Diminished finger volume pulse in borderline hypertension: Evidence for early structural vascular abnormality

Digital volume pulse during maximum dilation was determined in 27 patients with borderline essential hypertension (BHT) and 28 age-matched normotensive controls (NT). Finger pulse volume (PV) and finger systolic pressure (SBP) were measured by pneumoplethysmography during vasodilation induced by combining direct and reflex heat dilation and reactive hyperemia. Finger SBP was higher ($p < 0.01$) in BHT than in NT (120 ± 18.7 vs. 104 ± 14.8 mm Hg, respectively); PV was lower ($p < 0.01$) in BHT than in NT (9.7 ± 4.2 vs. 15.3 ± 6 ml/5 ml finger $\times 10^{-3}$). Pulse volume correlated inversely with SBP in BHT ($r = -0.40$, $p < 0.05$) but was unrelated to SBP in NT. Pulse volume was not altered by high or low sodium intake in BHT or NT despite significant changes in plasma renin activity (PRA). In BHT finger vessels are less distensible at a higher pressure than in NT. This abnormality is demonstrable during maneuvers to withdraw sympathetic tone and is not influenced by alteration of PRA. These data support the concept that BHT can produce structural vascular change, and demonstrate that this abnormality can be detected by a relatively simple method. (AM HEART J 104:812, 1982.)

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The need for an aggressive therapeutic approach to *all* patients with mild hypertension remains uncertain. Recent controlled trials[1,2] suggest that, in general, lowering the blood pressure of patients with diastolic blood pressure over 90 to 95 mm Hg reduces the death rate; however, the results were not conclusive for women or for men under 50 years of age. The decision to treat, particularly in patients with borderline hypertension and those with diastolic blood pressure 90 to 95 mm Hg, is still a difficult one. Evidence of cardiovascular damage such as left ventricular hypertrophy or nephrosclerosis would clearly justify therapeutic intervention, but techniques for detecting these cardiac and renal abnormalities are relatively crude. A more sensitive indicator of early vascular damage from hypertension would be of value. We report evidence that indicates that measurement of the digital volume pulse during maximum dilation is a method for detecting vascular change in patients with borderline hypertension.

METHODS

Study population. Twenty-seven patients, 19 men and 8 women, mean age $31.9 \pm$ SD 9.4 years, with borderline essential hypertension (BHT) were studied. All patients had had at least one blood pressure reading over 150 mm Hg systolic or 90 mm Hg diastolic, or both, and on another occasion a blood pressure of less than 150 mm Hg systolic or 90 mm Hg diastolic, or both, in the previous month. No patient had a serum creatinine concentration greater than 1.5 mg% or evidence of left ventricular hypertrophy on ECG. None had received antihypertensive medication in the preceding 4 weeks. Twenty-eight healthy normotensive volunteers (NT) (15 men and 13 women; mean age $31.9 \pm$ SD 8.8 years) were studied also. These studies were approved by the University of Michigan Medical School Committee to Review Grants for Clinical Research and Investigation Involving Human Beings. All persons read and signed an informed consent form describing the test to be done.

Plethysmography. The digital plethysmograph consisted of a translucent plastic cylinder, sealed at the distal end with a rubber stopper penetrated by a large-caliber steel needle attached to a three-way stopcock, and fixed with a malleable plastic material forming an airtight seal to the skin at a point midway between the proximal and distal interphalangeal joints of the third finger of the hand. The chamber was connected by stiff tubing from the stopcock to a Statham pressure transducer. Output from the transducer was recorded on a Gilson polygraph. The system was calibrated directly by the introduction of 0.015 ml air through the stopcock. An inflatable cuff with a
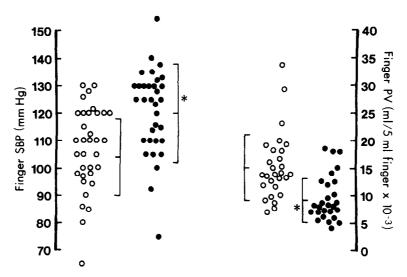
Fig. 1. Finger systolic blood pressure *(SBP)* and finger pulse volume *(PV)* in normal subjects *(open circles)* and in patients with borderline hypertension *(closed circles)*. Brackets indicate ± standard deviation around the mean. $*p < 0.01$, hypertensive patients vs. normal subjects.

Table I. Brachial and finger blood pressure and finger pulse volume during maximum vasodilation in patients with borderline hypertension and in normal subjects

	Brachial		Finger	
	Systolic (mm Hg)	Diastolic (mm Hg)	Systolic (mm Hg)	Pulse volume (ml/5 ml finger × 10⁻³)
Normal subjects	112 ± 10.6	69 ± 6.5	104 ± 14.8	15.3 ± 6.0
Patients with BHT	132 ± 11.3*	84 ± 7.9*	120 ± 18.7*	9.7 ± 4.2*

Values expressed as mean ± standard deviation.
*Hypertensive patients vs. normal subjects; $p < 0.01$.

bladder 24 mm wide was fastened firmly around the proximal phalanx for the measurement of finger systolic pressure and for inducing ischemia of the finger. Persons were studied in recumbency with the arm slightly elevated on foam rubber cushions.

Digital pulse volume and finger systolic pressure techniques. The amplitude of the digital volume pulse and the systolic blood pressure in the finger were measured under conditions designed to produce maximal dilation.[3] The room temperature was 80° F, and readings were obtained only after (1) the individual's body was heated with the aid of blankets and electric pads until oral temperature increased at least 0.5° F; (2) the hand had been warmed directly with a heating pad for 10 minutes; and (3) the finger had been subjected to ischemia for 5 minutes. Finger systolic pressure was determined by inflating the finger cuff to 30 mm Hg above brachial systolic pressure (previously determined by auscultation) and then deflating steadily while monitoring the cuff pressure and noting the pressure at which digital pulsations returned. Pulse volume was calculated based on a "pulse deflection" obtained by averaging 10 consecutive pulse-height measurements and was expressed per 5 ml finger volume utilizing the formula of pulse volume (ml/5

ml finger) = [(pulse deflection) (0.015/calibration deflection)]/[(finger volume/5)]. The coefficients of variation for these measurements determined in seven normal subjects on 3 separate days were 16.7% for pulse volume and 9.2% for finger systolic pressure.

Study protocol. Pulse volume (PV) and finger systolic pressure (SBP) during maximum dilation were determined in all individuals. In addition, these measurements were obtained in six patients with BHT and in six normal subjects after 4-day periods of high salt (250 mEq sodium/day) and low salt intake (20 mEq sodium/day). Supine plasma renin activity (PRA)[4] and 24-hour urinary sodium excretion were also measured in these individuals.

RESULTS

Digital PV and finger SBP. Brachial blood pressure by auscultation was 20/15 mm Hg higher in patients with BHT than in normal subjects (Table I). Similarly, systolic blood pressure in the finger was higher (120 mm Hg) in BHT than in NT (104 mm Hg). Maximum amplitude of the digital volume pulse, however, was smaller (9.7 ml/5 ml finger × 10⁻³) in BHT than in NT (15.3 ml/5 ml finger × 10⁻³). More

Table II. Finger systolic blood pressure and pulse volume, plasma renin activity, and urinary sodium excretion during low salt and high salt diet in patients with borderline hypertension and in normal subjects

	Diet	Systolic (mm Hg)	Pulse volume (ml)*	Sodium excretion (mEq/24 hr)	Supine PRA (ng/ml/hr)
Normal subjects	Low salt	110	11.6	58	2.5
(n = 6)	High salt	112	11.5	212†	0.58‡
Patients with BHT	Low salt	118	8.3	15	3.1
(n = 6)	High salt	128	8.7	194†	0.48†

*ml/5 ml finger × 10^{-3}.
†$p < 0.05$.
‡$p < 0.01$, high salt vs. low salt.

than 80% of normal subjects had PV greater than 10 ml/5 ml finger × 10−3, whereas 75% of the hypertensives had PV *less than* 10 ml/5 finger × 10^{-3}, and approximately 15% of the patients with BHT fell below the normal range (Fig. 1).

Digital PV related to finger SBP. The relationship of PV to SBP in the finger was determined by calculating linear correlation coefficients for these two variables in both BHT and NT. Pulse volume correlated inversely with SBP in hypertensives ($r = -0.40$, $p < 0.05$), while it was unrelated to SBP in normals ($r = -0.01$).

Influence of dietary sodium intake. Restriction of sodium intake with associated increase in PRA was not accompanied by any change in PV in normal subjects or in hypertensives (Table II). Finger blood pressure was not influenced significantly by dietary sodium intake in NT or BHT.

DISCUSSION

Reduced digital PV in borderline hypertension. These data document a reduction in the volume pulse in the finger of patients with BHT compared with age-matched normotensives. This pulse reflects volume changes in venules and capillaries primarily[5]; it represents the time course of the differences between volume of inflow and volume of outflow in the digit.[6] Three major determinants of the volume pulse are flow velocity, wave velocity, and tissue elasticity.[7] A diminished pulse volume could result from a reduction in flow velocity, a reduction of perivascular or vascular compliance, or an increase in wave velocity. Digital blood flow has been found to be normal in most hypertensives[8, 9] despite elevated inflow pressure, so it is unlikely that alteration of flow rate can explain the small volume pulses noted in our patients.

Decreased vascular compliance in hypertension. There is ample evidence, however, for the existence of structural change in peripheral arteries in established hypertension[10, 11] and in BHT.[12, 13] In addition,

there is evidence of structural change in peripheral veins in hypertension.[14, 15] Nakayama and Azuma[16] were able to calculate vascular compliance in the finger, utilizing the quantitative relationship between digital blood pressure and the volume pulse, and found it to be reduced in hypertensives. It seems most likely, then, that the abnormality of the digital volume pulse we have noted is related to structural changes in the vessels of the finger caused by hypertension. The fact that there is a significant inverse relationship between pulse volume and systolic blood pressure in our borderline hypertensive patients but not in normal subjects is consistent with this hypothesis. We have not studied patients with established hypertension, but demonstration of persistence of the inverse relationship between systolic blood pressure and pulse volume at higher pressures would strengthen the concept that the reduced pulse volume in BHT results from structural alterations in the vasculature. Finally, it is possible that an alteration in pulse wave velocity could contribute to the reduced digital pulse volume in BHT, because pulse wave velocity has been found to be increased in patients with established hypertension[17]; however, pulse wave velocity in BHT has not been measured by us or by others.

Structural vascular change in hypertension. Although it is possible that the reduction in amplitude of the volume pulse we observed in BHT is caused by increased neurogenic vascular tone with diminished blood flow rather than by structural changes, this seems unlikely. Our patients were studied under conditions demonstrated by others to eliminate nervous control of cutaneous vessels[18] and to produce maximum vasodilation in vessels of the upper extremity.[10, 11, 19] Although acute injection of vasoactive agents such as angiotensin II will not constrict vessels maximally dilated by heat and reactive hyperemia,[10] we were interested to observe the effect of manipulation of sodium intake with associated longer term changes in the renin-angio-

tensin system on the finger pulse volume measured during maximum dilation. Despite significant changes in plasma renin activity, no change in the volume pulse was observed. It does not appear likely, then, that angiotensin, or for that matter other humoral factors, causes the reduced finger pulse volume observed under the conditions of our study.

Clinical significance. The presence of a diminished volume pulse in the finger in some patients with BHT is of potential practical significance, particularly inasmuch as the method of measurement is relatively simple. If this hemodynamic abnormality does reflect structural vascular change, its detection could be of value in the clinical assessment of patients with borderline blood pressure elevation. Presence of this abnormality might be an early sign of cardiovascular injury in such patients. As such it could be an additional factor in therapeutic decision making in BHT.

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