

Body mass index and preinfarction angina in elderly patients with acute myocardial infarction¹⁻⁴

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ABSTRACT

Background: Preinfarction angina, a clinical equivalent of ischemic preconditioning, seems to protect against in-hospital death, cardiogenic shock, and the combined endpoints in adult but not in elderly patients with acute myocardial infarction. Experimental evidence indicates that caloric restriction may restore ischemic preconditioning in aged animals.

Objective: The objective was to verify whether body mass index (BMI) influences the cardioprotective effect of preinfarction angina in the elderly.

Design: We retrospectively studied 820 patients aged ≥ 65 y with acute myocardial infarction by evaluating BMI and major (death and cardiogenic shock) and minor in-hospital outcomes.

Results: In-hospital death, cardiogenic shock, and the combined endpoints were not significantly different between elderly patients with and without preinfarction angina. Interestingly, in-hospital death, cardiogenic shock, and the combined endpoints were significantly fewer in elderly patients with than without preinfarction angina in the subset of patients with the lowest BMI ($P < 0.01$, < 0.01 , and < 0.01 , respectively). Regression analysis showed that preinfarction angina did not protect against in-hospital death when analyzed in all patients independently of BMI, whereas it was protective in the subset of patients with the lowest BMI (odds ratio: 0.06; 95% CI: 0.00, 0.54).

Conclusions: Preinfarction angina does not protect against in-hospital death, cardiogenic shock, or the combined endpoints in elderly patients with acute myocardial infarction. With stratification by quartiles of BMI, the protective effect of preinfarction angina is preserved in elderly patients with the lowest BMI. *Am J Clin Nutr* 2003;78:796–801.

KEY WORDS Body mass index, preinfarction angina, elderly, aging, mortality

INTRODUCTION

Coronary artery disease (CAD) is linked to increased mortality with advancing age (1, 2). This phenomenon has been attributed to the higher rate of comorbidity and less frequent use of thrombolytic therapy in the elderly (3, 4). However, age still remains an independent predictor of mortality from CAD (5). This prompted the intriguing notion that some endogenous protective mechanism might become less effective with age. Ischemic preconditioning is the most powerful protective mechanism against myocardial ischemia (6, 7). In fact, brief episodes

of ischemia protect the heart from successive and more prolonged ischemic periods (6, 7). Preinfarction angina, a classic clinical equivalent of ischemic preconditioning, determines a better prognosis in patients with acute myocardial infarction (AMI) (8, 9). Experimental studies have shown that ischemic preconditioning becomes less effective with age (10, 11) and that the protective effect of preinfarction angina is reduced in elderly patients affected by AMI (12, 13).

Both physical activity and caloric restriction attenuate age-related cardiovascular modifications and significantly prolong the life span of mice and rats (14, 15). The antiaging molecular mechanism of physical activity and caloric restriction is not well understood. Physical activity (16) and caloric restriction (17) have been shown to restore ischemic preconditioning in aged rats by increasing norepinephrine release in response to a preconditioning stimulus. Moreover, a high level of physical activity preserves the cardioprotective effect of preinfarction angina in elderly patients with AMI (18).

Obesity increases mortality in adult and elderly patients with cardiovascular diseases (19, 20). This relation could be due to the higher prevalence of diabetes, hypertension, and hypercholesterolemia observed in obese patients (19). However, these conditions do not completely explain this relation, especially in elderly patients. Cholesterol concentrations, for example,

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decrease progressively after 70 y of age (21). However, it is not known whether clinical equivalents of ischemic preconditioning, such as preinfarction angina, are influenced by overweight in elderly patients.

The aim of our study was to verify whether body mass index (BMI) influences the cardioprotective effect of preinfarction angina on the in-hospital endpoints mortality and cardiogenic shock in elderly patients with AMI.

METHODS

Study design

Between January 1995 and December 2001, 820 patients aged ≥ 65 y with AMI were admitted to our Coronary Care Unit regardless of their functional status. Patients were included in this retrospective study if they met ≥ 2 of the following conditions: 1) typical chest pain, 2) electrocardiographic changes with Q waves (transmural infarction), and 3) elevated creatinine kinase concentrations. Nontransmural infarction was diagnosed by typical changes in the S-T segment and T-wave accompanied by increased creatine kinase-myoglobin fraction concentrations. The medical records of these patients were individually reviewed as described previously (12). Thrombolytic therapy was given to all patients, except to those with complicating illness, no chest pain on admission, or no nonspecific electrocardiographic abnormalities; an age ≥ 65 y was not considered a contraindication. Eighteen patients (2.1%) with terminal illness, 20 patients (2.4%) with cerebrovascular disease, and 35 patients (4.3%) with dementia were excluded. The final number of patients included in the study was 747.

Assessment of body mass index

BMI was defined as weight in kilograms divided by the square of the height in meters. BMI quartiles were determined before data collection and stratified according to Metropolitan Life Insurance tables (22). Because we excluded terminally ill patients, none of our patients had a BMI < 19 . Moreover, to simplify the analysis, all patients with a BMI ≥ 27 were considered in a single quartile. Thus, the final BMI quartiles were as follows: 19.0–21.9, 22.0–24.9, 25.0–26.9, and ≥ 27 .

Assessment of physical activity

Physical activity was evaluated with the Physical Activity Scale for the Elderly (PASE) (23). The total PASE score was computed by multiplying the time spent at each activity (hours/d) or participation (yes or no) in an activity by the empirically derived item weights and summing over all activities. The total PASE score obtained was stratified in 4 quartiles (0–40, 41–56, 57–90, and > 90) for statistical analysis.

Analysis of preinfarction angina

A staff physician collected a detailed clinical history for all patients. Patients who had not experienced chest pain, chest discomfort, or left arm and jaw pain 24 h before the episode leading to admission were defined as having no preinfarction angina. Patients with an angina episode lasting < 30 min, 24 h before the AMI, were defined as having preinfarction angina. Patients with a history of angina at any time before the AMI were defined as having chronic angina.

In-hospital outcomes

In-hospital endpoints in the coronary care unit were as follows: death, cardiogenic shock (marked and persistent hypotension with

systolic arterial pressure < 80 mm Hg and a reduction of the cardiac index to < 1.8 L/mm²), reinfarction, recurrent ischemic pain, creatine kinase-myoglobin peak, ventricular fibrillation and tachycardia, and high-grade atrioventricular block.

Statistical analysis

The analysis was performed with the use of EPIINFO (version 5.01, EpiInfo, Atlanta) and SPSS (Statistical Package for the Social Sciences version 11.0 for WINDOWS; SPSS, Inc, Chicago). The statistical power of detecting a clinically significant difference in elderly patients with AMI was 95% with a type I error of 0.05 and an odds ratio as large as 2.00 (corresponding to a mortality rate of 20% in patients with a BMI ≥ 27 before AMI). The power declined progressively to 90%, 80%, and 70% with an odds ratio of 1.90, 1.80, and 1.60, respectively. A two-factor analysis of variance on ranks analysis was performed, and the *P* values for the main effects of BMI and preinfarction angina and their interaction were reported. With the data stratified in 4 BMI categories (19.0–21.9, 22.0–24.9, 25.0–26.9, and ≥ 27) and divided on the basis of the absence or presence of preinfarction angina, categorical data and continuous variables were analyzed by chi-square and Student's *t* tests, respectively. Logistic regression analysis was used to assess the effect of preinfarction angina on death, cardiogenic shock, and the combined endpoints independently from baseline characteristics (age, sex, family history, physical activity, BMI, low educational level, chronic angina, previous AMI, smoking, hypertension, congestive heart failure, diabetes, cholesterolemia, and antianginal treatment), from the interaction of BMI and preinfarction angina, and from thrombolytic therapy and primary percutaneous transluminal coronary angioplasty. The effect of preinfarction angina on death, cardiogenic shock, and the combined endpoints was also assessed in elderly patients stratified in quartiles of BMI (19.0–21.9, 22.0–24.9, 25.0–26.9, and ≥ 27). *P* values are reported for two-sided testing and were considered significant if < 0.05 .

RESULTS

Baseline characteristics

Patients with preinfarction angina were older than patients without preinfarction angina only in the highest BMI group. Similarly, smoking, congestive heart failure, and diabetes were more frequent in elderly patients with than in those without preinfarction angina in the highest BMI group (Table 1). However, elderly patients with preinfarction angina in all BMI groups more frequently took nitrates and aspirin (Table 1).

In-hospital outcomes

In-hospital outcomes (death, cardiogenic shock, and the combined endpoints) were not significantly different between elderly patients with and without preinfarction angina. When elderly patients were stratified by BMI quartiles and by the absence or presence of preinfarction angina, in-hospital death, cardiogenic shock, and the combined endpoints were significantly fewer in patients with than in those without preinfarction angina, only in the lowest BMI group (Table 2, Figure 1). In contrast, preinfarction angina did not significantly affect the incidence of in-hospital death, cardiogenic shock, or the combined endpoints in the other BMI groups (Table 2, Figure 1).

Logistic regression models

We evaluated the role of preinfarction angina using logistic regression analysis with death, cardiogenic shock, and the combined endpoints (in-hospital death and cardiogenic shock) as dependent vari-

TABLE 1

Baseline characteristics of elderly patients according to quartiles of BMI and the absence or presence of preinfarction angina¹

Characteristic	BMI							
	19.0–21.9 (n = 122)		22.0–24.9 (n = 173)		25.0–26.9 (n = 168)		≥27.0 (n = 284)	
	Absence	Presence	Absence	Presence	Absence	Presence	Absence	Presence
Age ^{2,3}	74.4 ± 6.9 ⁴	74.7 ± 5.5	72.0 ± 5.1	73.1 ± 6.6	75.1 ± 6.3	74.4 ± 5.8	70.7 ± 5.2	73.1 ± 5.8 ⁵
Men [n (%)] ^{2,3}	30 (24.6)	44 (36.1)	52 (30.1)	66 (38.2) ⁶	59 (35.1)	58 (34.5) ⁵	60 (21.1)	120 (42.3)
Family history [n (%)] ^{2,3,7}	19 (15.6)	26 (21.3)	21 (12.1)	55 (31.8) ⁵	27 (16.1)	41 (24.4)	28 (9.9)	80 (28.2)
PASE ^{2,3,7}	67.9 ± 33.5	71.2 ± 32.8	60.9 ± 33.1	61.1 ± 36.5	61.3 ± 38.1	62.9 ± 33.0	59.5 ± 31.4	60.0 ± 29.9
Low educational level [n (%)] ^{2,3}	38 (31.1)	69 (56.6)	62 (35.8)	87 (50.3)	54 (32.1)	80 (47.6)	74 (26.1)	168 (59.2)
Chronic angina [n (%)] ^{2,3}	19 (15.6)	20 (16.4)	15 (8.7)	32 (18.5)	22 (13.1)	39 (23.2)	28 (9.9)	67 (23.3)
Previous AMI [n (%)] ^{2,3}	10 (8.2)	8 (6.6)	13 (7.5)	18 (10.4)	10 (6.0)	12 (7.1)	20 (7.0)	36 (12.7)
Smoking [n (%)] ^{2,3}	21 (17.2)	44 (36.1)	35 (20.2)	70 (40.5) ⁶	47 (28.0)	45 (26.8) ⁶	55 (19.4)	87 (30.6) ⁶
Hypertension [n (%)] ^{2,3}	30 (24.6)	39 (32.0)	30 (17.3)	61 (35.3) ⁶	23 (13.7)	45 (26.8)	48 (16.9)	104 (36.6)
CHF [n (%)] ^{2,3}	5 (4.1)	2 (1.2)	1 (0.6)	10 (5.8) ⁶	8 (4.8)	6 (3.6)	16 (5.6)	16 (5.6) ⁶
Diabetes [n (%)] ^{2,3}	13 (10.7)	27 (22.1)	20 (11.6)	47 (27.2) ⁶	28 (16.7)	42 (25.0)	58 (20.4)	65 (22.9) ⁵
Cholesterol ^{2,3}	174.1 ± 41.2	190.3 ± 36.3 ⁶	216.1 ± 86.5	174.8 ± 33.2 ⁵	196.3 ± 52.6	183.5 ± 48.1	193.0 ± 43.5	184.7 ± 45.3
Nitrates [n (%)] ^{2,3,7}	8 (6.6)	33 (27.0) ⁵	18 (10.4)	44 (25.4) ⁶	6 (3.6)	55 (32.7) ⁵	5 (1.8)	108 (38.0) ⁵
β-Blockers [n (%)] ^{2,3}	3 (2.5)	4 (3.3)	0 (0.0)	9 (5.2) ⁵	3 (1.8)	4 (2.4)	7 (2.5)	6 (2.1)
Calcium blockers [n (%)] ^{2,3}	18 (14.8)	16 (13.1) ⁶	15 (8.7)	25 (14.5)	10 (6.0)	36 (21.4) ⁵	18 (6.3)	39 (13.7)
Aspirin [n (%)] ^{2,3,7}	4 (3.3)	28 (23.0) ⁵	0 (0.0)	32 (18.5) ⁵	0 (0.0)	45 (26.8) ⁵	3 (1.1)	107 (37.7) ⁵
Rales > 1/3 lung fields [n (%)] ²	7 (5.8)	4 (3.3)	0 (0.0)	23 (13.5)	16 (9.9)	13 (8.0)	13 (4.8)	40 (14.8)

¹n = 747. PASE, Physical Activity Scale for the Elderly; AMI, acute myocardial infarction; CHF, congestive heart failure.^{2,3,7}Two-factor ANOVA on ranks was used to evaluate the main effect of BMI and preinfarction angina and their interactions: ²P < 0.01 for effect of BMI, ³P < 0.01 for interaction of BMI and preinfarction angina, ⁶P < 0.01 for effect of preinfarction angina.⁴ $\bar{x} \pm$ SD.^{5,6}Significantly different from respective patients without angina (chi-square test or Student's *t* test): ⁴P < 0.01, ⁵P < 0.05.

ables. When the analysis was done for all patients, both age and BMI were predictive with all dependent variables considered (Table 3). Preinfarction angina did not exert a significant protective effect when analyzed in all patients independently of BMI. Interestingly, the interaction of BMI and preinfarction angina did not significantly affect the logistic regression model, but it reduced to some extent the predictive value of BMI. More importantly, when we examined preinfarction angina according to BMI quartiles, we found a protective effect only in the subset of patients with the lowest BMI scores.

DISCUSSION

In the current study, preinfarction angina did not protect against major in-hospital outcomes (death and cardiogenic shock) in elderly patients with AMI. Moreover, a low BMI exerted its protective effect in elderly patients with preinfarction angina but not in those without preinfarction angina.

Ischemic preconditioning and the aging heart

Age is a powerful predictor of mortality from AMI (1–5). Although the high rate of comorbidity and the reduction of

TABLE 2

In-hospital outcomes of elderly patients according to quartiles of BMI and the absence or presence of preinfarction angina¹

In-hospital outcome	BMI							
	19.0–21.9 (n = 122)		22.0–24.9 (n = 173)		25.0–26.9 (n = 168)		≥27.0 (n = 284)	
	Absence	Presence	Absence	Presence	Absence	Presence	Absence	Presence
Death [n (%)] ^{2,3}	14 (11.5)	5 (4.1) ⁴	14 (8.1)	15 (8.7)	22 (13.1)	16 (9.5)	24 (8.5)	66 (23.3)
Cardiogenic shock [n (%)] ^{2,3}	16 (13.1)	9 (7.4) ⁴	17 (9.8)	20 (11.6)	17 (10.1)	24 (14.3)	26 (9.2)	68 (23.9)
Death and cardiogenic shock [n (%)] ^{2,3}	20 (16.4)	11 (9.0) ⁴	24 (13.9)	26 (15.0)	25 (14.9)	33 (19.6)	36 (12.7)	87 (30.6)
Thrombolysis [n (%)] ²	15 (12.3)	18 (14.8)	26 (15.0)	25 (14.5)	21 (12.5)	27 (16.1)	30 (10.6)	56 (19.7)
Time to T (< 4 h) [n (%)] ²	10 (8.2)	11 (9.0)	13 (7.5)	14 (8.1)	11 (6.5)	15 (8.9)	21 (7.4)	33 (11.6)
Time to T (≥ 4 h) [n (%)] ²	5 (4.1)	7 (5.7)	13 (7.5)	11 (6.4)	10 (6.0)	12 (7.1)	9 (3.2)	23 (8.1)
Primary angioplasty [n (%)] ^{2,3}	6 (4.9)	2 (1.6)	5 (2.9)	5 (2.9)	4 (2.4)	7 (4.2)	6 (2.1)	14 (4.9)
In-hospital reinfarction [n (%)] ²	5 (4.1)	2 (1.6)	7 (4.0)	8 (4.6)	9 (5.4)	10 (6.0)	11 (3.9)	23 (8.1)
Recurrent ischemic pain [n (%)] ^{2,3}	7 (5.7)	7 (5.7)	10 (5.8)	13 (7.5)	12 (7.1)	15 (8.9)	12 (4.2)	42 (14.8)
Non-Q wave AMI [n (%)]	8 (6.6)	12 (9.8)	9 (5.2)	13 (7.5)	9 (5.4)	8 (4.8)	11 (3.9)	14 (4.9)
CK-MB peak (IU/L) ²	176 ± 97 ⁵	149 ± 145	191 ± 136	156 ± 153	224 ± 185	179 ± 129	196 ± 125	203 ± 168
Ventricular fibrillation [n (%)] ^{2,3}	5 (4.1)	2 (1.6)	7 (4.0)	5 (2.9)	9 (5.4)	10 (6.0)	9 (3.2)	28 (9.9)
Ventricular tachycardia [n (%)] ^{2,3,6}	6 (4.9)	14 (11.5)	17 (9.8)	23 (13.3)	19 (11.3)	27 (16.1)	20 (7.0)	68 (23.9) ⁷
Atrioventricular block [n (%)] ^{2,3}	2 (1.6)	2 (1.6)	12 (6.9)	9 (5.2)	11 (6.5)	18 (10.7)	11 (3.9)	33 (11.6)

¹n = 747. T, thrombolysis; AMI, acute myocardial infarction; CK-MB, creatine kinase–myoglobin fraction.^{2,3,6}Two-factor ANOVA on ranks was used to evaluate the main effect of BMI and preinfarction angina and their interactions: ²P < 0.01 effect of BMI, ³P < 0.01 for interaction of BMI and preinfarction angina, ⁵P < 0.01 for effect of preinfarction angina.⁵ $\bar{x} \pm$ SD.^{4,7}Significantly different from respective patients without angina (chi-square test or Student's *t* test): ⁴P < 0.01, ⁶P < 0.05.

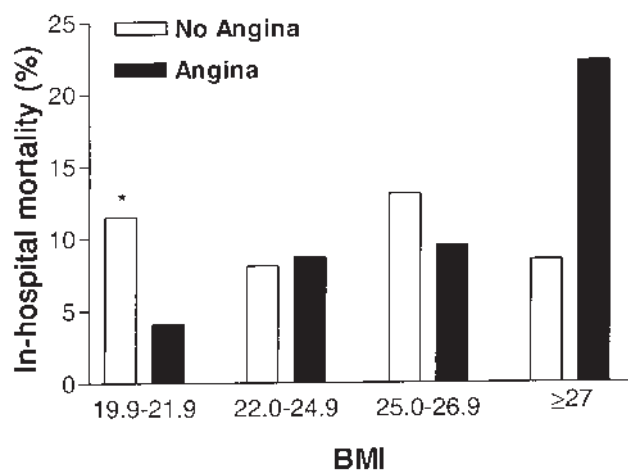


FIGURE 1. Prevalence of in-hospital mortality in elderly patients with acute myocardial infarction stratified into 4 BMI quartiles and according to the presence or absence of preinfarction angina. *Significantly different from patients with angina, $P < 0.01$.

thrombolytic therapy observed in the elderly seem more plausible explanations (3–4), neither factor completely explains the age-related increase in AMI mortality. The higher mortality and morbidity associated with advancing age could be due to the “winding down” of some endogenous protective mechanism against myocardial ischemia, a classic example being ischemic preconditioning. Ischemic preconditioning consists of brief, recurrent episodes of ischemia and reperfusion that reduce infarct size and postischemic dysfunction (6, 7). The most important clinical equivalent of ischemic preconditioning is preinfarction angina that reduces left ventricular dysfunction (24), infarct size (25), the number of in-hospital events (8), and the risk of right ventricular infarct (9). This protection exists in adult but not in elderly patients. Reduction of age-related ischemic preconditioning has been shown in both animals (10, 11) and

humans (12, 13, 26–28). These data indicate that the higher mortality from AMI observed in elderly patients should be attributed, at least in part, to the age-related reduction in this protective endogenous mechanism.

Body mass index, aging, and coronary artery disease

J- and U-shaped relations were observed between obesity and cardiovascular disease mortality (29, 30), but the relations became linear when smokers and concomitant diseases were excluded from the analysis (31, 32). When the correlation between BMI and cardiovascular disease mortality was related to age, the analysis became even more complex. Stevens et al (20) reported an association between increased BMI and increased cardiovascular disease mortality, but the phenomenon progressively declined with aging. More recently, Calle et al (32) showed that a high BMI is related to an increased risk of cardiovascular disease mortality in all age groups, including subjects aged > 75 y. Although the relative risk of mortality decreased progressively with age, the absolute risk increased progressively with advancing age and reached a maximum in elderly men (32). Studies of BMI in the elderly tend to be confounded by a high degree of comorbidity and cachexia due to terminal illness (33). To overcome this confounding variable, we excluded terminally ill elderly patients; consequently, none of our patients had a BMI < 19 .

Hypothetical protective mechanism of a low body mass index

Although the link with such other cardiovascular disease risks as diabetes and hypercholesterolemia could explain the higher incidence of CAD in obese patients, there is strong evidence for a direct relation between CAD and overweight (19). For example, hyperinsulinemia is a predictive factor for the development of CAD in obese patients (34). Overweight is also associated with increased plasminogen activator inhibitor activity and, therefore, with altered fibrinolytic activity (35). Moreover, 2 trials showed a reduction of thrombolytic therapy efficacy in obese patients with AMI (36, 37). Finally, hypertriglyceridemia is frequent in patients with a high BMI and is an independent risk factor for CAD (38). However, none of the foregoing observations are tenable when the

TABLE 3

Effect of age, BMI, preinfarction angina, and interaction of BMI and preinfarction angina in all patients and according to the quartiles of BMI on in-hospital death, cardiogenic shock, and the combined endpoints of in-hospital death and cardiogenic shock¹

	Death			Cardiogenic shock			Death and cardiogenic shock		
	β	OR (95% CI)	<i>P</i>	β	OR (95% CI)	<i>P</i>	β	OR (95% CI)	<i>P</i>
All patients ²									
Age	0.03	1.03 (1.00, 1.06)	0.04	0.03	1.03 (1.00, 1.06)	0.02	0.02	1.02 (1.00, 1.05)	0.05
BMI	0.11	1.12 (1.01, 1.23)	0.01	0.09	1.10 (1.00, 1.16)	0.03	0.11	1.10 (1.00, 1.22)	0.02
Preinfarction angina	-3.10	0.05 (0.00, 1.38)	0.07	-1.82	0.16 (0.00, 3.37)	0.24	-2.62	0.07 (0.00, 1.22)	0.07
BMI \times preinfarction angina	0.10	1.11 (0.98, 1.25)	0.97	0.06	1.06 (0.94, 1.19)	0.28	0.09	1.09 (0.98, 1.22)	0.09
Preinfarction angina ³									
BMI									
19.0–21.9	-2.75	0.06 (0.00, 0.54)	0.01	-1.91	0.16 (0.02, 0.89)	0.03	-2.10	0.12 (0.07, 0.60)	0.01
22.0–24.9	-0.88	0.41 (0.12, 1.39)	0.15	-1.09	0.33 (0.10, 1.08)	0.06	-0.90	0.40 (0.15, 1.06)	0.06
25.0–26.9	-0.09	0.90 (0.29, 3.08)	0.87	-0.14	0.86 (0.26, 2.82)	0.80	-0.30	0.73 (0.24, 2.17)	0.57
≥ 27	0.44	1.55 (0.70, 3.43)	0.27	0.47	1.61 (0.74, 3.50)	0.22	0.31	1.37 (0.66, 2.81)	0.38

¹OR, odds ratio.


²The model was simultaneously adjusted for age, sex, family history, physical activity, BMI, low educational level, chronic angina, previous acute myocardial infarction, smoking, hypertension, congestive heart failure, diabetes, cholesterolemia, antianginal treatment, thrombolytic therapy, primary angioplasty, and the interaction of BMI and preinfarction angina.

³The model was simultaneously adjusted for the same variables as for all patients, except for BMI.

relation between CAD and overweight is confounded by the variable age. For example, hypercholesterolemia, which is frequently associated with obesity, decreases with age (21).

Experimental and clinical studies have shown that caloric restriction reduces several age-related changes in the cardiovascular system, including arterial baroreflex (39), isoproterenol sensitivity (40), and diastolic dysfunction (41). Recently, it was shown that caloric restriction preserves the protective effect of ischemic preconditioning by restoring norepinephrine release in response to preconditioning ischemic stimulus (17). These data confirm previous results that showed that the norepinephrine content of hearts from food-restricted rats was higher than that of controls (42). Snyder et al (43) established that aging reduces the capacity of cardiac adrenergic nerve terminals to release norepinephrine, and this age-related reduction is abolished by dietary restriction. Therefore, cardiac norepinephrine release in response to the preconditioning stimulus, which is restored by dietary restriction in senescent animals, might be the mechanism by which dietary restriction preserves ischemic preconditioning in the aging heart. Thus, the age-related reduction in the cardioprotective effect of ischemic preconditioning that is restored by caloric restriction might be the experimental counterpart of the protective effect of preinfarction angina preserved in elderly patients with a low BMI.

Conclusion

We confirm a reduced protective effect of preinfarction angina in elderly patients. Moreover, the protective effect of preinfarction angina is preserved in elderly patients with the lowest BMI. This phenomenon may explain why a low BMI protects elderly patients against mortality and morbidity from CAD. 

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