Prognostic Significance of Exercise Blood Pressure and Heart Rate in Middle-aged Men

Jan Filipovský, Pierre Ducimetière, and Michel E. Safar

Systolic blood pressure and heart rate measured at rest and during a standardized exercise test were analyzed in the cohort of middle-aged male employees followed-up an average of 17 years in the Paris Prospective Study I. The population sample selected for the analysis included 4,907 men who completed at least 5 minutes of bicycle ergometry, who had no heart disease at entry, and whose resting blood pressure was ≤180/105 mm Hg. Exercise-induced increase in systolic blood pressure was positively correlated with resting systolic blood pressure (r = 0.104, p < 0.0001), whereas the correlation of exercise-induced heart rate increase with resting heart rate was negative (r = −0.169, p < 0.0001). Using Cox regression analysis with the inclusion of resting systolic blood pressure and heart rate; exercise-induced elevations of systolic blood pressure and heart rate; and controlling for age, smoking, total cholesterol, body mass index, electrical left ventricular hypertrophy, and sports activities, cardiovascular mortality was found to be associated with the systolic blood pressure increase (p < 0.05), whereas no association with resting systolic blood pressure was found. Total mortality was predicted by resting systolic blood pressure and its elevation (p < 0.01 for both) and by resting heart rate (p < 0.0001). The heart rate increase did not contribute to death prediction. In conclusion, the magnitude of the exercise-induced increase of systolic blood pressure, but not of heart rate, may represent a risk factor for death from cardiovascular as well as noncardiovascular causes, independently of resting blood pressure and heart rate. (Hypertension 1992;20:333–339)

KEY WORDS • blood pressure • heart rate • exercise test • prospective studies • mortality

Arterial blood pressure (BP) is a strong determinant of life prognosis, and cardiovascular (CV) as well as total mortality are definitely higher in subjects who are classified as hypertensive according to BP measurement at rest. Although relatively less attention has been drawn to heart rate (HR) level, it was also found to be associated with mortality.1 There is a logical tendency to measure these two parameters in basal conditions at rest to achieve maximal standardization; however, it does not imply that this way of assessing BP and pulse rate is the most predictive of individual risk. Exercise testing yields important information about circulatory status and perhaps about life prognosis since it may reflect, with other factors, physical fitness and presence or absence of coronary atherosclerosis.

Exercise tests are widely used in patients in whom coronary heart disease is suspected, and the strong predictive value of pathological electrocardiographic changes, mainly of the ST segment, has been confirmed several times.2-4 Several authors have studied BP and HR changes on physical load in various populations5-7; however, the prognostic value of these parameters in an intact population remains unestablished. Recently, Fagard et al8 published interesting results on the prognostic outcome of direct BP measurement at rest and at several workload levels in 143 hypertensive men. They found no additive prognostic information of exercise BP compared with resting BP in this relatively small group of hypertensive subjects.

The present report represents, to our knowledge, the first study based on a long-term follow-up of a large cohort that evaluates the prognostic significance of noninvasive systolic BP (SBP) and HR measured during bicycle ergometry. Since the majority of drugs that decrease resting BP seem less efficient in lowering BP levels during exercise, the knowledge of mortality risk predicted by exercise BP and HR might be valuable for future views on antihypertensive therapy. The main question raised in the present study is whether the exercise-induced elevations of SBP and HR are risk factors for CV and non-CV mortality in middle-aged normotensive and mild-to-moderate hypertensive men who are free of coronary heart disease at entry. Since all conditions that are related to the development of atherosclerosis are likely to influence circulatory response to exercise, we also studied the association of SBP and HR parameters with smoking, total cholesterol level, obesity expressed by the body mass index (BMI), history of sports activities, and the presence of electrical left ventricular hypertrophy (LVH).
Methods

Study Population

The details about the Paris Prospective Study I protocol are published elsewhere. In brief, 7,746 native-born Frenchmen aged 42–53 years and employed by the Paris Civil Service, were examined for the first time between 1967 and 1972 after giving their informed consent to participate in this study. The screening occurred in the morning, and it consisted mainly of a clinical examination by a cardiologist, a questionnaire concerning health history and smoking habits filled in by a technician, two BP measurements with a standard cuff technique while the subject was in a sitting position after a 5-minute rest, and a fasting blood sample obtained from the subject. Exercise ergometry was performed in 6,565 subjects. Weight and height were measured with the subjects in their underclothes.

The population sample selected for this study included those subjects who completed at least 5 minutes of the standardized bicycle ergometry test and whose resting BP (mean of two measurements) was ≤180/105 mm Hg; more severe hypertensive subjects were a priori excluded from the exercise test. We further excluded treated hypertensive subjects (n=114) and another 162 subjects whose electrocardiographic (ECG) changes during exercise were assessed as positive (horizontal or descending ST segment depression ≥1 mm). Altogether, 4,907 subjects were included in the present analysis.

Exercise Protocol

The standardized protocol of bicycle ergometry has been described previously. Subjects with any of the following conditions were excluded from the test: cardiac insufficiency of any etiology, resting 12-lead standard ECG signs of ischemic heart disease (Minnesota Code was used): definite Q waves, atrioventricular blocks and conduction defects, ST segment abnormalities, atrial fibrillation or flutter, ventricular ectopic beats with polymorphism), resting SBP >180 mm Hg, angina pectoris determined as definite according to the World Health Organization questionnaire, valvular heart disease, evidence of heart enlargement on x-ray film, and any history of cerebrovascular insufficiency. The test consisted of three successive work loads: 2 minutes at 82 W, 164 W from the third to the eighth minute, and finally 191 W up to a maximal 10-minute test duration. The criteria for interruption of the exercise were any chest pain, ECG pathological changes (ST segment abnormalities, i.e., horizontal or descending depression ≥1 mm, atrioventricular blocks and conduction defects, ventricular premature beats), HR >180 beats per minute, SBP ≥250 mm Hg. SBP was measured immediately before the test while the subject was sitting on the ergometer, at the fifth minute of exercise (SBP-5), and immediately at the end of the exercise test while the subject was still sitting on the ergometer. HR was assessed from the electrocardiogram, which was recorded at 2-minute intervals from leads V1 through V5.

Follow-up

During the follow-up period, the administrative department in charge of the population provided annually a list of the deceased subjects. All available data relevant to the causes of death were collected from specific inquiries, i.e., medical records from hospital departments or general practitioners indicated by the relatives of the deceased. The data were then reviewed by an independent medical committee. The eighth revision of the International Classification of Diseases was used for coding. CV deaths include codes N 390–438, 782, and 795. The deadline of the follow-up period was December 31, 1987. The basic information of whether the subject had died or was still alive was obtained for all the members of the cohort. Five hundred and seventy-eight deaths occurred in the population sample of the present study of which 146 were of CV and 337 of non-CV origin; in 95 deaths the cause could not be determined mainly because the family could not be reached at the time of the death report.

Definition of Parameters and Statistical Analysis

In this report, the mean of the two SBP values measured during the screening examination independently of the exercise test is called baseline SBP. Baseline HR is the mean of two measurements, one assessed clinically after one BP measurement and the other from the standard electrocardiogram. Initial SBP and HR are those measured immediately before the test while the subject was sitting on the ergometer. SBP during exercise is that measured at the fifth minute of the test (SBP-5), and HR during exercise is the pulse frequency at the fourth minute of the test (HR-4). The ASBP (ΔHR) differences are those between SBP-5 (HR-4) and the initial parameters. Diastolic BP during exercise was not evaluated since its recording is known to be less reliable. BMI is defined according to the usual formula weight (kg)/height (m)². Smoking is defined as the average daily cigarette consumption during the 5 years preceding the screening. Electrical LVH was assessed according to standard techniques involving the indexes of Sokolow and Lewis. To assess habitual physical activity, only data about sports activities were available. Men who reported participating in sports regularly for 50 hours or more per year at the time of screening were considered active.

Statistical analysis was performed using Statistical Analysis System procedures. Age-adjusted partial correlation coefficients were calculated among SBP and HR measurements. To assess relations with covariates, multiple linear regressions were performed (GLM procedure). The Cox proportional hazards model was used to analyze mortality (PHREG procedure). In all models, age was systematically introduced as an independent continuous variable. Age-adjusted relative risks among various groups are reported.

Results

Basic characteristics of the population sample, including age, BP and HR parameters, total cholesterol level, smoking, and BMI, are presented in Table 1.

Cross Correlations of Systolic Blood Pressure and Heart Rate

In Table 2, the age-adjusted partial linear correlation coefficients between all the considered SBP and HR values are indicated. There is a high correlation be-
TABLE 1. Characteristics of 4,907 Men, Followed-up in the Paris Prospective Study I, Who Completed at Least 5 Minutes of a Standardized Exercise Test

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>47.5±1.9</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td>133.8±14.5</td>
</tr>
<tr>
<td>Baseline SBP</td>
<td>77.5±10.3</td>
</tr>
<tr>
<td>Initial SBP</td>
<td>135.1±16.4</td>
</tr>
<tr>
<td>SBP-5 minutes</td>
<td>195.9±31.7</td>
</tr>
<tr>
<td>ΔSBP</td>
<td>60.8±25.5</td>
</tr>
<tr>
<td>HR (min⁻¹)</td>
<td>66.6±8.6</td>
</tr>
<tr>
<td>Baseline HR</td>
<td>73.7±13.0</td>
</tr>
<tr>
<td>Initial HR</td>
<td>10.0±10.1</td>
</tr>
<tr>
<td>HR-4 minutes</td>
<td>75.6±13.0</td>
</tr>
<tr>
<td>ΔHR</td>
<td>-0.030,-0.094, respectively</td>
</tr>
<tr>
<td>Total cholesterol (mg/dl)</td>
<td>221.4±41.2</td>
</tr>
<tr>
<td>Smoking (No. per day)</td>
<td>149.3±15.1</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>26.0±3.0</td>
</tr>
</tbody>
</table>

SBP, systolic blood pressure; Baseline SBP, DBP, and HR, means of two casual measurements; Initial SBP and HR, measurements taken immediately before the test while the subject was sitting on the ergometer; SBP-5 minutes and HR-4 minutes, measurements taken during the test; ΔSBP and ΔHR, difference between measurements taken during the test and the initial measurements; DBP, diastolic blood pressure; HR, heart rate; smoking, average daily cigarette consumption in the 5 years preceding the screening; BMI, body mass index. Values are mean±SD.

As far as the mutual relations of SBP and HR are concerned, the strongest correlation was between baseline SBP and HR (r=0.244), whereas the correlations between corresponding initial, exercise, and Δ values decreased progressively (r=0.184, 0.120, and 0.088, respectively).

 Associations of Systolic Blood Pressure and Heart Rate with Age, Smoking, Total Cholesterol, and Body Mass Index

Multiple linear regression analyses were further performed (Table 3) in which the SBP and HR parameters were successively dependent variables on age, smoking, total cholesterol level, and BMI. The results are expressed as partial correlation coefficients. Age was positively related to baseline SBP, SBP-5, and ΔSBP (r=0.377, 0.350, and 0.201, respectively). There was no significant correlation of smoking with baseline SBP, but slightly positive associations of smoking with SBP-5 and ΔSBP were found (r=0.050, r=0.056). Total cholesterol level was mainly related to baseline SBP (r=0.117); the positive association of BMI was stronger with baseline SBP than with SBP-5 (r=0.232, r=0.121, respectively) and only a weak positive association with ΔSBP existed (r=0.032).

Age was negatively associated with baseline HR and more markedly with HR-4 (r=-0.030, r=-0.094, respectively). Although smoking was positively related to baseline HR (r=0.071), its association with HR-4 and with ΔHR was negatively significant (r=-0.090, r=-0.107, respectively). Elevated total cholesterol level was slightly associated with both increased baseline HR and HR-4; BMI was positively associated with baseline HR (r=0.042), but there was a much stronger negative association with HR-4 and ΔHR (r=-0.155, r=-0.204, respectively). Altogether, age, smoking, and total cholesterol and BMI levels explain a relatively large proportion of the SBP variance (23% and 16%, respectively, for baseline SBP and SBP-5), but no more than 5% for ΔSBP and HR parameters.

TABLE 2. Age-Adjusted Partial Correlation Coefficients of Systolic Blood Pressure and Heart Rate at Rest and During Exercise

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Systolic blood pressure</th>
<th>Heart rate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline</td>
<td>Initial</td>
</tr>
<tr>
<td>SBP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Initial</td>
<td>0.774</td>
<td></td>
</tr>
<tr>
<td>5 Minutes</td>
<td>0.482</td>
<td>0.537</td>
</tr>
<tr>
<td>Δ</td>
<td>0.104</td>
<td>0.033</td>
</tr>
<tr>
<td>HR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline</td>
<td>0.244</td>
<td>0.216</td>
</tr>
<tr>
<td>Initial</td>
<td>0.176</td>
<td>0.184</td>
</tr>
<tr>
<td>4 Minutes</td>
<td>0.079</td>
<td>0.101</td>
</tr>
<tr>
<td>Δ</td>
<td>-0.069</td>
<td>-0.055</td>
</tr>
</tbody>
</table>

SBP, systolic blood pressure; HR, heart rate; baseline SBP and HR, means of two casual measurements; initial SBP and HR, measurements taken immediately before the test while the subject was sitting on the ergometer; SBP-5 minutes and HR-4 minutes, measurements taken during the test; Δ, difference between measurements taken during the test and the initial measurements.

r/>0.054: p<0.0001.
r/>0.045: p<0.001.
r/>0.032: p<0.05.
Analysis of Systolic Blood Pressure and Heart Rate According to Left Ventricular Hypertrophy

Table 4 gives the SBP and HR mean levels according to the presence or absence of electrical LVH. The presence of electrical LVH is associated with higher SBP both at baseline and during exercise (p < 0.0001, p < 0.001, respectively) but with lower HR at baseline and during exercise (p < 0.05, p < 0.01, respectively). Neither the SBP nor the HR changes are significantly associated with electrical LVH.

Table 5, the analysis of SBP and HR according to sports activities is shown. Men participating in sports regularly at the time of screening had lower baseline, exercise, and ASBP and baseline HR (p < 0.0001 for all), but HR-4 and ΔHR were not significantly different.

Mortality Experience

To analyze mortality, we used Cox proportional hazards models that included age, baseline SBP, baseline HR, ΔSBP, ΔHR, smoking, total cholesterol level, BMI, LVH, and sports activities as explanatory variables (Table 6). With the exception of LVH and sports activities for which only the presence or absence of the defined criteria were considered, all the other variables were introduced on a continuous scale. ΔSBP was significantly associated with CV mortality (p < 0.05), ranging behind smoking and total cholesterol level (p < 0.01 for both), whereas baseline SBP was not significant. Baseline HR just failed to reach statistical significance. There was a significant association of ΔSBP with non-CV mortality as well (p < 0.05), and it ranged behind smoking, baseline HR (p < 0.0001 for both), and total cholesterol level (p < 0.01), the association with the latter factor being negative. Total mortality, including deaths from unknown causes, was related both to baseline SBP and its exercise-induced increase (p < 0.01 for both); the most predictive factors were smoking and resting HR. ΔHR is not significantly associated with any end point.

In Figure 1, relative risks of CV mortality according to SBP-5 (panel A) and to baseline SBP (panel B), controlled for other variables, are shown. Relative risk increased with exercise SBP but did not rise with baseline SBP, when each was adjusted for the level of the other.
TABLE 6. Cox Regression Analysis of Cardiovascular, Noncardiovascular, and Total Mortality by Resting Systolic Blood Pressure and Heart Rate and Their Increase During Exercise Testing With Adjustment for Age, Smoking, Total Cholesterol, Body Mass Index, Electrical Left Ventricular Hypertrophy, and Sports Activities

<table>
<thead>
<tr>
<th>Measurement</th>
<th>CV (n=135)</th>
<th>Non-CV (n=323)</th>
<th>Total (n=458)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline SBP</td>
<td>0.6</td>
<td>3.8</td>
<td>8.8*</td>
</tr>
<tr>
<td>ΔSBP</td>
<td>3.8†</td>
<td>3.9†</td>
<td>7.2*</td>
</tr>
<tr>
<td>HR</td>
<td>Baseline HR</td>
<td>3.5</td>
<td>16.2‡</td>
</tr>
<tr>
<td>ΔHR</td>
<td>0.0</td>
<td>0.7 (-)</td>
<td>0.1 (-)</td>
</tr>
<tr>
<td>Smoking</td>
<td>10.7*</td>
<td>37.9‡</td>
<td>63.3‡</td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>7.7*</td>
<td>8.3* (-)</td>
<td>2.3 (-)</td>
</tr>
<tr>
<td>BMI</td>
<td>1.8</td>
<td>0.2 (-)</td>
<td>0.4</td>
</tr>
<tr>
<td>LVH</td>
<td>1.2</td>
<td>0.3</td>
<td>1.4</td>
</tr>
<tr>
<td>Sports activities</td>
<td>0.1</td>
<td>0.4 (-)</td>
<td>0.1 (-)</td>
</tr>
</tbody>
</table>

Values of χ² (one degree of freedom) are given. All the independent variables were included on a continuous scale except left ventricular hypertrophy and sports activities. CV, cardiovascular; n, number of deaths; SBP, systolic blood pressure; baseline SBP and HR, means of two casual measurements; ΔSBP and ΔHR, difference between measurements taken during the test and the initial measurements; smoking, average daily cigarette consumption in the 5 years preceding the screening; HR, heart rate; (-), parameter estimate is negative; BMI, body mass index; LVH, left ventricular hypertrophy. Twenty-five deaths (11 of CV and 14 of non-CV origin) could not be included due to some missing measurements.

Discussion

The major aim of the present study was to evaluate the prognostic significance of SBP and HR measured during exercise in middle-aged men. The population sample was selected according to several criteria: 1) the mean of two resting BP measurements ≤180/105 mm Hg, 2) an exercise test duration of at least 5 minutes, and 3) no ischemic heart disease at entry detected either at rest or during the exercise test. Consequently, the cohort under study represents a particularly low-risk group, and the results might not be applicable to unselected populations. This is evidenced by the lower all-cause mortality during the follow-up of this sample (11.8%) compared with the mortality of the whole population followed-up in the Paris Prospective Study I (16.8%).

Although all deaths that occurred in the population were reported from administrative files, the knowledge of the cause of death required multiple sources of information originating from a questionnaire mailed to the family of the deceased since access to the death certificate is not available in France. Thus, mainly due to address changes, causes of death could not be elucidated in 95 cases because private doctors and hospital departments could not be contacted. Therefore, the associations of CV and non-CV mortality with factors under study might be substantially underestimated, but this methodological limitation is not likely to bias our results severely. The proportion of CV mortality on all deaths from known cause in our cohort (30.7%) roughly corresponds to the proportion of CV mortality in French national statistics in 1981 (28.3%, after adjustment for age at death). Moreover, when we compared the Cox model for total mortality, which included all deaths that occurred (Table 6), with the one restricted to cause-specific total mortality only (data not shown), virtually identical results were obtained.

Both BP and HR increased during exercise, but this evidence should not be misinterpreted. Actually, although ΔHR is negatively correlated with baseline HR (r = -0.169), the correlation of ΔSBP with baseline SBP is weakly positive (r = 0.104; Table 2). In any case the "regression towards the mean" statistical phenomenon cannot be invoked since changes during exercise are measured from an initial measurement, which is different from baseline. The HR increase seems in accor-

FIGURE 1. Line graphs show relative risks (RR) of cardiovascular mortality according to exercise systolic blood pressure (SBP) (panel A), and baseline SBP (panel B). The relative risks are calculated from the coefficients of one Cox regression model in which baseline SBP, exercise SBP, age, baseline heart rate, smoking, total cholesterol, body mass index, left ventricular hypertrophy, and sports activities are included as independent variables. Mean values of SBP in each SBP class were used, and the class containing the median value (=190 mm Hg for exercise SBP, =130 mm Hg for baseline SBP) is chosen as the reference group (RR=1).
dance with the “law of initial value,” which stipulates that the higher the initial level of the parameter under study, the smaller the reaction on stimulus. This does not apply to BP change since subjects with elevated resting SBP have on the average higher ΔSBP. In some hemodynamic studies, it has been demonstrated that subjects with elevated baseline vascular resistance present a further exaggerated increase on various symptomatic stimuli. This fact is usually related to structural changes of the vascular bed of subjects with chronic BP elevation, i.e., hypertrophy of the vessel wall, and in these subjects, physical exercise would trigger an excessive rise in peripheral vascular resistance. Thus, we hypothesize that the different prognostic significance of ΔSBP and ΔHR observed in our study is related to the different characters of BP and HR elevation that roughly reflect mechanical and neurohumoral responses to exercise.

The positive independent partial correlations of SBP with age, BMI, and total cholesterol (Table 3) are consistent with other studies, but there is no relation of resting SBP with smoking; however, a slightly negative correlation is usually found within populations. This might be partly due to the adjustment with the other factors, especially BMI, in the present study, but also to the particular smoking rating used here that integrates tobacco consumption during the preceding 5 years. The SBP rise during exercise is, as expected, significantly higher in smokers, and a lesser compliance of large arteries in smokers might account for the steeper BP elevation during exercise.

Total, CV, and non-CV mortality experiences in the cohort were shown to be related to the SBP increase during exercise independently of the baseline SBP level. CV deaths, however, seem to be more strongly related to ΔSBP than to baseline SBP, which was no longer statistically significant in this analysis. This result is at variance with the conclusion of Fagard et al, who evaluated the prognostic significance of exercise BP in hypertensive men. Brachial artery BP levels measured at 50 W, peak work load, and 50% peak work were predictive of both total mortality and CV event rates (fatal and nonfatal) but did not add any prognostic precision to resting BP when age was taken into account. Obviously no definite explanation may be given for such a discrepancy that concerns completely different populations and methods; different exercise protocols, in particular, might be important. In the work of Fagard et al, the initial work load of 20 W was augmented successively by 30 W every 4 minutes. This relatively slow rise is usual in the majority of clinical studies and is likely to simulate changes of the physical charge in every day life. Conversely, in the protocol used in the Paris Prospective Study, the work load imposed on the subjects increased very sharply: after 2 minutes at 80 W, the work load was increased to 164 W without interruption. The heart load is thus far greater, and it is possible that SBP increase in these conditions reflects not only structural changes of peripheral vessels but the status of the whole CV system in subjects with elevated resting BP. Therefore, this type of exercise might improve the prognostic power of exercise-induced BP in comparison with more physiological protocols.

An exaggerated SBP increase during such a demanding exercise test might also be partly the consequence of poor physical fitness, which has been shown to be an independent risk factor of mortality when measured by the duration of a maximal treadmill test. In our current study, a positive history of sports activities at the time of screening was associated with lower baseline SBP and HR, SBP-5, and ΔSBP. These relations might explain the beneficial effects of physical fitness on life prognosis. However, for a more precise evaluation of habitual physical activity, more detailed data would be needed.

Men with electrical LVH had a higher resting BP but not a higher ΔSBP at exercise, and they were not at higher risk of death after adjusting for the other factors. The last finding differs from the well-known observations of the Framingham study. The particular population selection for our analysis, namely, excluding the severe hypertensive subjects and subjects with overt coronary heart disease or heart enlargement as shown on x-ray film, is very likely to account for this lack of association.

As found in some other cohort studies in both men and women, resting HR might be a potent independent risk factor for mortality, especially of non-CV origin. Explanations for these associations have not been established yet. Elevated resting HR might merely indicate a low level of physical activity and fitness, that may predispose an individual to CV and possibly to other chronic diseases. In two of three Chicago cohort studies, resting heart rate was significantly associated with sudden death and with non-CV mortality. Concerning CV risk, several specific mechanisms have been proposed: according to animal studies, high HR enhances myocardial oxygen consumption and therefore might be implied in the development of coronary atherosclerosis. Increased HR may be the result of enhanced neural activity that predisposes an individual to a lowered threshold for ventricular fibrillation. On the other hand, subjects with elevated HR seem to be prone to death from cancer. The similarity of the HR risk factor patterns for both CV and non-CV mortality in the present study might indicate that more general mechanisms are involved.

In conclusion, the present cohort study showed that an exaggerated systolic pressure but not a higher ΔSBP at exercise contributes independently to the mortality risk. Further studies are needed to validate these data and to establish to what extent the predictive power of exercise BP is dependent on the type of exercise protocol.

Acknowledgments


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