Fasting blood glucose levels are related to exercise capacity in patients with coronary artery disease

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Background and Aim  Previous studies have demonstrated reduced exercise capacity in patients with diabetes mellitus. This study evaluated the relationship between fasting blood glucose (FBG) levels and exercise capacity in patients with coronary artery disease (CAD).

Methods  We evaluated 986 consecutive patients with CAD referred for bicycle spiroergometry combined with gated myocardial perfusion imaging. Maximum oxygen consumption ($V\dot{O}_{2\text{max}}$) and maximal watts were measured. Patients were divided into 4 FBG categories: <100 mg/dL (n = 611), 100 to 109 mg/dL (n = 144), 110 to 125 mg/dL (n = 102), and ≥126 mg/dL (n = 129). Differences in clinical characteristics, exercise hemodynamics, perfusion imaging, and univariate as well as multivariate predictors of exercise capacity were determined.

Results  Maximal watts and $V\dot{O}_{2\text{max}}$ were significantly lower ($P < .0001$) in patients with higher FBG levels and were related to FBG values in univariate and multivariate analyses. Left ventricular volumes and ejection fractions did not differ between the FBG categories. Myocardial perfusion imaging showed a comparable degree of ischemia in the 4 FBG groups. However, patients with higher FBG levels had higher heart rate and blood pressure values at rest resulting in a higher rate-pressure product (values in the 4 FBG groups 8299 ± 2051, 8733 ± 2008, 9558 ± 2583, and 9588 ± 2468 beat/min × mm Hg, $P < .0001$), suggesting increased myocardial oxygen consumption per unit time at rest.

Conclusion  Exercise capacity in patients with CAD is related to FBG levels. Patients with impaired fasting glucose or an FBG level ≥126 mg/dL reached lower peak watts and lower $V\dot{O}_{2\text{max}}$ values. This could be attributed to a higher myocardial oxygen consumption per unit time at rest and the inability to adapt their coronary flow adequately to higher metabolic demands during maximal exercise. (Am Heart J 2006;152:486-92.)

Cardiovascular diseases—including coronary artery disease (CAD)—are the most common cause of death in men <65 years and the second most common cause in women. They are expected to be the main cause of death globally within the next 15 years owing to their rapidly increasing prevalence in developing countries and Eastern Europe and a rising incidence of obesity and diabetes in the western world. Exercise stress testing, often combined with myocardial perfusion imaging, is a validated examination in the follow-up of patients with a history of myocardial infarction or revascularization.

Bicycle testing is not only useful in detecting myocardial ischemia but also provides important information on patients’ exercise capacity and prognosis especially when combined with spiroergometry. Maximum oxygen consumption ($V\dot{O}_{2\text{max}}$) is an independent predictor of mortality particularly in patients with heart failure.

Diabetes mellitus (DM) is associated with a 2- to 4-fold increase in the risk of developing CAD. In patients with known CAD and diabetes, the rate of death is >70% over 10 years. According to the guidelines of the American Diabetes Association, a fasting blood glucose (FBG) level ≥126 mg/dL identifies individuals with diabetes. Fasting blood glucose levels between 110 and 125 mg/dL identify patients with impaired fasting glucose (IFG). Several authors have demonstrated—even in patients with CAD without diabetes—that there is a substantially increased mortality rate among patients with IFG.

The aims of our study were to (1) determine if exercise capacity is related to fasting glucose levels in patients with CAD and (2) investigate potential pathophysiologic mechanisms that could explain this relationship, including remodeling, ischemia, and hemodynamics.
Methods

Study population

Subjects were selected from a database comprising all individuals referred to our department for bicycle stress testing combined with spirometry and single photon emission cardiac tomography (SPECT) from May 2001 through December 2004. From the 1457 patients in the database, we selected 986 with a history of CAD. Patients were classified as having CAD if they had a history of myocardial infarction, a history of coronary revascularization either by percutaneous coronary intervention or by coronary artery bypass grafting, or if they had a significant stenosis on coronary angiography occluding at least 70% of the lumen.

Study protocol

Patients were informed to abstain from caffeine- and nicotine-containing products at least 24 hours before testing and to visit the Exercise Physiology Laboratory after overnight fasting.

Blood sample. A venous blood sample was collected before exercise testing. Serum glucose was assayed by a standard hexokinase enzymatic method. For serum creatinine, a rate-blanked kinetic Jaffe method was used. Glomerular filtration rate was calculated from the following equation: estimated glomerular filtration rate = 186 × (serum creatinine level [in mg/dL])−1.154 × (age [in years])−0.203. For women, this equation was multiplied by a correction factor of 0.742.8 Total serum cholesterol was assayed by the enzymatic colorimetric enzymatic colorimetric, cholesterol esterase/ peroxidase method of Allain et al. Serum high-density lipoprotein cholesterol was determined by the homogenous colorimetric enzymatic colorimetric, cholesterol esterase/ peroxidase method of Allain et al. Serum high-density lipoprotein cholesterol was calculated using the classic Friedewald formula. All serum parameters were determined at 37°C on a Modular system (Roche Diagnostics, Mannheim, Germany). All tests were performed according to an ISO 17025 Beltest accreditation.

Bicycle spiroergometry. Before starting the stress test, we determined the length and weight and calculated the body mass index of the patients. Patients were clinically evaluated by a cardiologist and were classified according to the New York Heart Association functional classes. After resting for 5 minutes, the patients had their arterial blood pressure measured at the right brachial artery with a mercury sphygmomanometer. A 12-lead electrocardiogram was recorded, and resting heart rate (HR) was calculated from the R-R interval.

Each subject underwent maximal exercise testing on a computer-driven bicycle ergometer (Ergoselect, Ergoline GmbH, Bitz, Germany) using a ramp protocol starting at 50 W, with gradual increase of 25 W every 2 minutes in 48% of the patients. In 30% of the patients, the exercise started at 50 W with gradual increase of 10 W every minute; in 18%, it started at 25 W with gradual increase of 10 W every minute. Most of the patients were familiar with the procedure and were encouraged to exercise to exhaustion.

During the test, patients wore a tightly fitting face mask connected to an Oxycon Pro spirometer (Jaeger-Visys Healthcare, Hoechberg, Germany). Oxygen consumption per unit time (\(\text{VO}_2\)), carbon dioxide production (\(\text{VCO}_2\)), and minute ventilation were measured on a breath-by-breath basis. A standard 12-lead electrocardiogram was continuously recorded, and the HR was followed. Blood pressure was measured by means of a mercury sphygmomanometer at each stage and at the peak of exercise.

Subjects were exercised to their self-determined maximal capacity or until the physician stopped the test because of symptoms such as chest pain or dizziness, potentially dangerous arrhythmias or ST-segment deviations, or marked systolic hypotension or hypertension. A respiratory exchange ratio (\(\text{VCO}_2/\text{VO}_2\) >1 was taken to indicate maximal effort. When patients were not able to perform maximal bicycle stress, they received an additional intravenous infusion of dipyridamole (n = 116, 15%). Additional pharmacologic stress testing was also used in individuals with pacing rhythm and left bundle branch block.

Maximum oxygen consumption was defined as the highest \(\text{VO}_2\) obtained at the end of the test and is expressed in mL/min per kilogram. Heart rate reserve was calculated as follows: (peak HR – rest HR)/(220 – age – rest HR) × 100, with values <80% considered as abnormal and indicative of chronotropic incompetence.9 Rate-pressure product (beat/min × mm Hg) was calculated as the product of HR and systolic blood pressure both at rest and at peak exercise.

Technetium-99m tetrofosmin was injected at peak stress or 4 minutes after infusion of dipyridamole. Stress and rest studies were performed in a 2-day protocol as described previously.10 In brief, myocardial perfusion imaging was started 30 minutes after injection at peak stress using a triple-headed camera (Picker Prism 3000, Marconi, Philips, Cleveland, OH). Acquisitions were gated for 8 frames per cardiac cycle. After overnight fasting, patients came to the laboratory and were injected with technetium-99m tetrofosmin. Resting-state myocardial perfusion imaging was started 30 to 60 minutes after injection. Gated images were processed using a Quantified Gated SPECT software (Cedars-Sinai, Los Angeles, CA) to obtain resting left ventricular volumes and ejection fraction.

Scoring of the perfusion images. For perfusion imaging, the raw gated SPECT data were un gated and reconstructed using filtered backprojection (ramp filter) and postfiltered using a low-pass filter (order 5, cutoff frequency 0.21). The left ventricle was reoriented manually to obtain short axis images. Perfusion images were scored by viewing short, vertically long, and horizontally long axis images at stress and at rest. Patients with complete or partial reversible segments were considered having myocardial ischemia.

Patient categories

Patients were divided into 4 categories according to their FBG levels: (1) <100 mg/dL, (2) 100 to 109 mg/dL, (3) 110 to 125 mg/dL, and (4) ≥126 mg/dL.

Statistical analysis

All statistical analyses were performed using SPSS 11.5 statistical software (SPSS Inc, Chicago, IL). Continuous data are shown as mean ± SD; categorical data, as percentages. Analysis of variance was used to compare continuous variables; \(\chi^2\) testing, to compare categorical variables. Rate-pressure products at rest and at peak stress were compared...
with a paired t test. Determinants of maximal reached watts at peak exercise and \( \dot{V}O_2\text{max} \) were evaluated in univariate analysis by means of Pearson’s correlations. Significant parameters in univariate models were entered into a linear regression model to predict maximal reached watts at peak exercise and \( \dot{V}O_2\text{max} \). Significance level was set at .05.

**Results**

**Clinical characteristics**

The patients’ clinical characteristics are shown in Table I according to their FBG levels. Patients with IFG were, on average, a few years older than patients with normal FBG levels. Patients with a FBG level between 110 and 125 mg/dL as well as those with that of \( \geq 126 \) mg/dL had a higher body mass index and had more frequently a history of hypertension. Most patients were in New York Heart Association class I (83.5%).

Revascularization rates were higher in the highest FBG categories. As expected, more patients in the 2 highest glucose categories had a history of DM reported in their medical files; however, DM was diagnosed for the first time in 18 patients with glycemia \( \geq 126 \) mg/dL. Patients with high FBG levels tended to take less \( \beta \)-blockers and were more likely to receive diuretics (mainly thiazides to treat arterial hypertension).

**Hemodynamics at rest and during exercise**

Patients in FBG categories \( \geq 110 \) mg/dL had higher HRs and higher blood pressure values at rest compared with patients with lower FBG levels (Table II). Patients with higher FBG levels performed worse during exercise testing, as can be observed from the lower watts reached at peak exercise (Figure 1) and the lower \( \dot{V}O_2\text{max} \) (Figure 2). Most patients reached their designated maximal effort as indicated by the identical mean respiratory exchange ratio value in each FBG group (Table II). The percentages of patients who needed an additional injection with dipyridamole were 14%, 18%, 13%, and 16%, respectively, in the 4 groups (\( P = \) not significant). Chronotropic incompetence as assessed by means of HR reserve \( \leq 80\% \) was present in all groups, but the average HR reserve was lower among patients with FBG levels \( \geq 110 \) mg/dL. The rate-pressure product (Figure 3) at rest was significantly higher in patients with FBG levels between 110 and 125 mg/dL (\( P < .0001 \)) and in patients with FBG levels \( \geq 126 \) mg/dL (\( P < .0001 \)) compared with patients with

### Table I. Clinical characteristics

<table>
<thead>
<tr>
<th>FBG</th>
<th>n</th>
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<th>n</th>
<th>FBG</th>
<th>n</th>
<th>P</th>
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<td>&lt;100 mg/dL</td>
<td>611</td>
<td>100-109 mg/dL</td>
<td>144</td>
<td>110-125 mg/dL</td>
<td>102</td>
<td>126 mg/dL and</td>
<td>129</td>
<td></td>
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<tr>
<td>Age (y, mean ± SD)</td>
<td>62 ± 11</td>
<td>64 ± 10</td>
<td>66 ± 10</td>
<td>63 ± 10</td>
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<td>Male sex (%)</td>
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<td>81</td>
<td>81</td>
<td>74</td>
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<td>BMI (kg/m², mean ± SD)</td>
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<td>27 ± 4</td>
<td>28 ± 5</td>
<td>28 ± 4</td>
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<td>NYHA class (mean ± SD)</td>
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<td>1.2 ± 0.4</td>
<td>1.2 ± 0.5</td>
<td>1.1 ± 0.3</td>
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<td>4</td>
<td>2</td>
<td>3</td>
<td>NS</td>
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<td>Previous MI (%)</td>
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<td>29</td>
<td>25</td>
<td>24</td>
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<td>Previous revasc (%)</td>
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<td>59</td>
<td>63</td>
<td>67</td>
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<td>Hypertension (%)</td>
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<td>Smoking (%)</td>
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<td>9</td>
<td>11</td>
<td>13</td>
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<td>History of DM (%)</td>
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<td>14</td>
<td>33</td>
<td>86</td>
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<td>FBG (mg/dL, mean ± SD)</td>
<td>0.83 ± 0.09</td>
<td>1.0 ± 0.07</td>
<td>1.13 ± 0.05</td>
<td>1.75 ± 0.52</td>
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<td>GFR (mL/min per 1.73 m², mean ± SD)</td>
<td>77 ± 20</td>
<td>78 ± 20</td>
<td>72 ± 23</td>
<td>74 ± 24</td>
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<td>Total cholesterol (mg/dL, mean ± SD)</td>
<td>197 ± 38</td>
<td>199 ± 40</td>
<td>192 ± 39</td>
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<td>50 ± 12</td>
<td>50 ± 14</td>
<td>48 ± 13</td>
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<td>LDL-C (mg/dL, mean ± SD)</td>
<td>120 ± 34</td>
<td>119 ± 35</td>
<td>113 ± 33</td>
<td>115 ± 33</td>
<td>NS</td>
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<td>Triglycerides (mg/dL, mean ± SD)</td>
<td>134 ± 68</td>
<td>148 ± 72</td>
<td>150 ± 84</td>
<td>199 ± 260</td>
<td>&lt;.0001</td>
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<td>Medication (%)</td>
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<td>Aspirin</td>
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<td>87</td>
<td>77</td>
<td>73</td>
<td>NS</td>
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<tr>
<td>( \beta )-Blocker</td>
<td>54</td>
<td>48</td>
<td>47</td>
<td>38</td>
<td>.05</td>
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<td>ACE-I/AT-II ant</td>
<td>38</td>
<td>41</td>
<td>38</td>
<td>48</td>
<td>NS</td>
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<tr>
<td>Ca ant</td>
<td>11</td>
<td>14</td>
<td>10</td>
<td>18</td>
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<tr>
<td>Diuretics</td>
<td>9</td>
<td>12</td>
<td>11</td>
<td>28</td>
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<td>Statins</td>
<td>34</td>
<td>30</td>
<td>34</td>
<td>30</td>
<td>NS</td>
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<td>Oral antidiabetics</td>
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<tr>
<td>Insulin</td>
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<td>0</td>
<td>0</td>
<td>16</td>
<td>.0001</td>
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</table>

NS, Not significant; BMI, body mass index; NYHA, New York Heart Association; MI, myocardial infarction; Revasc, revascularization/hypertension (blood pressure \( \geq 140/90 \) mm Hg and/or history of hypertension and/or antihypertensive treatment); GFR, glomerular filtration rate; HDL-C, high-density lipoprotein cholesterol; LDL-C, high-density lipoprotein cholesterol; ACE-I, angiotensin-converting enzyme inhibitor; AT-II ant, angiotensin II receptor antagonist; Ca ant, calcium antagonist.
FBG levels <100 mg/dL. It was also significantly higher in patients with FBG levels between 110 and 125 mg/dL (P = .02) and in patients with FBG levels ≥126 mg/dL (P = .001) compared with patients with FBG levels between 100 and 109 mg/dL. The rate-pressure product at peak exercise was significantly higher than the rate-pressure product at rest: P < .0001 (Figure 3). The percentage of increase during exercise of the rate-pressure product was 170% in the FBG <100 mg/dL group, 166% in the FBG 100 to 109 mg/dL group, 142% in the FBG 110 to 125 mg/dL group, and 137% in the FBG ≥126 mg/dL group, resulting in absolute values at peak exercise that did not differ significantly between the FBG categories.

Single photon emission cardiac tomography results

Left ventricular volumes and ejection fraction did not differ significantly between the 4 FBG categories (Table II). In addition, no apparent statistically significant difference could be observed regarding myocardial ischemia as assessed by perfusion between the 4 FBG categories (Table II).
Univariate and multivariate predictors of exercise capacity

Univariate predictors of maximal watts reached at peak exercise and $V\dot{O}_2$ max are reported in Table III.

Multivariate analysis

Linear regression analysis was used to predict maximal watts at peak exercise. Significant parameters in univariate analysis, male sex, $\beta$-blocker treatment, and diuretic treatment were entered into the model. The factors remaining significant in this model ($R^2 = 0.58$, $P < .0001$) are reported in Table IV. Linear regression analysis was also used to predict $V\dot{O}_2$ max. The following parameters were entered into the model: sex, $\beta$-blocker treatment, diuretic treatment, and the significant parameters in univariate analysis. The factors remaining significant in this model ($R^2 = 0.46$, $P < .0001$) are reported in Table IV.

Discussion

In a well-defined population of 986 patients with CAD referred for stress testing combined with gated myocardial perfusion imaging, exercise capacity was related to FBG levels. Maximal watts at peak exercise and $V\dot{O}_2$ max were significantly lower in patients with higher FBG levels.

The strength of our study is that $V\dot{O}_2$ max—in contrast to other studies—was actually measured with spirometry in all 986 participating subjects and not defined by calculation of metabolic equivalents or by estimation of $V\dot{O}_2$ max. Demir et al. studied the relation between metabolic equivalents and hemoglobin.
A1c in 275 diabetes patients without CAD and found a moderate correlation. Hickner et al evaluated the relation between aerobic capacity and categorization of type 2 diabetes in 3700 younger men without known or suspected cardiovascular disease. The VO2 max was calculated from the speed and grade of the treadmill over the final stage of the exercise using reported prediction equations. The VO2 max values were lower in subjects with FBG levels between 126 and 140 and \( \geq 140 \text{ mg/dL} \).

In the present study, patients with IFG (FBG level range 110-25 mg/dL) had lower maximal watts than patients with normoglycemia. An FBG level \( \geq 110 \text{ mg/dL} \) is 1 of the 5 diagnostic criteria of the metabolic syndrome. Spies et al evaluated the association of the metabolic syndrome with treadmill exercise capacity and HR recovery in 943 subjects with known CAD and found that metabolic syndrome was associated with poor exercise capacity and poor HR recovery in patients with CAD. Waist circumference was not available in our database, but it is likely that more patients in the IFG group have metabolic syndrome because triglycerides and blood pressure values were also higher in this group.

The lower exercise capacity of patients with CAD with higher FBG levels could theoretically be attributed to differences in left ventricular dimensions and pump function, ischemia, or differences in baseline and exercise-induced hemodynamics, compared with patients with normoglycemia.

Left ventricular dimensions and ejection fraction were evaluated in all patients by a validated and highly reproducible technique: gated SPECT. No significant difference was observed between FBG categories regarding resting left ventricular end-systolic and end-diastolic volumes or ejection fraction. Ischemia was evaluated with perfusion imaging, and no major difference was observed between the FBG categories. These SPECT findings suggest that the relation between exercise capacity and FBG levels is less likely to be explained by differences in left ventricular remodeling or ischemia.

Hemodynamic parameters did differ between FBG categories, however. Resting HR was significantly higher in patients with an FBG level between 110 and 125 mg/dL as well as in those with that of \( \geq 126 \text{ mg/dL} \) and was independently associated with lower exercise capacity. Patients unable to reach high HRs at peak exercise had a lower exercise capacity. Most of the included patients with CAD had HR reserves \(<85\%\), indicating chronotropic incompetence; however, this was more prominent in the highest FBG categories. The pathophysiologic condition underlying an inadequate HR response to exercise is not well understood; some factors contributing to this include the following: autonomic dysfunction, sinus node disease, and left ventricular dysfunction. Important pathophysiologic information can be derived from the rate-pressure product. This double product, calculated from 2 easily measured hemodynamic variables (HR and systolic blood pressure), is a valid predictor of myocardial VO2 during exercise in patients with ischemic heart disease and offers prognostic information. Interestingly, the rate-pressure product at rest was significantly higher in patients in the upper FBG levels than in patients with normoglycemia. This suggests a higher myocardial VO2 at rest in patients with IFG or an FBG level \( \geq 126 \text{ mg/dL} \). The procentual increase of the rate-pressure product at peak exercise values tended to be lower in patients with IFG and an FBG level \( \geq 126 \text{ mg/dL} \). The inability of CAD patients with elevated FBG levels to adapt their coronary flow adequately to higher metabolic demands during maximal exercise could contribute to their lower exercise capacity.

Our findings could have clinical consequences. First, because FBG is an independent predictor of exercise capacity in patients with CAD, FBG levels should be taken into account when interpreting exercise capacity in terms of prognosis. Second, intense metabolic control and aggressive treatment of hyperglycemia could possibly improve exercise capacity among patients with CAD and this merits further investigation in clinical trials.

Although it is reasonable to assume that higher FBG levels are associated with a lower exercise capacity especially in subjects with IFG and DM, it is also reasonable to inverse this relationship. Wei et al studied 8633 nondiabetic men, determined their cardiorespiratory fitness by a treadmill test, and followed them for 6 years. Subjects with a history of cardiovascular disease were excluded from their study. Level of fitness was defined by total time on the treadmill. Low cardiorespiratory fitness was associated with increased risk for developing IFG and type 2 DM.

Limitations

Information on the duration of DM or on metabolic control (hemoglobin A1c) in patients with DM were not available, but the FBG test, a simple and inexpensive procedure, predicted exercise capacity independently. Sample size was inadequate to allow reliable subanalysis of patients with diabetes. No information was available on pulmonary function, a possible confounding factor in the relation between aerobic capacity and FBG.

Conclusion

In patients with CAD, FBG levels are related to exercise capacity. Patients with an FBG level between 110 and 125 mg/dL as well as those with that of \( \geq 126 \text{ mg/dL} \) reached lower watts at peak exercise and lower VO2 max. Their lower exercise capacity could be attributed to a higher myocardial VO2 at rest and their...
inability to adapt their coronary flow adequately to higher metabolic demands during maximal exercise.

We thank Krista Van Vlaenderen, MEng, for her technical support.

References