Cardiovascular and Plasma Catecholamine Responses to Exercise in Blacks and Whites

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The purpose of the present study was to assess possible racial differences in cardiovascular and plasma catecholamine responses to dynamic exercise. A biracial group of normotensive college-age men (15 blacks, 15 whites) were tested for maximal oxygen uptake, resting blood pressure, and heart rate. Subjects then rode a cycle ergometer at 25%, 50%, and 75% of peak oxygen uptake (6 minutes at each stage). Blood pressure and heart rate were measured during supine rest, seated rest, and at each stage of exercise with an automated blood pressure monitor. At each stage, venous blood was sampled to allow determination of plasma norepinephrine and epinephrine, and cardiac output was measured with the carbon dioxide rebreathing technique. The results indicated that resting blood pressure was similar for blacks and whites (114/68 versus 115/68 mm Hg, respectively). Blacks exhibited greater systolic and diastolic blood pressures during submaximal dynamic exercise. However, blacks also showed a trend toward a positive parental history of hypertension, which has been associated with an increased pressor response. Racial differences did not exist for heart rate or cardiac output, but blacks had higher values for total peripheral resistance both at rest and during exercise. Although no overall racial differences were seen for plasma catecholamine concentrations at rest, blacks had significantly lower levels of norepinephrine (1,275 versus 1,556 pg/ml) and higher levels of epinephrine (306 versus 216 pg/ml) than whites at the highest work rate. The current study confirms the increased pressor response to exercise in normotensive blacks. Blacks had an elevation in total peripheral resistance that was not accompanied by an increase in plasma norepinephrine levels. (Hypertension 1992;20:542–548)

KEY WORDS • blood pressure • exercise • heart rate • cardiac output • negroid race • norepinephrine • epinephrine

Hypertension is one of the most prominent health issues facing Americans in this century.1 Reports from the Hypertension Detection and Follow-up Program study2 and a recent update from the National Health and Nutrition Examination Survey3 place the incidence of essential hypertension among blacks at approximately twice that of whites. In addition, mortality and morbidity from hypertensive cardiovascular disease are elevated in the black population. Black Americans have increased rates of myocardial infarction, stroke, and renal disease and failure6,5 compared with whites.

Recently a great deal of research has focused on the possible mechanisms responsible for the elevated incidence of essential hypertension among blacks in the United States. Although a specific cause for essential hypertension in any race has not been determined, it is likely that the mechanism is related to a variety of variables rather than any one source.6,7 Possible contributors to racial differences in hypertension include disparities in renal physiology,8,9 potassium and calcium intake,5,10,11 endocrine factors,12,13 autonomic nervous system function,14 and socioeconomic status.7,15

Racial differences in hypertension may also be related to the finding that blacks have an increased cardiovascular response to dynamic exercise. An exaggerated blood pressure response to dynamic exercise is associated with an increased risk of future hypertension.16 In a recent review Jette et al17 concluded that there is a twofold to 10-fold increase in the risk of future hypertension for hyperresponders. Additionally, borderline hypertensive individuals18–21 and normotensive offspring of hypertensive parents22,23 exhibit exaggerated blood pressure (BP) responses to exercise.

Several studies have documented an exaggerated BP response to exercise in normotensive black children24,25 and black adults.26,27 The mechanism of the increased BP response in blacks is uncertain. However, blacks have higher BP values than whites during exercise without accompanying increases in heart rate (HR), suggesting an elevation in peripheral resistance.

The present study examined the physiological responses to a graded exercise test on a cycle ergometer in normotensive blacks and whites. The hypotheses were 1) that the increased BP reactivity of blacks during dynamic exercise is related to an increased total peripheral resistance (TPR) rather than an increased cardiac output and 2) that blacks would have an increased plasma norepinephrine response to exercise.
Methods

Subjects

Subjects were a biracial group of 30 normotensive, college-aged men (15 whites, 15 blacks) who volunteered to participate in the study. Volunteers were solicited from the university campus and surrounding community by newspaper articles and posters. Prospective subjects were screened on the basis of race (using self-definition) and resting BP in the normotensive range. They then completed a health questionnaire and signed an informed consent form under a protocol approved by the University of Tennessee Committee on Research Participation.

BP and HR were measured in the right arm with the Colin STBP 780 automated blood pressure monitor (Colin Medical Instruments, Plainfield, N.J.) after 20 minutes of supine rest. The Colin monitor has previously been validated against auscultation and intraarterial recordings during rest and exercise.28 A mercury sphygmomanometer was connected to the cuff, and a stereo headset was used by the investigator to simultaneously monitor the STBP 780 recordings. Subjects were excluded from the study if, during the initial screening or on the two subsequent test days, any of their supine resting BP values were greater than 140 mm Hg systolic BP (SBP), 90 mm Hg diastolic BP (DBP), or both.

Anthropometric measurements were taken on each subject, including height, weight, and sum of skinfolds. Skinfold thicknesses were measured at three sites (chest, abdomen, subscapular) with Harpenden calipers.29 Subjects were given a questionnaire on parental history of hypertension,30 which they returned at a later date.

Peak Oxygen Uptake Test

The subjects performed a maximal, graded exercise test on a cycle ergometer. Each subject began by exercising at 30 W for 1 minute, and the work rate was continually increased by 30 W each minute until the subject was unable to sustain the pedal cadence. Minute ventilation was measured by a calibrated RAM 9200 (Rayfield Airflow Meter, Rayfield Equipment, Waitsville, Vt.). Percentages of expired oxygen (O₂) and carbon dioxide (CO₂) were measured with Applied Electrochemistry S3-A O₂ and CD-3A CO₂ analyzers (Sunnyvale, Calif.) connected to a computerized system. These analyzers were calibrated against known gases analyzed by the Scholander technique. Oxygen uptake (VO₂) and carbon dioxide production (VCO₂) were calculated using the Haldane transformation of the Fick equation. Additional criteria for subjects in the study included a peak oxygen uptake (VO₂peak) value in the range of 40-55 ml·kg⁻¹·min⁻¹.

Dynamic Exercise

For the dynamic submaximal exercise test, the subjects reported to the lab in the early morning before breakfast, strenuous physical activity, or caffeine. After 15 minutes of supine rest, a Teflon catheter was inserted into the antecubital vein to allow for multiple blood sampling throughout the exercise test. After the insertion of the catheter and an additional 15-minute rest period, a supine resting blood sample (3 ml) was taken.

The trial began with 6 minutes of seated rest with the subject in position on the cycle ergometer. The subject’s VO₂ and VCO₂ were measured at 30-second intervals, and the value obtained at minute 4 was determined to be the steady-state value. A 3-ml venous blood sample was then obtained, and measurements of cardiac output were completed by minute 6, when the subject was taken to the next stage. The subjects performed a continuous submaximal exercise test at 25%, 50%, and 75% VO₂peak (6 minutes per stage).

Cardiac output was measured noninvasively by the CO₂ rebreathing technique of Collier.31 End-tidal partial pressure of CO₂ (Pco₂) was measured through the use of a rapid-response infrared CO₂ analyzer. The 10 end-tidal Pco₂ values before a rebreathing trial were averaged, and this value was used to estimate arterial Pco₂ according to the formula of Jones et al.32

The subject then rebreathed into a latex bag containing CO₂ and O₂ to permit rapid equilibration with venous Pco₂. A valid equilibrium during rebreathing was determined by a “plateau” in Pco₂ according to the criteria of Ashton and McHardy33 and Jones et al.34 This requires that during the first 6-8 seconds, there is an inspiration followed by an expiration in which the Pco₂ values are within ±1 mm Hg of each other. In most cases, a plateau was obtained in 6-8 seconds without having to repeat a trial. The “downstream” correction factor of Jones35 was used. An automated gas mixing apparatus was used to adjust the initial gas volume and initial Pco₂ in the rebreathing bag, according to the procedures described by Jones.35 If a trial had to be repeated, the end-tidal Pco₂ values were allowed to return to baseline values before redoing the procedure.

Arterial and venous CO₂ content (CaCO₂ and CVCO₂) were determined using a standard dissociation curve. Cardiac output was calculated from the indirect Fick equation:

\[ CO = \frac{VCO₂}{(CaCO₂ - CVCO₂)} \]

where CO is cardiac output. Mean arterial blood pressure (MABP) was calculated from the formula:

\[ MABP = DBP + \frac{1}{3}(SBP - DBP) \]

TPR was calculated from the formula:

\[ TPR = \frac{MABP}{CO} \]

where TPR is expressed as mm Hg·l⁻¹·min⁻¹, MABP as mm Hg, and cardiac output as l·min⁻¹.

Blood was sampled five times during the submaximal exercise test (supine rest, seated rest, 25%, 50%, and 75% VO₂peak) before CO₂ rebreathing. Blood samples for epinephrine and norepinephrine analysis were placed in cold tubes containing ethyleneglycol-bis (β-amino-ethyl ether)-N,N′-tetracetate acid and glutathione to give a final concentration of 1.8 and 1.2 mg/ml blood, respectively. The plasma was separated and stored frozen (−70°C) until analysis. The epinephrine and norepinephrine samples were measured in duplicate by a modification of the radioenzymatic technique of Cryer et al.36 Catechol-O-methyl transferase was extracted from rat liver by the method of Axelrod and Tomchick.37 Tritium-labeled 5-adenosyl methionine was purchased from New England Nuclear Research Products, Du Pont Company, Boston, Mass. All samples...
from a subject were analyzed within a single assay, and seven or eight pairs (white/black) of subjects were analyzed in each assay. An internal standard (100 pg) for epinephrine and norepinephrine were run with each sample. The coefficient of variation for the internal standards averaged 9.3% for epinephrine and 10.9% for norepinephrine.

### Data Analysis

Means and standard errors were calculated for all variables under each condition. An unpaired Student’s t test was used to compare values for the subjects’ physical characteristics (height, weight, sum of skinfolds, age, and resting SBP and DBP). Parental history of hypertension was analyzed by a χ² nonparametric analysis.

A two-way analysis of variance (ANOVA) with repeated measures in one direction was used to analyze HR, SBP, and DBP data for the VO2peak test. When appropriate, a test for simple main effects was used to determine if there were significant racial differences for HR, SBP, and DBP. An unpaired Student’s t test was used to test for differences in VO2peak (1·min⁻¹) and VO2peak (ml·kg⁻¹·min⁻¹).

A two-way ANOVA (race x intensity) with repeated measures on the second factor was used to analyze hemodynamic and catecholamine data for measures obtained during preexercise rest and at each exercise intensity. A significant interaction effect was used to indicate a racial difference in the blood pressure response to exercise. Where appropriate, tests for simple main effects were used to determine if there were significant differences between blacks and whites at each intensity.

### Results

#### Descriptive and Physical Characteristics of Subjects

Characteristics of the 30 normotensive subjects (15 blacks and 15 whites) are shown in Table 1. The Student’s t test indicated no significant differences between group means for blacks and whites on the selected physical variables. Of those subjects able to report on parental history of hypertension, the incidence of a positive parental history of hypertension was not significantly higher for blacks (8 of 14) than for whites (4 of 14), p = 0.085.

### Peak Oxygen Uptake Test

Results of the VO2peak tests are shown in Table 2. Mean scores for VO2peak were indicative of average values for their age group (blacks M=44.1 ml·kg⁻¹·min⁻¹; whites M=47.1 ml·kg⁻¹·min⁻¹). No significant differences were found for absolute VO2peak (1·min⁻¹), VO2peak relative to body mass (ml·kg⁻¹·min⁻¹), or in maximal mean HR values. Mean values for exercise intensity SBP during the VO2peak test were not significantly different between blacks and whites. Although DBP values demonstrated a trend toward being higher for being black subjects, the groups were not significantly different (p<0.06). The 2x2 (race x intensity) repeated-measures ANOVA for HR, SBP, and DBP responses (rest to maximal effort) during the VO2peak tests failed to demonstrate a racial difference in BP response from rest to maximal effort.

#### Dynamic Exercise

As shown in Table 3, blacks had higher blood pressures during the graded, submaximal exercise test. Racial differences in SBP (p<0.0001) and DBP (p<0.002) values were statistically significant. Tests for simple main effects showed that racial differences occurred at 50% and 75% VO2peak for SBP and 25%, 50%, and 75% VO2peak for DBP. Subjects with a positive parental history of hypertension had a greater increase in DBP from rest to 75% VO2peak (from 66 to 88 mm Hg) compared with those with a negative parental history of hypertension (from 69 to 79 mm Hg) (p<0.02). There was no significant difference in the SBP response in subjects with a positive (from 114 to 192 mm Hg) and a negative (from 115 to 186 mm Hg) parental history of hypertension.

Overall, HR and cardiac output were not significantly different for blacks and whites. There was a racial difference in TPR, with blacks having greater overall peripheral resistance than whites (p<0.01). Neither cardiac output nor TPR demonstrated a significant interaction (race x intensity). Since a number of the subjects did not attain a steady state at the highest work rate (75% VO2peak), cardiac output and TPR were not computed for this exercise.

Epinephrine and norepinephrine responses to dynamic exercise are presented in Figure 1. Plasma catecholamine values were not different between blacks and whites at rest. However, blacks had higher plasma epinephrine (p<0.05) and lower plasma norepinephrine (p<0.02) levels overall compared with whites. A test of simple main effects showed a significant racial difference in epinephrine and norepinephrine (p<0.05) at 75% VO2peak.

### Discussion

#### Hemodynamic Measures During Dynamic Exercise

The increased SBP response in blacks was consistent with the findings of other studies.24,25,27 Treiber et al.

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### Table 1. Descriptive and Physical Characteristics of the Subjects

<table>
<thead>
<tr>
<th>Variable</th>
<th>Blacks</th>
<th>Whites</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
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<td>15</td>
</tr>
<tr>
<td>Age (years)</td>
<td>21.0±0.8</td>
<td>22.4±0.7</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>177.5±2.0</td>
<td>178.3±1.5</td>
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<td>Weight (kg)</td>
<td>75.4±2.2</td>
<td>76.8±2.7</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>23.9±0.5</td>
<td>24.1±0.6</td>
</tr>
<tr>
<td>Body surface area (m²)</td>
<td>1.92±0.04</td>
<td>1.94±0.04</td>
</tr>
<tr>
<td>Sum of skinfolds (mm)</td>
<td>27±7</td>
<td>27±8</td>
</tr>
<tr>
<td>Positive parental history</td>
<td>8 of 14</td>
<td>4 of 14</td>
</tr>
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</table>

Values are listed as mean±SEM.

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### Table 2. Peak Oxygen Uptake Test Data

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<tr>
<th>Variable</th>
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</thead>
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<tr>
<td>VO2peak (l/min)</td>
<td>3.33±0.13</td>
<td>3.62±0.15</td>
</tr>
<tr>
<td>VO2peak (ml·kg⁻¹·min⁻¹)</td>
<td>44.1±1.2</td>
<td>47.1±1.1</td>
</tr>
<tr>
<td>Peak HR (bpm)</td>
<td>184±3</td>
<td>184±2</td>
</tr>
<tr>
<td>Peak SBP (mm Hg)</td>
<td>215±5</td>
<td>209±4</td>
</tr>
<tr>
<td>Peak DBP (mm Hg)</td>
<td>87±3</td>
<td>83±2</td>
</tr>
</tbody>
</table>

VO2peak, peak oxygen uptake; HR, heart rate; bpm, beats per minute; SBP, systolic blood pressure; DBP, diastolic blood pressure. Values are listed as mean±SEM.
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TABLE 3. Cardiovascular Measures at Rest and During Submaximal Exercise

<table>
<thead>
<tr>
<th>Variable</th>
<th>SBP (mm Hg)</th>
<th>DBP (mm Hg)</th>
<th>MABP (mm Hg)</th>
<th>HR (bpm)</th>
<th>V02 (l/min)</th>
<th>CO (l/min)</th>
<th>TPR (mm Hg • l•min⁻¹)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Supine</td>
<td>Seated</td>
<td>25</td>
<td>50</td>
<td>75</td>
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<td></td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blacks</td>
<td>114±2</td>
<td></td>
<td>146±4</td>
<td>174±5*</td>
<td>199±6†</td>
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<tr>
<td>Whites</td>
<td>115±2</td>
<td></td>
<td>137±3</td>
<td>160±4</td>
<td>180±4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Blacks</td>
<td>68±2</td>
<td>83±2</td>
<td>86±2†</td>
<td>85±2*</td>
<td>89±4†</td>
<td></td>
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</tr>
<tr>
<td>Whites</td>
<td>68±2</td>
<td>81±2</td>
<td>80±1</td>
<td>74±2</td>
<td>77±2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MABP (mm Hg)</td>
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<td></td>
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<td></td>
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</tr>
<tr>
<td>Blacks</td>
<td>84±2</td>
<td>98±2</td>
<td>106±3*</td>
<td>114±3†</td>
<td>125±4†</td>
<td></td>
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<tr>
<td>Whites</td>
<td>83±2</td>
<td>95±2</td>
<td>99±2</td>
<td>102±2</td>
<td>111±2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HR (bpm)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blacks</td>
<td>58±2</td>
<td>73±3</td>
<td>100±2</td>
<td>130±3</td>
<td>165±3</td>
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<tr>
<td>Whites</td>
<td>58±2</td>
<td>71±2</td>
<td>95±3</td>
<td>123±3</td>
<td>162±3</td>
<td></td>
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<tr>
<td>V02 (l/min)</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blacks</td>
<td>...</td>
<td>0.37±0.02</td>
<td>0.96±0.04</td>
<td>1.67±0.07</td>
<td>2.59±0.10</td>
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<tr>
<td>Whites</td>
<td>...</td>
<td>0.36±0.02</td>
<td>1.03±0.07</td>
<td>1.82±0.10</td>
<td>2.82±0.13</td>
<td></td>
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</tr>
<tr>
<td>CO (l/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blacks</td>
<td>...</td>
<td>5.0±0.2</td>
<td>8.8±0.3</td>
<td>12.9±0.5</td>
<td>...</td>
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</tr>
<tr>
<td>Whites</td>
<td>...</td>
<td>5.3±0.3</td>
<td>10.0±0.5</td>
<td>13.6±0.4</td>
<td>...</td>
<td></td>
<td></td>
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<tr>
<td>TPR (mm Hg • l•min⁻¹)</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blacks</td>
<td>...</td>
<td>20.0±0.9†</td>
<td>12.1±0.3†</td>
<td>9.0±0.3†</td>
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<tr>
<td>Whites</td>
<td>...</td>
<td>18.4±0.8</td>
<td>10.1±0.4</td>
<td>7.6±0.2</td>
<td>...</td>
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</table>

VO2peak, peak oxygen uptake; SBP, systolic blood pressure; DBP, diastolic blood pressure; MABP, mean arterial blood pressure; HR, heart rate; bpm, beats per minute; VO2, oxygen consumption; CO, cardiac output; TPR, total peripheral resistance. Values are listed as mean±SEM. *Significant racial difference, p<0.05; †p<0.01.

Blacks also demonstrated elevated DBP values during exercise in the present study. Perhaps because of the difficulty of measurement during exercise, many studies have failed to report changes in DBP across exercise conditions. Blacks also demonstrated elevated DBP values during exercise in the present study. Perhaps because of the difficulty of measurement during exercise, many studies have failed to report changes in DBP across exercise conditions. Blacks also demonstrated elevated DBP values during exercise in the present study. Perhaps because of the difficulty of measurement during exercise, many studies have failed to report changes in DBP across exercise conditions. The only other study to examine DBP in blacks and whites did not find a significant difference, although it was done on young children. In addition, the MABP response to exercise was found to be greater in blacks than in whites. Although there was a racial difference in submaximal BP, blacks showed only a trend toward greater SBP and DBP values during peak exercise.

Previous studies speculated that blacks may have an increased peripheral resistance in exercise due to the existence of elevated pressures without accompanying increases in HR. However, these reports often did not examine the immediate determinants of MABP: cardiac output and TPR. In the present study blacks had an increased TPR at rest and during exercise compared with whites. The findings are supported by Arensman et al., who found that black children had a greater systemic vascular resistance (31 versus 24 at rest and 20 versus 15 at maximal exercise) and lower cardiac index (2.2 versus 2.9 l • min⁻¹ • m⁻² at rest and 4.5 versus 5.8 l • min⁻¹ • m⁻² at maximal exercise). We considered the possibility that the racial differences we observed were the result of equating subjects on the basis of relative rather than absolute power output. Since the blacks tended to have slightly lower values for VO2peak (3.33 versus 3.62 l/min), they exercised...
at slightly lower absolute work rates. To account for this difference, we also analyzed the MABP, cardiac output, and TPR responses as a function of absolute \( V_O_2 \) (1·min\(^{-1} \)) rather than percentage of \( V_O_2^{peak} \). This widened the difference in the MABP response for blacks and whites and resulted in identical cardiac output responses for the two races. Thus, this method of analyzing the data also supports the conclusion that the exaggerated BP response in blacks is due to increased TPR rather than cardiac output.

Other researchers have shown that, in response to a cold-face stimulus, blacks have greater increases in SBP, DBP, TPR, and forearm vascular resistance than whites.\(^7\) The differences observed in the exercise studies mentioned above in that blacks did not exhibit higher resistance values at rest. In either case, it is apparent that racial differences in BP during physical stress result from an elevated vascular resistance in blacks compared with whites.

Given the twofold increase in prevalence of hypertension among blacks, it was expected that a positive parental history of hypertension would occur more frequently in our black subjects. Two thirds (65.5%) of the black subjects reported a positive parental history of hypertension compared with 28.6% of the white subjects; however, due to the sample size the difference was not statistically significant (\( p=0.085 \)). Since parental history is associated with an exaggerated BP response to exercise,\(^22\,23 \) the differences we observed may reflect a difference in parental history rather than race per se. Statistical analysis revealed that a positive parental history of hypertension was associated with increased DBP but not SBP values during exercise.

### Catecholamine Response to Dynamic Exercise

It has been suggested that the exaggerated BP response to stress in blacks may be due to greater norepinephrine responses.\(^42 \) Resting levels of plasma norepinephrine and epinephrine were similar in blacks and whites, in agreement with other studies.\(^26\,44\,45 \) During the final stage of the submaximal exercise test, blacks had lower norepinephrine levels than whites. Blacks also had higher epinephrine levels at the end of the exercise test, although the difference was small and of questionable physiological significance. Norepinephrine causes \( \alpha \)-mediated systemic vasoconstriction to all vascular beds, whereas epinephrine causes a decrease in total systemic vascular resistance.\(^46 \) Thus, the catecholamine response in the present study does not explain the racial difference in TPR.

Two other studies have examined plasma catecholamine levels during exercise. Hohn et al.\(^47 \) studied adolescents 10–17 years old for possible racial differences in plasma catecholamine levels before and after treadmill exercise. They found norepinephrine values 1 minute after exercise to be higher for whites (2,051 ± 100 pg/ml) compared with blacks (1,779 ± 97 pg/ml); however, these values were not significantly different. Tisch enkel et al.\(^48 \) examined catecholamine responses to various stressors as a function of race and sex. After 4 minutes of cycle exercise (65% age-predicted HR\(_{\text{max}} \)), they found no significant effects of race or sex for either norepinephrine or epinephrine. However, in agreement with the present study, both of these studies showed a trend toward lower norepinephrine responses in blacks.

Venous plasma norepinephrine levels are generally viewed as a valid index of "overall" body sympathetic nerve activity.\(^47\,49 \) Wallin et al.\(^50 \) and Morlin et al.\(^51 \) have shown that plasma norepinephrine levels are correlated with intraneural recordings of muscle sympathetic nerve activity. However, there are limitations to the use of plasma norepinephrine as an index of sympathetic nerve activity. During leg exercise, the forearm can undergo vasoconstriction, which would cause local sympathetic nerve activity to go up and also reduce blood flow to the area. Thus, two key determinants of local norepinephrine spillover to plasma would change and possibly alter the plasma norepinephrine levels. Plasma norepinephrine levels are also affected by nor epinephrine clearance and reuptake as well as norepinephrine release from sympathetic nerve terminals.\(^52\,53 \)

In addition, recent studies have provided evidence of regional specificities in sympathetic nerve activity, demonstrating that sympathetic outflow to various tissues is highly differentiated.\(^54\,55 \) Studies using direct, intraneur al recordings are needed to further examine the hypothesis that blacks have an increased sympathetic reactivity during exercise.

The current study adds to the growing body of evidence that blacks have exaggerated BP responses to dynamic exercise compared with whites.\(^24\,26\,27\,43 \) The black subjects demonstrated an elevation in TPR both at rest and during exercise. An elevation in TPR would have a more pronounced effect on MABP during exercise than at rest. This can be shown mathematically by the formula:

\[
\text{MABP} = \text{CO} \times \text{TPR}
\]

Under resting conditions, the effects of a slight increase in resistance will have only a minor effect on MABP. However, during exercise (when there is a threefold to fourfold increase in cardiac output) the effects of an increase in resistance are magnified accordingly. Thus, dynamic exercise may be a particularly relevant stressor in trying to elucidate early peripheral resistance abnormalities.

The current data do not permit identification of the precise mechanisms responsible for the increased TPR in blacks. It could be due to racial differences in sympathetic nervous system activity, as previously discussed. Another possibility is that increased salt sensitivity in blacks\(^57\,58 \) may lead to increased vasoconstriction.\(^59 \) A third possibility is that structural (i.e., nonneurogenic) differences in vascularity of skeletal muscle exist. This would create an increased resistance to blood flow for any given level of sympathetic tone. A recent study by Ama et al.\(^60 \) reported that blacks have an increased percentage of fast-twitch skeletal muscle fibers compared with whites. Since these muscle fibers are poorly vascularized relative to slow-twitch fibers, a decrease in the size or number of blood vessels could contribute to an increased TPR in blacks. Previous studies have demonstrated that human essential hypertension is characterized by an increased percentage of fast-twitch muscle fibers\(^61\,62 \) and a decreased vascularity of skeletal muscle.\(^63 \)

In summary, the increased BP response in normotensive blacks appeared to be caused by an elevation in TPR. Blacks did not have increased venous plasma
norepinephrine levels, suggesting that other factors (e.g., vascular structure or salt sensitivity) may have contributed to the increased resistance. Identification of the mechanisms responsible for racial differences in the BP response to exercise could provide insights into the pathogenesis of hypertension in blacks.

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