Limited Maximal Vasodilator Capacity of Forearm Resistance Vessels in Normotensive Young Men with a Familial Predisposition to Hypertension

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SUMMARY. The study was performed to determine whether there is a structural vascular abnormality in normotensive subjects with hypertensive relatives. We examined maximal vasodilator capacity of forearm resistance vessels in 23 normotensive young men (mean blood pressure 94 ± 0.4 mm Hg, mean ± SE) with hypertensive relatives (age 24 ± 0.1 years) and in 17 normotensive subjects (mean blood pressure 85 ± 0.4 mm Hg) with no family history of hypertension (age 24 ± 0.1 years). Maximal vasodilator capacity was examined by measuring minimal vascular resistance during peak reactive hyperemia after release from 10 minutes of arterial occlusion. Minimal forearm vascular resistance after release from 10 minutes of arterial occlusion was 25% higher (P < 0.02) in subjects with hypertensive relatives (2.0 ± 0.02 units) than that in subjects with no family history (1.5 ± 0.01 units). We confirmed the previous findings that increasing metabolic vasodilator stimulus by performing intermittent handgrip exercise during 10 minutes of arterial occlusion did not augment peak dilation. This suggests that 10 minutes of arterial occlusion produced maximal vasodilation. Forearm vascular responses to ice on the forehead was greater in subjects with hypertensive relatives than those in subjects with no family history. These results suggest that there may be a structural abnormality in the forearm resistance vessels in normotensive subjects with family history of hypertension. (Circ Res 50: 671–677, 1982)
the past few years, and there was no history of hypertension defined as systolic and diastolic pressure over 150 and 90 mm Hg, respectively.

Twenty-three subjects (24 ± 0.1 years, mean ± se) had a family history of essential hypertension in the first-degree relatives (parents and/or siblings), whereas the other 17 subjects (24 ± 0.1 years old) had no such family history. Information on the presence or absence of family history of hypertension were given by the subjects, but only those who had documented hypertension or normal blood pressure recordings in the first-degree relatives were included in this study.

The study protocol was explained and the consent was obtained from all subjects.

Procedures

The details of the procedures have been reported in a previous study by Takeshita and Mark (1980). Forearm blood flow was measured, using a mercury-in-silastic strain gauge plethysmograph with a venous occlusion technique. The strain gauge was placed approximately 5 cm below the antecubital crease. The arm circumference encompassed by the plethysmograph did not differ significantly in the two groups (24 ± 0.4 cm in subjects with a family history of hypertension and 26 ± 0.4 cm in subjects with no family history). The pressure in the venous occlusion or congesting cuff was 40 mm Hg (Folkow et al., 1958).

Circulation to the hand was arrested by inflating a cuff around the wrist to a suprasystolic pressure for 10 minutes (Takeshita and Mark, 1980). After release of arterial occlusion, forearm blood flow was measured at 8 seconds after release and every 15 seconds thereafter for 2 minutes (Fig. 1). Peak blood flow after release of arterial occlusion and blood pressure measured simultaneously in the opposite arm were used to calculate minimal forearm vascular resistance.

It was previously suggested (Takeshita and Mark, 1980) that vasodilation was maximal during peak reactive hyperemia following arterial occlusion for 10 minutes. To confirm this, we studied the effects of the combination of arterial occlusion and intermittent handgrip exercise in six subjects (four subjects with a family history of hypertension and two subjects with no family history). The subjects underwent intermittent handgrip exercise of the occluded arm approximately 40 times a minutes during the last minute of 10 minutes of arterial occlusion.

In addition, we examined reproducibility of minimal forearm vascular resistance during peak reactive hyperemia after 10 minutes of arterial occlusion by repeating the measurements three times in five subjects at intervals of approximately 25 minutes.

We also examined the effect of acute elevation of blood pressure produced by phenylephrine on minimal forearm vascular resistance during peak reactive hyperemia in 10 subjects (five subjects with a family history of hypertension and 5 with no family history). Phenylephrine was infused intravenously during the last 2–3 minutes of arterial occlusion and continuously during measurements of reactive hyperemia to raise systolic blood pressure by approximately 20 mm Hg.

If structural vascular changes were present, such changes should augment vascular responses to vasoconstrictor stimuli. We examined forearm vascular responses to ice on the forehead in 16 subjects with a family history of hypertension and 13 subjects with no family history. Ice was placed on the forehead for 45 seconds, and forearm blood flow was measured continuously. Forearm vascular resistance before

To produce reactive hyperemia, blood flow to the forearm was occluded by inflating a cuff on the upper arm to suprasystolic pressure for 10 minutes (Takeshita and Mark, 1980). After release of arterial occlusion, forearm blood flow was measured at 8 seconds after release and every 15 seconds thereafter for 2 minutes (Fig. 1). Peak blood flow after release of arterial occlusion and blood pressure measured simultaneously in the opposite arm were used to calculate minimal forearm vascular resistance.

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Figure 1. Recordings of forearm blood flow at rest and during reactive hyperemia after 10 minutes of arterial occlusion. Two recordings of reactive hyperemia flows obtained in a series are shown (First and Second measurement). Resting blood flows were recorded at a paper speed of 15 mm/sec while blood flows during reactive hyperemia were at a paper speed of 25 mm/sec.
and at the termination of cold stimulus was compared.

Calculation and Statistical Analysis
Calculation of forearm blood flow was done independently by three of the authors from the copied records, and the average values were used for statistical analysis. Calculation was done without knowledge of whether each record was obtained from a subject who did or did not have a family history of hypertension. Unpaired and paired Student's t-tests were used for statistical analysis, and P < 0.05 was considered significant. Data are expressed as mean ± SEM.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Comparison of Subjects with and without a Family History of Hypertension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>BP (mm Hg)</td>
</tr>
<tr>
<td>A.Y. 25</td>
<td>126/80</td>
</tr>
<tr>
<td>G.T. 25</td>
<td>122/74</td>
</tr>
<tr>
<td>M.I. 25</td>
<td>124/86</td>
</tr>
<tr>
<td>Y.W. 24</td>
<td>142/72</td>
</tr>
<tr>
<td>M.I. 21</td>
<td>136/60</td>
</tr>
<tr>
<td>S.M. 24</td>
<td>138/70</td>
</tr>
<tr>
<td>K.M. 24</td>
<td>128/70</td>
</tr>
<tr>
<td>T.Y. 23</td>
<td>124/68</td>
</tr>
<tr>
<td>S.K. 23</td>
<td>118/78</td>
</tr>
<tr>
<td>M.S. 23</td>
<td>130/60</td>
</tr>
<tr>
<td>R.I. 24</td>
<td>144/94</td>
</tr>
<tr>
<td>T.R. 30</td>
<td>122/82</td>
</tr>
<tr>
<td>T.A. 22</td>
<td>110/66</td>
</tr>
<tr>
<td>R.T. 20</td>
<td>126/86</td>
</tr>
<tr>
<td>K.K. 22</td>
<td>144/90</td>
</tr>
<tr>
<td>T.O. 20</td>
<td>146/91</td>
</tr>
<tr>
<td>Y.S. 24</td>
<td>111/69</td>
</tr>
<tr>
<td>U.Y. 21</td>
<td>120/88</td>
</tr>
<tr>
<td>N.K. 24</td>
<td>140/80</td>
</tr>
<tr>
<td>Y.K. 24</td>
<td>141/73</td>
</tr>
<tr>
<td>M.I. 22</td>
<td>142/88</td>
</tr>
<tr>
<td>N.F. 24</td>
<td>128/80</td>
</tr>
<tr>
<td>S.N. 24</td>
<td>106/66</td>
</tr>
<tr>
<td>Mean</td>
<td>23.5</td>
</tr>
<tr>
<td>se</td>
<td>0.08</td>
</tr>
</tbody>
</table>

Family history (+)
- U.K. 24 | 128/60 | 83 | 3.0 | 27.7 | 78 | 65 | 1.2 | +5 | -1.0 | +8.5 |
- H.M. 24 | 140/68 | 92 | 3.7 | 24.9 | 83 | 52 | 1.6 | +7 | -0.2 | +5.3 |
- K.F. 24 | 130/62 | 85 | 6.3 | 13.5 | 66 | 47 | 1.4 | +3 | -0.8 | +2.0 |
- S.C. 24 | 124/72 | 92 | 4.3 | 21.4 | 86 | 41 | 2.1 | +6 | -0.7 | +2.5 |
- K.Y. 23 | 118/58 | 78 | 3.3 | 23.6 | 75 | 44 | 1.7 | +2 | 0 | +0.5 |
- K.Y. 24 | 114/60 | 78 | 3.0 | 26.0 | 74 | 53 | 1.4 | +4 | -0.7 | +9.6 |
- J.K. 24 | 106/68 | 81 | 3.3 | 24.5 | 80 | 53 | 1.5 | +2 | -0.3 | +5.1 |
- Y.Y. 24 | 136/64 | 88 | 4.1 | 21.5 | 85 | 61 | 1.4 | 0 | -0.6 | +3.6 |
- Y.H. 26 | 130/70 | 90 | 6.1 | 14.8 | 85 | 47 | 1.8 | +2 | -0.9 | +6.2 |
- T.O. 22 | 108/66 | 80 | 3.3 | 24.0 | 81 | 60 | 1.3 | +4 | -0.3 | +4.2 |
- T.W. 25 | 130/82 | 98 | 3.4 | 30.0 | 98 | 66 | 1.5 | +3 | -0.9 | +11.0 |
- M.O. 23 | 108/66 | 80 | 2.2 | 36.0 | 80 | 55 | 1.4 | +3 | -0.4 | +17.3 |
- K.K. 32 | 120/78 | 92 | 3.5 | 26.0 | 91 | 60 | 1.6 | +5 | -0.9 | +11.0 |
- Y.Y. 24 | 112/64 | 80 | 4.9 | 16.3 | 78 | 56 | 1.4 | +3 | -0.4 | +17.3 |
- U.K. 21 | 125/70 | 88 | 3.0 | 29.3 | 83 | 46 | 1.8 | +5 | -0.9 | +11.0 |
- T.T. 20 | 108/51 | 70 | 4.5 | 15.5 | 69 | 49 | 1.4 | +5 | -0.9 | +11.0 |
- H.K. 24 | 128/72 | 91 | 2.8 | 32.0 | 91 | 56 | 1.6 | +5 | -0.9 | +11.0 |
| Mean | 24.0 | 121/67 | 85.0 | 3.8 | 23.9 | 81.4 | 53.6 | 1.5 | 3.5 | -0.6 | 7.2 |
| se | 0.14 | 0.6/0.4 | 0.4 | 0.06 | 0.36 | 0.46 | 0.42 | 0.01 | 0.14 | 0.02 | 0.4 |

Abbreviations: BP = blood pressure, mBP = mean blood pressure, FoBF = forearm blood flow, FoVR = forearm vascular resistance. **, *, and † indicate P < 0.01, P < 0.02 and P < 0.05, respectively, comparing subjects with a family history of hypertension vs. those with no family history.
Results

Blood Pressure and Resting Forearm Vascular Dynamics

The results are summarized in the Table 1. Systolic blood pressure was not different between subjects with and without a family history of hypertension (129 ± 1 mm Hg for subjects with a family history of hypertension and 121 ± 1 mm Hg for subjects with no family history). Diastolic pressure was significantly higher ($P < 0.01$) in subjects with a family history (77 ± 1 mm Hg) than that in subjects with no family history of hypertension (67 ± 1 mm Hg).

Resting forearm blood flow of subjects with a family history of hypertension (4.2 ± 0.01 ml/min per 100 ml) was not different from that of subjects with no family history (3.8 ± 0.01 ml/min per 100 ml). Resting forearm vascular resistance also was not different between subjects with a family history of hypertension (24.7 ± 0.3 units) and subjects with no family history (23.9 ± 0.4 units).

Forearm Vascular Resistance during Peak Reactive Hyperemia

Maximal forearm blood flow during peak reactive hyperemia following release of 10 minutes of arterial occlusion was not different between subjects with a family history of hypertension (50 ± 0.5 ml/min per 100 ml) and subjects with no family history (54 ± 0.4 ml/min per 100 ml).

Forearm vascular resistance during peak reactive hyperemia was significantly higher ($P < 0.02$) in subjects with a family history of hypertension (2.0 ± 0.02 units) than that in subjects with no family history (1.5 ± 0.01 units).

The equation relating mean blood pressure ($y$) to minimal forearm vascular resistance ($x$) for all subjects (with and without a family history of hypertension) was $y = 69x + 12, r = 0.54$ ($P < 0.01$).

Minimal forearm vascular resistance following the combination of 10 minutes of arterial occlusion and intermittent handgrip exercise in six subjects (1.6 ± 0.2 units) was not different from that after 10 minutes of arterial occlusion alone (1.6 ± 0.1 units).

Minimal forearm vascular resistance during peak reactive hyperemia after 10 minutes of arterial occlusion was similar in three successive measurements (Table 2). The average difference in minimal forearm vascular resistance in successive measurements was 6%.

Phenylephrine elevated blood pressure from 91 ± 4 mm Hg to 109 ± 2 mm Hg in 10 subjects and increased peak reactive hyperemia flow from 57 ± 3 ml/min per 100 ml to 66 ± 2 ml/min per 100 ml. However, minimal forearm vascular resistance during peak reactive hyperemia was not altered (1.6 ± 0.03 units before vs. 1.6 ± 0.05 units after) during phenylephrine infusion.

The average difference between three observers in calculating maximal flow was 7%.

Vascular Responses to Cold Stimulus

Ice on the forehead increased mean blood pressure ($P < 0.01$), decreased forearm blood flow ($P < 0.01$), and increased forearm vascular resistance ($P < 0.01$) in subjects with a family history of hypertension, as well as in those with no family history. However, the extent of changes in these values in response to cold stimulus was significantly greater in subjects with a family history of hypertension than that in subject with no family history ($P < 0.01$ for mean blood pressure, $P < 0.05$ for forearm blood flow, and $P < 0.02$ for forearm vascular resistance).

Discussion

The results of this study indicate that forearm vascular resistance at maximal vasodilation during peak reactive hyperemia was significantly greater in normotensive young subjects who had a family history of essential hypertension in first-degree relatives than that in age-matched normotensive subjects with no family history of hypertension. Forearm vascular responses to ice on the forehead were augmented in subjects with a family history of hypertension. These results suggest that there may be a structural vascular abnormality in resistance vessels in normotensive young subjects with a family history of essential hypertension.

### Table 2

<table>
<thead>
<tr>
<th></th>
<th>First measurement</th>
<th>Second measurement</th>
<th>Third measurement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>pFoBF (ml/min per 100 ml)</td>
<td>mFoVR (units)</td>
<td>mBP (mm Hg)</td>
</tr>
<tr>
<td>A.T.</td>
<td>68</td>
<td>1.4</td>
<td>94</td>
</tr>
<tr>
<td>T.I.</td>
<td>69</td>
<td>1.4</td>
<td>98</td>
</tr>
<tr>
<td>T.A.</td>
<td>59</td>
<td>1.8</td>
<td>106</td>
</tr>
<tr>
<td>K.Y.</td>
<td>85</td>
<td>1.3</td>
<td>109</td>
</tr>
<tr>
<td>Y.N.</td>
<td>45</td>
<td>2.0</td>
<td>90</td>
</tr>
<tr>
<td>Mean ± se</td>
<td>65 ± 6</td>
<td>1.6 ± 0.3</td>
<td>99 ± 3</td>
</tr>
</tbody>
</table>

Abbreviations: pFoBF = peak forearm blood flow, mFoVR = minimal forearm vascular resistance, mBP = mean blood pressure. There was no difference between the first, second, and third measurements.
Experimental Methods

The validity of plethysmographic measurements of blood flow at high flow rates have been discussed in the previous report by Takeshita and Mark (1980). Several studies have shown that the plethysmographic method can be validly applied to the measurements of high blood flow during reactive hyperemia (Folkow et al., 1958; Conway, 1963) as well as to resting blood flow.

A few aspects of the method, in addition to those commented on in the previous report, may deserve discussion here. The linear portions of the plethysmographic recordings are so steep at high flow that a small difference in drawing lines on them might yield a considerable difference in calculated flows. Accordingly, in this study, three of the authors independently calculated flows from the records without knowing whether a record was obtained from a subject with or without a family history of essential hypertension. The interobserver difference on the measurement of peak reactive hyperemia flow was 7%.

In this study, we have employed 10 minutes of arterial occlusion to produce maximal vasodilation, since it was previously demonstrated (Takeshita and Mark, 1980) that increasing metabolic vasodilator stimulus by combining handgrip exercise and 10 minutes of arterial occlusion did not lower minimal vascular resistance more than that following 10 minutes of arterial occlusion alone. Such results suggested that there was maximal vasodilation during peak reactive hyperemia after 10 minutes of arterial occlusion. This observation was confirmed in this study in six subjects. Peak blood flow and minimal vascular resistance in six subjects after 10 minutes of arterial occlusion (55 ± 5 ml/min per 100 ml and 1.6 ± 0.2 units, respectively) were not different from those after the combination of arterial occlusion and handgrip exercise (55 ± 3 ml/min per 100 ml and 1.6 ± 0.1 units, respectively).

An intravenous infusion of phenylephrine increased mean blood pressure and peak reactive hyperemia flow following 10 minutes of arterial occlusion. The extent of the increase in peak reactive hyperemia flow was proportional to that in mean blood pressure, so that minimal forearm vascular resistance at peak reactive hyperemia was not altered. These results support the hypothesis that there was maximal vasodilation during peak reactive hyperemia after 10 minutes of arterial occlusion.

Patterson and Whelan (1955) reported that the first reactive hyperemia blood flow measurement in a series of measurements is often lower than the rest in series. It might thus be suggested that the first value of reactive hyperemia blood flow is discarded. However, Patterson and Whelan did not study reactive hyperemia flow at maximal vasodilation. In their study, reactive hyperemia blood flow was measured after 3 minutes of arterial occlusion. We found that peak reactive hyperemia blood flow after 10 minutes of arterial occlusion was reproducible in this and the previous study (Takeshita and Mark, 1980). The values in five subjects in this study were similar at the first, second, and third measurements and an average difference in minimal forearm vascular resistance in repeated measurements was 0.1 unit (Table 2).

In calculating minimal forearm vascular resistance, we used mean blood pressure which was recorded at the opposite arm during flow measurements. If there were a significant pressure gradient along the large arteries during reactive hyperemia, then the blood pressure measured at the opposite arm would not reflect the true inflow pressure to the small arteries in the vasodilated arm. However, Folkow et al. found that the large artery gradient during reactive hyperemia was small and did not differ in hypertensive and normotensive subjects (Folkow et al., 1958).

Reduced Maximal Vasodilator Capacity

It is unlikely that neurohumoral vasoconstrictor stimuli limit maximal vasodilation during peak reactive hyperemia. Intravenous administration of phenylephrine in this study and intraarterial or intravenous administration of norepinephrine or angiotensin in previous studies did not alter minimal vascular resistance during peak reactive hyperemia (Folkow et al., 1958; Conway, 1963; Zelis et al., 1968). Furthermore, increased sympathetic vasoconstrictor activity produced by lower body negative pressure does not limit peak reactive hyperemia flow following 10 minutes of arterial occlusion (Takeshita and Mark, 1980).

Based on these considerations, we interpret the results to suggest that there is a structural abnormality in resistance vessels in normotensive young men who have a family history of hypertension in first-degree relatives. This notion is supported by the findings that forearm vascular responses as well as blood pressure responses to cold stimulus were augmented in subjects with a family history of hypertension as compared with those in subjects with no family history of hypertension.

Minimal forearm vascular resistance in our normotensive subjects with hypertensive relatives (2.0 units) was similar to that in patients with borderline hypertension (2.1 units) in the previous study (Takeshita and Mark, 1980), but was lower than values reported in studies of patients with moderate to severe hypertension (2.5 to 3.7 units) (Conway, 1963; Amery et al., 1969; Sivertsson, 1970; Sivertsson and Hansson, 1976). It is interesting to note that eight of the 11 borderline hypertensive subjects had a family history of hypertension in the previous study (Takeshita and Mark, 1980).

The etiology of structural abnormality in these subjects is not clear. Although they were normotensive, the mean blood pressure of subjects with hypertensive relatives (94 ± 0.4 mm Hg) was higher (P < 0.01) than that of control subjects with no family history of hypertension (85 ± 0.4 mm Hg). We examined the relationship between mean blood pressure and minimal forearm vascular resistance in these subjects. There was a weak but significant correlation (r = 0.54, P < 0.01) between mean blood pressure and
minimal forearm vascular resistance. Thus, it may be that structural vascular abnormality in normotensive subjects with hypertensive relatives might be an adaptive change in response to a small elevation of blood pressure.

However, the following considerations suggest that hereditary factors—in addition to blood pressure elevation—had contributed to structural vascular changes in subjects with a family history of hypertension. First, we compared minimal forearm vascular resistance between subgroups of subjects of each group who were selected so that the average mean blood pressures in the two subgroups were not different. The subgroups were selected by excluding subjects from the group with a family history who had mean blood pressure of 96 mm Hg or greater and excluding subjects from the group with no family history who had mean blood pressure of 79 mm Hg or less. The two subgroups had similar blood pressures, 88 ± 2 mm Hg for the group with a family history (n = 13) and 87 ± 1 mm Hg for the group with no family history (n = 14) (P > 0.05). Corresponding minimal forearm vascular resistance was greater in the subgroup with a family history of hypertension (1.9 ± 0.1 units) than in the subgroup with no family history of hypertension (1.5 ± 0.1 units) (P < 0.05). Furthermore, forearm vascular responses to ice on the forehead was also greater in the subgroup with a family history of hypertension (ΔFoVR 17 ± 3 units, n = 12) than that in the subgroup with no family history (ΔFoVR 8 ± 2 units, n = 10) (P < 0.05). Second, although there was a significant correlation between mean blood pressure and minimal forearm vascular resistance, the coefficient of variation (r²) was only 0.24. This suggests that the contribution of blood pressure elevation to structural vascular changes may be small. It is conceivable that hereditary predisposition might make vascular smooth muscle more susceptible to pressure loads (Folkow et al., 1958; Brody and Zimmerman, 1976) or neurohumoral stimuli (Bevan, 1975; Bevan and Tsuru, 1977) and produce abnormality in response to even a small increase of such stimuli.

We obviously cannot prove the existence of structural abnormalities or delineate their nature from physiological observations. Structural changes might involve a decrease in number of resistance vessels (Hutchins et al., 1974) or a reduction of the size of vascular lumen with or without wall thickening (Sivertsson, 1970). Augmented vascular responses to cold stimuli may suggest the increase in the wall-to-lumen ratio (Sannerstedt et al., 1976). However, morphological or biochemical studies would be necessary to document and delineate such changes in normotensive subjects with a family history of essential hypertension.

Although we are not certain as to the etiology of structural abnormality in these subjects, the results of this study suggest that structural vascular abnormality already exists in young men with hypertensive relatives whose blood pressure was 129 mm Hg systolic and 77 mm Hg diastolic by average. Resting vascular resistance was not elevated in these subjects, but such mild structural changes might produce significant hemodynamic consequences by augmenting the responses to vasoconstrictor stimuli or by limiting vasodilation (Folkow et al., 1958; Folkow et al., 1973) and thus might contribute to the development of hypertension.

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