Structural Vascular Changes in Young Patients with Borderline Hypertension

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Maximal vasodilator capacity of arteries as well as venous distensibility in the forearm were decreased in young patients with borderline hypertension as compared to those in age-matched normotensive men. These results are consistent with the consideration that there is structural vascular abnormality in arteries and veins in young patients with borderline hypertension.

Structural vascular changes of arteries have been demonstrated in established human hypertension and in experimental hypertension by both morphological methods and physiological measurements. These structural changes contribute importantly to increased vascular resistance in established hypertension.

Structural changes of blood vessels have been noted not only in arteries but also in veins in animals with spontaneous hypertension. In humans, it is shown that venous distensibility is decreased in established essential hypertension.

Recent studies in animals suggest that structural changes may occur in the early stage of hypertension and may also be observed in veins. These findings suggest that structural changes may occur in response to mild elevation of blood pressure (BP) or as a primary abnormality.

Thus, the goal of this study was to examine the possibility that there is a structural vascular abnormality in arteries and veins in young patients with borderline hypertension. We assessed structural vascular changes of arteries by measuring maximal vasodilator capacity of forearm resistance vessels and venous abnormality by measuring forearm venous distensibility.

Borderline hypertension was defined as BP intermittently above 150 mmHg in systole or 90 mmHg in diastole, as measured with a sphygmomanometer with the subjects sitting. We studied a total of 20 patients with borderline hypertension (average age 25 years old) and 23 age-matched normotensive subjects.

Maximal Vasodilator Capacity of Arteries in the Forearm

We measured forearm blood flow using a mercury in silastic strain gauge plethysmograph with a venous occlusion technique. The BP was measured in the other arm with a sphygmomanometer. Forearm vascular resistance was calculated by dividing mean arterial pressure by forearm blood flow. Maximal vasodilator capacity was examined by measuring minimal forearm vascular resistance during peak reactive hyperemia following 10 min of arterial occlusion. Increasing of the metabolic vasodilator stimulus by performing intermittent handgrip exercise during 10 min of arterial occlusion did not augment peak vasodilatation; this suggests that 10 min of arterial occlusion produced maximal vasodilatation.

Blood pressure at the time of study was higher (p < 0.01) in patients with borderline hypertension (systolic 144 ± 4 mmHg and diastolic 89 ± 3

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mmHg) than in normotensive subjects (systolic 121 ± 3 mmHg and diastolic 74 ± 2 mmHg). However, resting forearm blood flow and vascular resistance were not different between two groups.

Forearm vascular resistance during peak reactive hyperemia after 10 min of arterial occlusion was 40% higher (p < 0.05) in subjects with borderline hypertension (2.1 ± 0.2 units) than in normotensive subjects (1.5 ± 0.1 units).

It is unlikely that neurohumoral vasoconstrictor stimuli, which are reportedly augmented in borderline hypertension, limit maximal vasodilatation during peak reactive hyperemia. Intravenous administration of phenylephrine, norepinephrine or angiotensin do not alter minimal vascular resistance during peak reactive hyperemia. In this study, we examined whether increased sympathetic vasoconstrictor activity evoked by lower body negative pressure alters peak reactive hyperemia flow following 10 min of arterial occlusion. Reflex sympathetic activation did not alter minimal forearm vascular resistance.

These results suggest that there are structural vascular changes in the forearm resistance vessels in young subjects with borderline hypertension. These structural changes of the resistance vessels may produce significant hemodynamic consequences by augmenting the responses to vasoconstrictor stimuli, and thus, may contribute to the development of essential hypertension.

Distensibility of Veins in the Forearm

We used a water-filled plethysmograph to obtain a venous pressure-volume curve of the veins in the forearm. The venous pressure-volume curve in patients with borderline hypertension as compared to that in normotensive subjects was shifted to the pressure axis (p < 0.05). This indicates that venous distensibility was significantly decreased in borderline hypertension. Phentolamine, 1 mg intravenously for 5 min, increased venous distensibility slightly but significantly (p < 0.05) in patients with borderline hypertension, but not in normotensive subjects. Thus, decreased venous distensibility in borderline hypertensive patients was due in part to α-adrenergic venuconstriction. Venous distensibility after phentolamine was less in borderline hypertensive patients than in normotensive subjects (p < 0.05). This finding suggests that most of the decrease in venous distensibility in borderline hypertension resulted from non-adrenergic mechanisms, perhaps structural changes in veins.

Functional significance of the decreased venous distensibility in young men with borderline hypertension remains to be investigated. The decreased venous distensibility might contribute to redistribution of venous blood from peripheral to cardiopulmonary circulation, which is reportedly present in young patients with borderline hypertension.

REFERENCES