Systolic Blood Pressure During Recovery From Exercise and the Risk of Acute Myocardial Infarction in Middle-Aged Men

Jari A. Laukkanen, Sudhir Kurl, Riitta Salonen, Timo A. Lakka, Rainer Rauramaa, Jukka T. Salonen

Abstract—We prospectively assessed the association of systolic blood pressure (SBP) after exercise with the risk of an acute myocardial infarction. Limited information exists currently on the role of SBP during recovery period with the risk of acute myocardial infarction. SBP was measured every 2 minutes during and after a progressive cycle ergometer exercise test in a representative sample of 2336 men (aged 42 to 61 years). During an average follow-up period of 13.1 years, 358 acute myocardial infarctions occurred. An incremental rise of 10 mm Hg per minute in SBP at 2 minutes after exercise (relative risk, 1.07-fold; 95% confidence interval [CI], 1.03 to 1.12; \( P = 0.001 \)) was associated with the risk of acute myocardial infarction after adjustment for age, alcohol consumption, smoking, serum lipids, diabetes mellitus, body mass index, resting SBP, regular use of antihypertensive medications, physical fitness, heart rate, and ischemic ECG findings during exercise. Men with elevated SBP of > 195 mm Hg after exercise had a 1.69-fold (95% CI, 1.24 to 2.30; \( P = 0.001 \)) risk for an acute myocardial infarction compared with those with SBP < 170 mm Hg after adjustment for age, other risk factors, and resting SBP. SBP after exercise provides an incremental predictive value for acute myocardial infarction beyond that of resting SBP. This emphasizes the importance of SBP measurements after the exercise test because it provides additional valuable prognostic measure with regard to acute myocardial infarction.

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Key Words: epidemiology ■ exercise ■ blood pressure ■ myocardial infarction ■ risk factors

Systolic blood pressure (SBP) changes during exercise and recovery period are analogous with blood pressure responsiveness to daily physical stress conditions.\(^1\),\(^2\) An exercise-induced rise in SBP has been found to be a predictor of future hypertension,\(^3\)–\(^6\) left ventricular hypertrophy,\(^7\)–\(^9\) stroke,\(^10\) and cardiovascular disease (CVD) mortality\(^11\)–\(^13\) in apparently healthy people. Some studies have reported that a blunted decline in SBP and elevated SBP after exercise are associated with an increased risk of coronary heart disease (CHD),\(^14\),\(^15\) stroke,\(^10\) and hypertension.\(^6\)

In addition to the traditional observations made during the exercise, those made during recovery period may provide further important prognostic information. In previous studies, the prognostic importance of ST segment depression in exercise electrocardiogram (ECG), delayed slowing of heart rate (HR), and ventricular arrhythmias that appear during recovery are shown to be at least as valuable as the abnormal prognostic variables during exercise.\(^16\)–\(^19\) An abnormal delay in the decrease in HR after exercise is suggested because of inadequate reaction of vagal tone resulting from an increase in activity of the sympathetic nervous system.

Although HR and blood pressure during recovery may reflect cardiovascular reactivity after exercise, the prognostic value of SBP after exercise has not been documented previously with respect to acute myocardial infarction (AMI). We therefore investigated the prognostic significance of SBP response after a standardized cycle ergometer exercise test with regard to risk of future AMI in a population-based sample of men.

Methods

Participants

The analysis was performed with participants of the Kuopio Ischemic Heart Disease Risk Factor Study (KIHD), a longitudinal population-based study designed to investigate risk factors for CVD and related outcomes. The study population is a representative sample of men living in the city of Kuopio and its surrounding rural communities who were 42, 48, 54, or 60 years of age at baseline examinations performed between March 1984 and December 1989. Of 3235 potentially eligible men, 2682 (83%) volunteered to participate in this study. The KIHD was approved by the research ethics committee of the University of Kuopio, and each participant gave written informed consent. The study reported here is based on...
data obtained from 2336 participants who had undergone exercise testing at baseline.

**Exercise Testing**

A maximal symptom-limited exercise test was performed at baseline between 8:00 AM and 10:00 AM using an electrically braked cycle ergometer as described previously.\(^{16,20}\) The standardized testing protocol consisted of a progressive increase in the workload of 20 W per minute. For 605 men (25.9%) examined before June 1986, the testing protocol comprised 3-minute warm-up at 50 W followed by a step-by-step increase in workload by 20 W per minute. The remaining 1731 men (74.1%) were tested with a linear increase in the workload at 20 W per minute. The SBP rise did not differ markedly between the 2 protocols.\(^{10}\) Tests were supervised by an experienced physician with the assistance of a trained nurse. The ECG was registered continuously during the test. The criteria for ischemia in ECG during exercise were horizontal or downsloping ST depression with ≥1 mm at 80 milliseconds after J point or any ST depression of >1 mm at 80 milliseconds after J point.\(^{16}\) HR was recorded by ECG at rest and during the exercise. Maximal oxygen uptake (VO\(_{\text{max}}\)) was defined as the highest value or the plateau of directly measured oxygen consumption using a respiratory gas analyzer (Mijnhardt and Medical Graphics).\(^{20}\)

The most common reasons for stopping the exercise test were leg fatigue (1202 men), exhaustion (362 men), breathlessness (334 men), and pain in the leg muscles, joints, or back (118 men). The test was discontinued because of cardiorespiratory symptoms or abnormalities for 240 men. These included chest pain (81 men), arrhythmias (72 men), a decrease or no increase in SBP or diastolic blood pressure (DBP, 44 men), ischemic ECG changes (32 men), or dizziness (11 men).

**Blood Pressure Determination**

Resting blood pressure was measured by an experienced nurse using a random-zero sphygmomanometer (cuff size 14×54 cm; Hawkinsley) after 5 and 10 minutes of rest in a seated position in a quiet room between 8:00 AM and 10:00 AM 1 week earlier than the exercise test.\(^{21}\) The mean of these 2 values was used as resting blood pressure.

Pre-exercise blood pressure was measured manually when a subject was sitting on the cycle ergometer immediately before the test, and blood pressure was measured every 2 minutes during and after the exercise test using cuff stethoscope method. The maximal SBP was the highest value achieved during the test. Blood pressure was measured during recovery at regular intervals of 2, 4, and 6 minutes with subjects seated on the cycle without pedaling.\(^{10}\) Of these postexercise measurement points, SBP at 2 minutes was selected as the main variable because it was available for all men. The SBP difference between rest and recovery was calculated as SBP at recovery from exercise minus SBP at rest.

**Assessment of Covariates**

Collection of blood specimens, measurement of fasting levels of serum lipids,\(^{22}\) assessment of smoking, alcohol consumption, and physical activity, and the definition of diabetes mellitus\(^{25}\) are described previously. Body mass index (BMI) was computed as weight in kilograms divided by the square of height in meters.

**Ascertainment of Follow-Up Events**

Collection of data on and the diagnostic classification of nonfatal and fatal coronary events by the end of 1992 were performed as part of the multinational WHO MONICA (MONItoring of trends and determinants in CArdiovascular diseases) project, in which detailed information on all CHD events was collected prospectively.\(^{23}\) Regional coronary register teams collected data on coronary events from hospitals and health centers and classified the events on all KIHD participants living in the province of Kuopio, a monitoring area of the Finnish part of the WHO MONICA project (FINMONICA), as explained in detail previously.\(^{23}\) Sources of information included interviews, hospital documents, death certificates, autopsy reports, and medical and legal records. Diagnostic classification of coronary events was based on cardiac symptoms, ECG findings, cardiac enzyme elevations, autopsy findings, and history of CHD.\(^{16,23}\) The FINMONICA coronary register data were annually cross-checked with data obtained from the computerized national hospital discharge and death registers. Data on coronary events from the beginning of 1993 to the end of 2001 were obtained by computer linkage to the national hospital discharge and death certificate registers. Diagnostic information was collected from hospitals and classified using identical diagnostic criteria. If a subject had multiple nonfatal coronary events during the follow-up period, the first event after baseline was defined as the outcome event.

All cardiovascular deaths that occurred between study entry (March 1984 to December 1989) and December 2001 were included. Cardiovascular causes of death were coded according to the Ninth International Classification of Disease (ICD) codes (numbers 390 to 459) and the Tenth ICD codes (numbers 100 to 99).

**Statistical Analysis**

Descriptive data are presented as mean and SDs for continuous data and percentages for categorical data. Correlations between resting blood pressures with SBP after exercise were analyzed using Pearson’s correlation test. Associations of SBP during the exercise test with the risk of AMI and CVD death were analyzed using multivariable Cox proportional hazards models (SPSS 11.5 for Windows; SPSS). To demonstrate the prognostic value of exercise SBP, it was entered with and without resting SBP into Cox multivariable models including age, examination year (1985 to 1989), use of antihypertensive medication (β-blockers, diuretics, angiotensin-converting enzyme inhibitors, or calcium-channel blockers), and other covariates (alcohol consumption, cigarette smoking, serum HDL and LDL cholesterol, serum triglycerides, presence of diabetes, BMI, myocardial ischemia demonstrated on the ECG during exercise, VO\(_{\text{max}}\) and maximal HR). In additional multivariable models, DBP at rest and recovery were also included with these covariates. Covariates were entered as uncategorized into the Cox models, with the exception of the use of medications, diabetes, and exercise-induced myocardial ischemia.

Analysis of tertiles divided the subjects into thirds on the basis of the distribution of SBP after exercise observed in the sample. Tertiles were slightly uneven because SBP after exercise was measured to the nearest blood pressure value. Relative hazards, adjusted for risk factors, were estimated as antilogarithms of coefficients for independent variables. Their confidence intervals (CIs) were estimated under the assumption of asymptotic normality of the estimates. A value of P<0.05 was considered significant. All statistical analyses were performed using SPSS 11.5 for Windows.

**Results**

**Baseline Characteristics**

At baseline, the mean age of the subjects was 52.9 years (SD 5.1; range 42.0 to 61.0). Major baseline characteristics are shown in Table 1. A total of 30% of the men had diagnosed hypertension, of whom 21% were using antihypertensive medications regularly. An average resting SBP was 134 mm Hg (range 89 to 221 mm Hg).

During the symptom-limited exercise test, an average maximal HR during exercise was 155 bpm. Mean maximal SBP and SBP at 2 minutes after exercise were 202 mm Hg and 183 mm Hg, respectively. The summary of the exercise testing data is given in Table 2. Resting SBP had a positive correlation with SBP at 2 minutes after exercise (r = 0.526; P < 0.001). DBP at rest had a slightly weaker positive correlation with SBP during recovery (r = 0.386; P < 0.001). The correlation between maximal SBP and SBP at recovery was 0.599 (P < 0.001).
**TABLE 1. Characteristics of Study Population**

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Mean (SD)</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>52.9 (5.1)</td>
<td>42.0–61.2</td>
</tr>
<tr>
<td>BMI kg/m²</td>
<td>26.8 (3.4)</td>
<td>18.8–40.3</td>
</tr>
<tr>
<td>Smoker, %</td>
<td>31.7</td>
<td></td>
</tr>
<tr>
<td>Cigarette smoking, pack years*</td>
<td>8.5 (16.8)</td>
<td>0–144.0</td>
</tr>
<tr>
<td>Alcohol consumption g/week</td>
<td>75.2 (136.5)</td>
<td>0–2853.0</td>
</tr>
<tr>
<td>Leisure time physical activity kcal/week†</td>
<td>992.3 (1228.3)</td>
<td>0–17448.8</td>
</tr>
<tr>
<td>Mean intensity of reported physical activities, METs†</td>
<td>4.6 (1.2)</td>
<td>2.0–12.5</td>
</tr>
<tr>
<td>Serum total cholesterol, mmol/L</td>
<td>5.90 (1.07)</td>
<td>2.60–10.02</td>
</tr>
<tr>
<td>Serum LDL cholesterol, mmol/L</td>
<td>4.04 (1.01)</td>
<td>0.82–7.89</td>
</tr>
<tr>
<td>Serum HDL cholesterol, mmol/L</td>
<td>1.29 (0.30)</td>
<td>0.52–3.05</td>
</tr>
<tr>
<td>Serum triglycerides, mmol/L</td>
<td>1.29 (0.81)</td>
<td>0.18–7.90</td>
</tr>
<tr>
<td>Resting SBP, mm Hg</td>
<td>133.8 (16.6)</td>
<td>88.7–201.2</td>
</tr>
<tr>
<td>Resting DBP, mm Hg</td>
<td>88.7 (10.3)</td>
<td>58.7–136.8</td>
</tr>
<tr>
<td>Blood fasting glucose, mmol/L</td>
<td>4.8 (1.2)</td>
<td>3.2–16.2</td>
</tr>
</tbody>
</table>

Family history of disease or diagnosed diseases

- Family history of CHD, %: 46.9
- Family history of hypertension, %: 49.2
- Hypertension, %: 30.0
- Congestive heart failure, %: 6.0
- CVD, %: 2.4
- Claudication, %: 4.0
- Atrial fibrillation, %: 1.0
- Diabetes mellitus, %: 5.1
- Regular use of medications
  - Antihypertensive medications, %: 20.9
  - Medications for hypercholesterolemia, %: 0.6
  - \(\beta\)-blockers, %: 16.9
  - Aspirin, %: 6.8

*METs indicates metabolic equivalents of oxygen consumption.

*Pack years denotes the lifelong exposure to smoking that was estimated as the product of years smoked and the number of tobacco products smoked daily at the time of examination.

†Physical activity was assessed using a 12-month leisure-time history modified from the Minnesota Leisure Time Physical Activity Questionnaire to represent the 16 most common conditioning leisure-time physical activities of middle-aged Finnish men.20

**Strongest Risk Factors for AMI**

A total of 358 AMIs occurred during an average follow-up period of 13.1 years (range 0.1 to 17.7 years). The strongest predictors for AMI were smoking, serum LDL cholesterol, and diabetes, followed by serum HDL cholesterol and BMI. \(VO_{\text{2max}}\), SBP after exercise, and exercise-induced myocardial ischemia were significant predictors measured during the exercise test. Maximal HR was a borderline significant \((P=0.05)\) predictor for AMI in the multivariate model. The risk ratios and their 95% CIs for AMI are presented in detail in Table 3. As a continuous variable, 10 mm Hg increment in SBP at 2 minutes recovery from exercise was related to a 1.07-fold (95% CI, 1.03 to 1.12; \(P=0.001)\) risk of AMI after additional adjustment for resting SBP. Furthermore, adjustment for DBP at 2 minutes recovery did not change the observed associations. SBP at recovery was more strongly related to the risk of AMI than DBP at 2 minutes recovery.

**SBP During Recovery and AMI**

SBP at 2 minutes recovery was a significant predictor as a continuous variable as well as classified in tertiles. SBP during recovery in tertiles was related directly to the risk of AMI after adjustment for age and other risk predictors for AMI (Table 4). SBP of >195 mm Hg at 2 minutes recovery was related to a 1.7-fold risk of AMI after adjustment for age, examination year, other risk factors (alcohol consumption, cigarette smoking, serum HDL and LDL cholesterol, serum triglycerides, diabetes, and BMI), resting SBP, use of antihypertensive medications, maximal HR, \(VO_{\text{2max}}\), and myocardial ischemia during exercise. If the adjustment was not made by resting SBP, the respective risk for AMI remained unchanged (RR, 1.68; 95% CI, 1.27 to 2.21; \(P<0.001)\). Kaplan–Meier curves for AMI, according to tertiles of SBP after exercise diverged as the follow-up, continued as shown in the Figure.

**Difference in SBP From Rest to Exercise Recovery and AMI**

The mean difference in SBP from rest to recovery was 53.5 mm Hg (SD 23.7 mm Hg; Table 2). As a continuous variable, 10 mm Hg increment in the difference in SBP from rest to 2 minutes recovery was related to a 1.07-fold (95% CI 1.02 to 1.11; \(P=0.011)\) adjusted risk of AMI. Men with the largest difference in SBP from rest to recovery (≥64 mm Hg, highest tertile) had a 1.39-fold (95% CI, 1.06 to 1.84; \(P=0.019)\) risk of AMI compared with men with lowest difference in SBP (<44 mm Hg, lowest tertile) after adjustment for age, examination year, other risk factors, resting SBP, use of antihypertensive medications, maximal HR, \(VO_{\text{2max}}\), and myocardial ischemia during exercise.
ST depression of downsloping ST depression with calcium-channel antagonist, and angiotensin-converting enzyme inhibitors.

Clinical variables

1.06-fold (95% CI, 1.00 to 1.11; exercise test variables. SBP after exercise was related to a vascular death after adjustment for age and other clinical and SBP during recovery was related directly to risk of cardiovascular death per 10 mm Hg increase in the value. Men with high SBP (≥195 mm Hg) during recovery had a 1.45-fold risk of cardiovascular death compared with men with SBP of <175 mm Hg after adjustment for age, examination year, other risk factors, use of antihypertensive medications, maximal HR, VO_{2max}, and myocardial ischemia during exercise.

Discussion

Elevated SBP after exercise test was related to an increased risk of AMI in a prospective population-based study including men from eastern Finland. This study showed an additional prognostic value of blood pressure during recovery despite taking into account the SBP at rest.

In this study, exercise test predictors including exercise capacity, ST depression during exercise, and maximal HR and SBP after exercise were significant predictors for AMI. SBP during recovery was a strong risk factor, and it was comparable to these previously documented exercise test predictors. It has been suggested previously that abnormal SBP response is an important indicator for coronary artery disease not only during exercise but also during the recovery phase. Some studies observed that an abnormal ratio of recovery to peak exercise was even more sensitive than exercise-induced angina or ST depression for diagnosing the

### TABLE 3. Clinical and Exercise Predictors of AMI

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Relative Risk* (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinical variables</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (per 1 year)</td>
<td>1.01 (0.98–1.04)</td>
<td>0.448</td>
</tr>
<tr>
<td>Smoking (per 10 pack years increment)†</td>
<td>1.18 (1.12–1.25)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>BMI (per 5 kg/m² increment)</td>
<td>1.19 (1.01–1.40)</td>
<td>0.041</td>
</tr>
<tr>
<td>Serum LDL cholesterol (mmol/L)</td>
<td>1.20 (1.09–1.34)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Serum HDL cholesterol (mmol/L)</td>
<td>0.61 (0.40–0.94)</td>
<td>0.024</td>
</tr>
<tr>
<td>Serum triglycerides (mmol/L)</td>
<td>1.01 (0.90–1.13)</td>
<td>0.877</td>
</tr>
<tr>
<td>Diabetes‡ (yes vs no)</td>
<td>2.12 (1.52–2.95)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Alcohol consumption (g/wk)</td>
<td>1.00 (0.99–1.00)</td>
<td>0.479</td>
</tr>
<tr>
<td>Regular use of antihypertensive drugs (yes vs no)§</td>
<td>1.54 (1.18–2.02)</td>
<td>0.002</td>
</tr>
<tr>
<td>Exercise testing variables</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exercise capacity (per 100 mL/min increment in VO_{2max})</td>
<td>0.94 (0.92–0.97)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Exercise-induced ST depression¶</td>
<td>1.52 (1.11–2.09)</td>
<td>0.010</td>
</tr>
<tr>
<td>Maximal HR (per 10 bpm increment)‖</td>
<td>0.95 (0.90–1.00)</td>
<td>0.050</td>
</tr>
<tr>
<td>SBP at 2 minutes recovery (per 10 mm Hg increment)</td>
<td>1.07 (1.03–1.12)</td>
<td>0.001</td>
</tr>
</tbody>
</table>

*Relative risks are derived from multivariable model adjusted for all other factors shown in the table. †Pack years denotes the lifelong exposure to smoking that was estimated as the product of years smoked and the number of tobacco products smoked daily at the time of examination. ‡Diabetes was defined as fasting blood glucose ≥6.1 mmol/L or a clinical diagnosis of diabetes with dietary, oral, or insulin treatment. §Use of antihypertensive medications includes β-blockers, diuretics, calcium-channel antagonist, and angiotensin-converting enzyme inhibitors. ¶Criteria for exercise-induced ST depression in ECG were horizontal or 1 mm at 80 milliseconds after J point. ‖Use of antihypertensive medications includes β-blockers, diuretics, calcium-channel antagonist, and angiotensin-converting enzyme inhibitors.

### TABLE 4. Relative Risks of AMI According to the Tertiles of the SBP During 2 Minutes Recovery of Exercise Test

<table>
<thead>
<tr>
<th>SBP During Recovery</th>
<th>Relative Risk* (95% CI)</th>
<th>P Value</th>
<th>Relative Risk† (95% CI)</th>
<th>P Value</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>First tertile, ≤170 mm Hg (n=816)</td>
<td>1.00 (reference)</td>
<td>1.00 (reference)</td>
<td>99</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second tertile, 170–195 mm Hg (n=761)</td>
<td>1.17 (0.89–1.55)</td>
<td>0.266</td>
<td>1.30 (0.98–1.72)</td>
<td>0.071</td>
<td>112</td>
</tr>
<tr>
<td>Third tertile, &gt;195 mm Hg (n=781)</td>
<td>1.40 (1.04–1.89)</td>
<td>0.028</td>
<td>1.69 (1.24–2.30)</td>
<td>0.001</td>
<td>147</td>
</tr>
</tbody>
</table>

*Relative risks are adjusted for age, examination year, alcohol consumption, cigarette smoking, serum LDL and HDL cholesterol, diabetes, resting SBP, BMI, and use of antihypertensive medications. †Relative risks are adjusted for age, examination year, alcohol consumption, cigarette smoking, serum LDL and HDL cholesterol, diabetes, BMI, resting SBP, myocardial ischemia during exercise, VO_{2max} (L/min), maximal HR, and use of antihypertensive medications.
severity of coronary artery disease.24,25 In patients with angina pectoris, an abnormal SBP response during recovery was very sensitive for the diagnosis of multivessel coronary artery disease, suggesting the close correlation between this response and the severity of CHD.14 Elevated SBP response during the exercise anticipation phase and attenuated SBP responses after exercise were related to increased risk of hypertension6,21 and stroke.10

Consistent with a previous study showing the role of delayed slowing of HR,19 elevated SBP immediately after exercise may also reflect the overactivity of sympathetic nervous system and attenuated vagal reactivation. During graded exercise, HR and SBP progressively increase because of an increase in activity of the sympathetic nervous system with a concomitant decrease in the parasympathetic activity.17,18 Autonomic dysfunction and vasoreactivity abnormalities may account for the gradual decrease of SBP after exercise.6,26 Furthermore, the structural adaptations of the cardiovascular function serve to maintain higher SBP levels and contribute to a structurally induced hyper-reactivity with no decrease in vascular resistance.21,26 An attenuated decrease in exercise blood pressure also may be attributable to poor arterial compliance in individuals with underlying vascular smooth muscle hypertrophy and subclinical arteriosclerotic changes.27

It is also possible that a high preload continued during recovery in patients for high risk of AMI and myocardial ischemia. Thus, an abnormal SBP response may be caused by recovery of myocardial ischemia and an increase in systemic vascular resistance secondary to exaggerated sympathetic nervous activity. Norepinephrine and epinephrine levels increased in response to exercise, and immediately after exercise, both levels have been shown to increase continuously.28 It is biologically plausible that repeated sympathetic activation in the absence of metabolic need leads to SBP elevations beyond the normotensive range.

The rate at which SBP decreases after exercise may be a reflection of a person’s level of physical activity and fitness. The more rapid decline indicates the higher level of physical fitness, and a greater decrease in SBP from peak exercise to the recovery may reflect good aerobic capacity. There is growing evidence that aerobic exercise training via an increase in NO synthase activity can increase the capacity of endothelial cells to evoke vasodilation and decrease exercise blood pressure;29 however, there seems to be heterogeneity in SBP responsiveness to exercise.30 Furthermore, exercise training and increased aerobic capacity can further improve vascular arterial stiffness and vasodilatory capabilities, leading to decreased systemic vascular resistance.29,31 It is also known that physical exercise is independently predictive of the magnitude of the nocturnal changes in blood pressure.32

The strength of our study is that the participation rate was high and there were no losses during follow-up. Our study differs from previous studies17,19 with respect to patient populations, protocols for exercise testing, and end points. This study is based on a high-risk population in an area known for its high prevalence and incidence of atherosclerotic vascular diseases at the time of baseline examination.33,34 We used a standard exercise test protocol with increasing workloads, which has certain advantages compared with treadmill exercise test.2,11,13,24 One of the strengths is that with the cycle ergometer, it is easy to obtain reliable measurements of blood pressure, especially during recovery period. The cycle ergometer usually consists of progressive incremental workloads that may have a minor effect on SBPs achieved between the cycle and the treadmill exercise testing protocols. However, maximal HR has been shown to be lower but the rate pressure product to be similar because of a higher blood pressure in the cycle ergometer than in treadmill test,35 whereas another study showed that rate pressure product may be higher during treadmill exercise compared with cycle exercise.36 The recovery protocols may include cool-down period with walking, lying, or sitting. In this study, subjects were in sitting position during recovery period. It is plausible that the mode of exercise itself may have no, if any, effect on SBP response after exercise is stopped.

This study shows that SBP during the recovery period provides an additional risk marker for identifying asymptomatic individuals at an increased risk for AMI. SBP response after the progressive cycle exercise can be considered a risk predictor for AMI. Thus, SBP measurement immediately after exercise test provides supplementary information for the risk of AMI by comparison with resting blood pressure.

**Perspectives**

Exercise testing is not widely recommended in asymptomatic subjects because of a lack of its prognostic value and false-positive exercise ECG findings. However, in addition to the traditional observations made during the exercise, those made during recovery period may provide further valuable prognostic information. On the basis of previous knowledge, the prognostic importance of ST segment depression, delayed slowing of HR, and ventricular arrhythmias after exercise may be at least as valuable as the abnormal prognostic variables during exercise. SBP after the exercise test provides an additional valuable prognostic measure with regard to AMI, emphasizing the importance of regular SBP measurement during recovery period. Further studies are needed to confirm the role of SBP during recovery as a useful clinical measure with other exercise test variables.

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