	1.23 (0.82-1.84)

Abbreviations: CI, confidence interval; COPD, chronic obstructive pulmonary disease; CVD, cardiovascular disease; FEV₁, forced expiratory volume in 1 second; FVC, forced vital capacity; MI, myocardial infarction; OR, odds ratio.

(Table 2). Little association was seen between height loss and CVD, type 2 diabetes, and prevalent cancer.

MORTALITY

Figure 2 shows age-adjusted mortality rates by the 4 height-loss groups. All-cause mortality increased with increasing height and was substantially increased in men with a height loss of 3.0 cm or more. The excess deaths were largely attributable to CVD and respiratory and other noncardiovascular, noncancer causes (Figure 2 and Table 3). No significant excess death was seen for cancer causes (age-adjusted RR, 1.28; 95% confidence interval [CI], 0.88-1.85). Initial height did not predict total mortality followed up from the 20-year reexamination (Q20).

A height loss of 3 cm or more was associated with significantly increased mortality even after adjustment for potential confounders and cardiovascular risk factors, including age, social class, smoking, alcohol intake, physical activity, BMI, preexisting CHD, stroke, diabetes, systolic blood pressure, serum total cholesterol, high-density lipoprotein cholesterol, and FEV₁-FVC ratio (Table 3). Further adjustment for poor/fair health, presence of musculoskeletal problems, and serum albumin levels (a marker of poor nutrition) attenuated the association slightly, but the increased risk seen for CVD mortality and other noncardiovascular, noncancer deaths remained significant ($P = .03$ and $P = .001$, respectively). The association between height loss and respiratory death was greatly attenuated after these adjustments, largely because of the lower FEV₁-FVC ratio.

HEIGHT LOSS AND WEIGHT LOSS OVER 20 YEARS

Height loss was significantly correlated with 20-year weight loss (Q1-Q20) ($r = 0.20$; $P < .001$). Further adjustment for weight loss attenuated the increased mortality risks, but the associations with total mortality ($P = .002$) and other noncardiovascular, noncancer deaths ($P = .002$) remained significant (Table 3). However, the

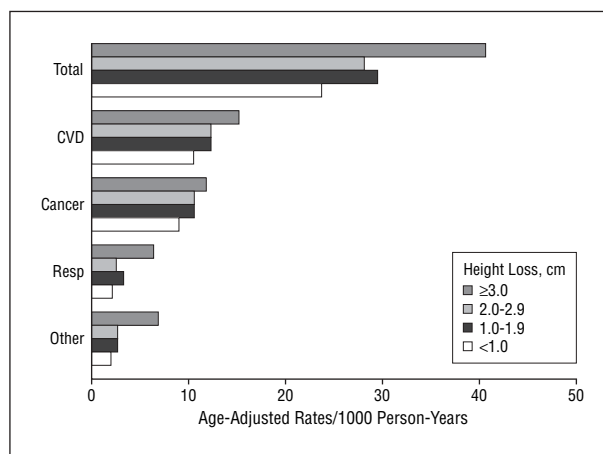


Figure 2. Height loss and age-adjusted cause-specific mortality rates per 1000 person-years. CVD indicates cardiovascular disease; Resp, respiratory disease; and Other, other causes.

increased risk of CVD mortality became of marginal significance.

EXCLUSION OF MEN WITH SUBSTANTIAL HEIGHT LOSS

The significantly increased risk of all-cause mortality in men with a height loss of 3.0 cm or more was observed even after exclusion of men with a height loss of 4.0 cm or more ($n = 283$) (adjusted RR, 1.48; 95% CI, 1.12-1.95). Thus, the increased mortality risk was already seen in men with a height loss in the range of 3.0 to 4.0 cm and was not solely attributable to extreme height loss. Indeed, we observed no further increase in mortality risk in those with a height loss of 4.0 cm or more after adjustment.

INCIDENCE OF CVD

A height loss of 3.0 cm or more was associated with a significant increase in risk of major incident CHD events after adjustment for preexisting CVD and CVD risk factors ($P = .03$) (Table 4). Further adjustment for albumin,

Table 3. Height Loss and Adjusted Relative Risk (RR) of Cause-Specific Mortality*

Variable	Height Loss, cm				P Value†	P Value for Trend Across Groups
	<1.0 (n = 1471)	1.0-1.9 (n = 244)	2.0-2.9 (n = 807)	≥3.0 (n = 605)		
Men, No.	1471	1330	807	605		
All cause mortality (n = 760)						
Cases, No.	187	244	156	173		
Age-adjusted RR	1.00	1.20 (0.99-1.46)	1.14 (0.92-1.42)	1.64 (1.33-2.03)	<.001	<.001
Adjusted RR‡	1.00	1.23 (1.00-1.51)	1.14 (0.91-1.44)	1.60 (1.27-2.06)	<.001	<.001
Adjusted RR§	1.00	1.26 (1.03-1.54)	1.12 (0.89-1.41)	1.53 (1.25-1.97)	<.001	.002
Adjusted RR	1.00	1.25 (1.02-1.53)	1.12 (0.89-1.41)	1.45 (1.15-1.82)	.002	.01
CVD death (n = 318)						
Cases, No.	81	102	69	66		
Age-adjusted RR	1.00	1.14 (0.85-1.53)	1.13 (0.82-1.57)	1.39 (1.00-1.95)	.05	.08
Adjusted RR‡	1.00	1.21 (0.88-1.65)	1.21 (0.85-1.72)	1.53 (1.07-2.19)	.02	.03
Adjusted RR§	1.00	1.22 (0.89-1.67)	1.19 (0.84-1.69)	1.48 (1.04-2.13)	.03	.04
Adjusted RR	1.00	1.22 (0.87-1.67)	1.18 (0.83-1.67)	1.39 (0.97-1.82)	.07	.11
Respiratory death (n = 87)						
Cases, No.	16	25	16	30		
Age-adjusted RR	1.00	1.25 (0.66-2.34)	1.10 (0.55-2.21)	2.59 (1.39-4.81)	.003	.003
Adjusted RR‡	1.00	1.38 (0.72-2.65)	0.96 (0.45-2.05)	1.84 (0.94-3.58)	.08	.16
Adjusted RR§	1.00	1.58 (0.82-3.06)	0.95 (0.44-2.04)	1.75 (0.89-3.45)	.11	.28
Other non-CVD, noncancerous death (n = 85)						
Cases, No.	15	28	13	29		
Age-adjusted RR	1.00	1.74 (0.93-3.28)	1.21 (0.57-2.57)	3.56 (1.88-6.76)†	<.001	<.001
Adjusted RR‡	1.00	2.07 (1.04-4.12)	1.44 (0.65-3.21)	3.41 (1.67-7.11)†	.001	.003
Adjusted RR§	1.00	2.12 (1.07-4.20)	1.38 (0.62-3.07)	3.20 (1.57-6.56)†	.001	.006
Adjusted RR	1.00	2.17 (1.01-4.30)	1.39 (0.63-3.09)	3.27 (1.60-6.68)†	.002	.007

Abbreviation: CVD, cardiovascular disease.

*Values in parentheses are 95% confidence intervals.

†Comparisons between height loss of 3 cm or more and height loss of less than 1 cm.

‡Adjusted for age, social class, smoking, alcohol intake, physical activity, body mass index (calculated as weight in kilograms divided by height in meters squared), preexisting coronary heart disease, stroke, diabetes, systolic blood pressure, cholesterol, high-density lipoprotein cholesterol, and forced expiratory volume in 1 second–forced vital capacity ratio.

§Adjusted for factors listed above as well as for albumin, poor/fair health, and musculoskeletal problems.

||Adjusted for factors listed above as well as for 20-year weight loss (from initial screening to 20-year reexamination [Q1-Q20]).

Table 4. Height Loss and Incidence of Major Coronary Heart Disease (CHD) and Stroke Events

Variable	Height Loss, cm				P Value*	P Value for Trend Across Groups
	<1.0 (n = 1471)	1.0-1.9 (n = 1330)	2.0-2.9 (n = 807)	≥3.0 (n = 605)		
Major CHD Events (n = 249)						
Cases, No.	74	67	56	52		
Rate per 1000 person-years	10.1	10.1	14.0	18.3		
Age-adjusted RR	1.00	0.88 (0.63-1.23)	1.13 (0.31-1.61)	1.38 (0.96-1.99)	.09	.06
Adjusted RR (95% CI)†	1.00	0.92 (0.65-1.31)	1.20 (0.81-1.75)	1.55 (1.05-2.29)	.03	.02
Adjusted RR (95% CI)‡	1.00	0.93 (0.65-1.32)	1.19 (0.81-1.74)	1.47 (0.99-2.18)	.05	.04
Adjusted RR (95% CI)§	1.00	0.92 (0.65-1.32)	1.18 (0.81-1.72)	1.43 (0.97-2.17)	.07	.05
Major Stroke Events (n = 153)						
Cases, No.	49	57	22	25		
Rate per 1000 patient-years	6.6	8.5	5.4	8.6		
Age-adjusted RR (95% CI)	1.00	0.95 (0.65-1.40)	0.54 (0.32-0.90)	0.86 (0.52-1.41)	.62	.14
Adjusted RR (95% CI)†	1.00	1.10 (0.73-1.67)	0.57 (0.33-0.99)	0.95 (0.56-1.62)	.88	.26

Abbreviation: RR, relative risk.

*Comparisons between height loss of 3 cm or more and height loss of less than 1 cm.

†Adjusted for age, social class, smoking, alcohol intake, physical activity, body mass index (calculated as weight in kilograms divided by height in meters squared); preexisting coronary heart disease; stroke, diabetes, systolic blood pressure, cholesterol, high-density lipoprotein cholesterol, and forced expiratory volume in 1 second–forced vital capacity ratio.

‡Adjusted for factors listed above as well as for albumin, poor/fair health, and musculoskeletal problems.

§Adjusted for all factors listed, including 20-year weight loss. The adjusted RR (95% confidence interval) for 3.0 cm or more vs less than 3.0 cm is 1.42 (1.02-1.98); P = .04.

poor/fair health, musculoskeletal problems, and weight loss made small differences, but the difference between men with a height loss of 3.0 cm or more compared with those with a height loss of less than 1.0 cm became of marginal significance ($P=.07$). However, men with a height loss of 3.0 cm or more had a significantly higher CHD risk than all men with a height loss of less than 3.0 cm combined (adjusted RR, 1.42; 95% CI, 1.02-1.98). No association was seen between height loss and risk of stroke.

COMMENT

In this study of 4213 men aged 60 to 79 years, a height loss of 3 cm or more over the preceding 20 years (present in about 15% of the men) was associated with a significantly increased risk of all-cause mortality largely owing to an excess in cardiovascular, respiratory, and other non-CVD, noncancer deaths. Height loss was associated with major CHD events but not stroke. Height loss was strongly and positively associated with increasing age, physical inactivity, and indicators of ill health (reduced lung function, COPD, poor/fair health, musculoskeletal and joint disorders, mobility limitation, weight loss, and low albumin levels) but inversely associated with serum cholesterol levels. The association with respiratory deaths was largely attributable to the higher prevalence of COPD, but this did not explain the excess in CVD deaths. Although the increased risks of total mortality and CHD were to some extent associated with measures of poor health, weight loss, and physical inactivity, a significant increase in mortality associated with height loss remained even after adjustment for these factors. Despite a strong association between height loss and weight loss, we observed no association between height loss and cancer mortality, even in age-adjusted analysis.

POTENTIAL MECHANISMS RELATING HEIGHT LOSS AND MORTALITY

The basis for the association between height loss and subsequent mortality is unclear. Osteoporosis is associated with increased mortality^{30,31} and could be important as one of the main causes of height loss in men.^{4,6} However, height loss resulting from osteoporosis, particularly when complicated by fracture, is likely to be more than 6.0 cm.^{32,33} Because mortality was markedly increased in our study, even in men with a height loss of 3.0 to 4.0 cm, osteoporotic disease complicated by vertebral fractures is unlikely to explain the increased mortality risk associated with height loss, although lesser degrees of bone loss (possibly associated with minor degrees of kyphosis, which could also be responsible for height loss) could still be implicated.

It is possible that bone loss (an important determinant of height loss^{34,35}) may share common pathophysiologic mechanisms with coronary diseases such as dyslipidemia, oxidative stress, inflammation, hyperhomocystinemia, hypertension, and diabetes.^{15,16} However, the association between height loss and CHD in this study was not explained by established cardiovascular risk factors, and men with significant height loss tended to have more favorable levels of cardiovascular risk factors than men with

minimal height loss. Furthermore despite the known association between smoking and bone loss,³⁶⁻³⁹ we observed no association between smoking and height loss, suggesting that the height loss in our study is not just reflecting bone resorption and bone density.

A third potential explanation for the relation of height loss to mortality and CHD may lie in the relation of height loss to weight loss at older ages. Poor muscle strength and low skeletal muscle mass have been associated with bone loss and poor bone structure in men, which may result in height loss.⁷ The increased risk of CHD and all-cause mortality associated with height loss may thus reflect poor muscle strength and loss in skeletal muscle mass with aging (sarcopenia), both of which have been shown to be predictors of mortality.¹²⁻¹⁴ Therefore, height changes could be a consequence of aging or could reflect the presence of disease in 1 of several organ systems. This possibility is consistent with the findings that height loss is associated with lower body weight and weight loss, low albumin levels (marker of undernutrition), physical inactivity, COPD and mobility limitations, factors that are strongly associated with sarcopenia and loss of skeletal muscle mass^{11,40,41} and that have been shown to predict increased mortality in the elderly.^{42,43} Inflammation, which is an established risk factor for CVD, may also contribute to decreased skeletal muscle in old age.⁴⁴ However, adjustment for C-reactive protein, a marker of inflammation, made little difference in our findings. Although the increased risk of mortality was seen even after adjustment for weight loss and markers of ill health, we do not have measures of muscle strength or of loss in skeletal muscle mass. Adjustment for fat-free mass, as a possible marker of muscle mass, made little difference in the findings. However, fat-free mass, as assessed by bioelectrical impedance methods, is not the most precise method for measuring skeletal muscle mass. We are therefore unable to confirm or refute this possibility in the present study.

STRENGTHS AND LIMITATIONS

Our study was based on measured height using similar methods at initial screening and reexamination, thus reducing measurement errors. We also assessed a wide range of biologic risk factors and objective markers and indicators of ill health. Although the data from the present study are restricted to men who participated in both initial and repeated examinations (including 77% of survivors at reexamination), the mean initial height of men who attended reexamination and that of men who did not were almost identical, suggesting that any selection bias introduced was limited. Our study was carried out in a population-based study of men. Although it is possible that our findings would apply to women, such generalization must be cautious.

In conclusion, height loss (≥ 3 cm) is relatively common in older men; is associated with poor health, mobility limitation, weight loss, physical inactivity, and reduced pulmonary function; and is a predictor of CHD and total mortality. Height loss may be a marker for sarcopenia and frailty in older men. Further studies are warranted to understand the nature of this relationship between height loss and CHD and total mortality.

Accepted for Publication: May 9, 2006.

Correspondence: S. Goya Wannamethee, PhD, Department of Primary Care and Population Sciences, Royal Free and University College Medical School, Rowland Hill Street, London NW3 2PF, England (goya@pcps.ucl.ac.uk).

Author Contributions: *Study concept and design:* Wannamethee and Whincup. *Acquisition of data:* Lennon and Whincup. *Analysis and interpretation of data:* Wannamethee, Shaper, and Whincup. *Drafting of the manuscript:* Wannamethee, Shaper, and Lennon. *Critical revision of the manuscript for important intellectual content:* Wannamethee, Shaper, and Whincup. *Statistical analysis:* Wannamethee. *Obtained funding:* Whincup. *Administrative, technical, and material support:* Lennon and Whincup. Dr Whincup was responsible for the 20-year rescreening of the study population, and Ms Lennon was responsible for the follow-up of the men and for obtaining the morbidity and mortality data.

Financial Disclosure: None reported.

Funding/Support: The British Regional Heart Study is a British Heart Foundation Research Group and receives support from the Department of Health (England).

Disclaimer: The views expressed in this publication are those of the authors and not necessarily those of the Department of Health (England).

REFERENCES

1. Sorkin JD, Muller DC, Andres R. Longitudinal change in height of men and women: implications for interpretation of the body mass index. *Am J Epidemiol*. 1999;150:969-977.
2. Sorkin JD, Muller DC, Andres R. Longitudinal change in height of men and women: consequential effects on body mass index. *Epidemiol Rev*. 1999;21:247-260.
3. Dequeker JV, Baeyens JP, Claessens J. The significance of stature as a clinical measurement of aging. *J Am Geriatr Soc*. 1969;17:169-179.
4. Ross PD. Clinical consequences of vertebral fractures. *Am J Med*. 1997;103(suppl 2A):30S-43S.
5. Papaioannou A, Watts NB, Kendler DL, Yuen CK, Adachi JD, Ferko N. Diagnosis and management of vertebral fractures in elderly adults. *Am J Med*. 2002;113:220-228.
6. Old JL, Calvert M. Vertebral compression fractures in the elderly. *Am Fam Physician*. 2004;69:111-116.
7. Szulc P, Beck TJ, Marchand F, Delmas PD. Low skeletal muscle mass is associated with poor structural parameters of bone and impaired balance in elderly men—the MINOS study. *J Bone Miner Res*. 2005;20:721-729.
8. Rantanen T, Harris T, Leveille SG, et al. Muscle strength and body mass index as long-term predictors of mortality in initially healthy men. *J Gerontol A Biol Sci Med Sci*. 2000;55:M168-M173.
9. Metter EJ, Talbot LA, Schrager M, Conwit R. Skeletal muscle strength as a predictor of all cause mortality in healthy men. *J Gerontol A Biol Sci Med Sci*. 2002;57:B359-B365.
10. Roubenoff R, Parise H, Payette HA, et al. Cytokines, insulin-like growth factor 1, sarcopenia, and mortality in very old community-dwelling men and women: the Framingham Heart Study. *Am J Med*. 2003;115:429-435.
11. Castillo EM, Goodman-Gruen D, Kritz-Silverstein D, Morton DJ, Wingard DL, Barrett-Connor E. Sarcopenia in elderly men and women: the Rancho Bernardo Study. *Am J Prev Med*. 2003;25:226-231.
12. Iannuzzi-Sucich M, Prestwood KM, Kenny AM. Prevalence of sarcopenia and predictors of skeletal muscle mass in healthy, older men and women. *J Gerontol A Biol Sci Med Sci*. 2002;57:M772-M777.
13. Hughes VA, Frontera WR, Roubenoff R, Evans WJ, Singh MAF. Longitudinal changes in body composition in older men and women: role of body weight change and physical activity. *Am J Clin Nutr*. 2002;76:473-481.
14. Roubenoff R. Sarcopenia: effects on body composition and function. *J Gerontol A Biol Sci Med Sci*. 2003;58:1012-1017.
15. McFarlane SI, Muniyappa R, Shin JJ, Bahtiyar G, Sowers JR. Osteoporosis and cardiovascular disease: brittle bones and boned arteries, is there a link? *Endocrine*. 2004;23:1-10.
16. Whitney C, Warburton DE, Frohlich J, Chan SY, McKay H, Khan K. Are cardiovascular disease and osteoporosis directly linked? *Sports Med*. 2004;34:779-807.
17. Shaper AG, Pocock SJ, Walker M, Cohen NM, Wale CJ, Thomson AG. British Regional Heart Study: cardiovascular risk factors in middle-aged men in 24 towns. *BMJ (Clin Res Ed)*. 1981;283:179-186.
18. Wannamethee SG, Shaper AG, Whincup PH. Body fat distribution, body composition, and respiratory function in elderly men. *Am J Clin Nutr*. 2005;82:996-1003.
19. Deurenberg P, van der Kooij K, Evers P, Hulshof T. Assessment of body composition by bioelectrical impedance in a population aged >60 years. *Am J Clin Nutr*. 1990;51:3-6.
20. Wannamethee G, Shaper AG. Weight change in middle-aged British men: implications for health. *Eur J Clin Nutr*. 1990;44:133-142.
21. Wannamethee SG, Lowe GDO, Whincup PH, et al. Physical activity and hemostatic and inflammatory variables in elderly men. *Circulation*. 2002;105:1785-1790.
22. Wannamethee SG, Shaper AG, Whincup PH. Overweight and obesity and the burden of disease and disability in elderly men. *Int J Obes*. 2004;28:1374-1382.
23. Cole TJ. Linear and proportional regression models in the prediction of ventilatory function: with discussion. *J R Stat Soc Ser A*. 1975;138:297-338.
24. Pauwels RA, Buist AS, Ma P, Jenkins CR, Hurd SS; GOLD Scientific Committee. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: National Heart, Lung, and Blood Institute and World Health Organization Global Initiative for Chronic Obstructive Lung Disease (GOLD): executive summary. *Respir Care*. 2001;46:798-825.
25. Lampe FC, Walker M, Lennon LT, et al. Validity of a self reported history of doctor-diagnosed angina. *J Clin Epidemiol*. 1999;52:73-81.
26. Walker MK, Whincup PH, Shaper AG, et al. Validation of patient recall of doctor-diagnosed heart attack and stroke. *Am J Epidemiol*. 1998;148:355-361.
27. *British National Formulary*. London, England: British Medical Association and Royal Pharmaceutical Society of Great Britain; 1994.
28. Walker M, Shaper AG, Lennon L, Whincup PH. Twenty year follow-up of a cohort study based in general practices in 24 British towns. *J Public Health Med*. 2000;22:479-485.
29. Rose G, Blackburn H, Gillum RF, Prineas RJ. *Cardiovascular Survey Methods*. 2nd ed. Geneva, Switzerland: World Health Organization; 1982.
30. Center JR, Nguyen TV, Schneider D, Sambrook PN, Eisman JA. Mortality after all major types of osteoporotic fracture in men and women: an observational study. *Lancet*. 1999;353:878-882.
31. Hasselius R, Karlsson MK, Nilsson BE, Redlund-Johnell I, Johnell O. Prevalent vertebral deformities predict increased mortality and increased fracture rate in both men and women: a 10-year population-based study of 598 individuals from the Swedish cohort in the European Vertebral Osteoporosis Study. *Osteoporos Int*. 2003;14:61-68.
32. Campion JM, Maricic MJ. Osteoporosis in men. *Am Fam Physician*. 2003;67:1521-1526.
33. Siminowski K, Warshawski H, Jen H, Lee K. The accuracy of historical height loss for the detection of vertebral fractures in postmenopausal women. *Osteoporos Int*. 2006;17:290-296.
34. Knoke JD, Barrett-Connor E. Weight loss: a determinant of hip bone loss in older men and women. *Am J Epidemiol*. 2003;158:1132-1138.
35. Ensrud KE, Fullman RL, Barrett-Connor E, et al; Osteoporotic Fractures in Men Study Research Group. Voluntary weight reduction in older men increases hip bone loss. *J Clin Endocrinol Metab*. 2005;90:1998-2004.
36. Dennison E, Eastell R, Fall CHD, Kellingray S, Wood PJ, Cooper C. Determinants of bone loss in elderly men and women: a prospective population-based study. *Osteoporos Int*. 1999;10:384-391.
37. Orwoll ES, Bevan L, Phipps KR. Determinants of bone mineral density in older men. *Osteoporos Int*. 2000;11:815-821.
38. Hannan MT, Felson DT, Dawson-Hughes B, et al. Risk factors for longitudinal bone loss in elderly men and women: the Framingham Osteoporosis Study. *J Bone Miner Res*. 2000;15:710-720.
39. Izumotani K, Hagiwara S, Izumotani T, Miki T, Morii H, Nishizawa Y. Risk factors for osteoporosis in men. *J Bone Miner Metab*. 2003;21:86-90.
40. Visser M, Kritchevsky SB, Newman AB, et al. Lower serum albumin concentration and change in muscle mass: the Health, Aging and Body Composition Study. *Am J Clin Nutr*. 2005;82:531-537.
41. Sergi G, Coin A, Marin S, et al. Body composition and resting energy expenditure in elderly male patients with chronic obstructive pulmonary disease. *Respir Med*. 2006;Epub ahead of print.
42. Thomas DR. Weight loss in older adults. *Rev Endocr Metab Disord*. 2005;6:129-136.
43. Corti M, Guralnik JM, Salive ME, Sorkin JD. Serum albumin level and physical disability as predictors of mortality in older persons. *JAMA*. 1994;272:1036-1042.
44. Visser M, Pahor M, Taaffe DR, et al. Relationship of interleukin-6 and tumor necrosis factor-alpha with muscle mass and muscle strength in elderly men and women. *J Gerontol A Biol Sci Med Sci*. 2002;57:M326-M332.