CLINICAL INVESTIGATION

Ventricular/vascular interaction in patients with mild systemic hypertension and normal peripheral resistance

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ABSTRACT Total left ventricular external power and aortic input impedance spectra were calculated from recordings of pulsatile pressure and flow in the ascending aorta of 22 human subjects undergoing cardiac catheterization. Eleven subjects had increased aortic pressure (systolic 153 ± 3.8(SEM) mm Hg, p < .001; diastolic 91 ± 2.4 mm Hg, p < .03; mean 118 ± 2.4 mm Hg, p < .001) and constituted the group with mild hypertension (average age 50 ± 1.9 years). The other 11 (age-matched) subjects had normal arterial pressures and constituted the control group. Cardiac output in the hypertensive group was abnormally high (6.9 ± 0.3 liters/min, p < .04) compared with that in control subjects (6.1 ± 0.2 liters/min), so that peripheral resistance was similar. Characteristic aortic impedance (index of aortic elastance) was increased in the hypertensive group (142 ± 19 vs 72 ± 4.5 dyne-sec-cm⁻³, p < .002), as was the fluctuation of impedance moduli and phase. These elevated pulsatile components of arterial load were associated with a significant (p < .002) increase in pulsatile left ventricular external power (89%), and the increased cardiac output was associated with a significant (p < .001) increase in steady flow power (31%). The ratio of pulsatile to total power was also increased (38%) in the hypertensive group (p < .001). Increased characteristic aortic impedance in the hypertensive group suggests that the human aorta is stiffer, and fluctuations in the impedance spectra suggest increased or less dispersed wave reflections. These alterations in the systemic arterial tree suggest that factors other than blood pressure and peripheral resistance impose an additional, functionally important hydraulic load on the left ventricle of subjects with mild or borderline hypertension. Circulation 74, No. 3, 455-462, 1986.

THE TOTAL external hydraulic power developed by the left ventricle to propel blood through the systemic circulation can be determined from analysis of pulsatile pressure and flow recordings at the entrance to the system.¹ ² ³ Hydraulic power determined in this manner depends not only on the ability of the left ventricle to do external work but also on the properties of the arterial tree into which blood is ejected. The aortic input impedance spectrum can be used as an expression of these properties.³ For example, the stiffer the aorta, the larger the impedance moduli¹ ² ³ ⁶ and the greater the amount of power required to produce a given blood flow.¹ ⁶ External ventricular power can be separated into two components — one associated with mean blood flow (steady power) and the other with vascular pulsations (pulsatile power).¹ ² ³ ⁶

Experimentally produced hypertension, arteriosclerosis, and aortic coarctation² ⁶ ⁷ cause higher impedance values over the frequency band that contains most of the energy of the flow wave delivered from the ventricle. Under these conditions more energy is lost in vascular pulsations. Porje⁸ has measured pulsatile and steady components of external left ventricular power in humans and has shown increases in pulsatile as well as steady components when blood pressure was elevated with norepinephrine. In patients with aortic coarctation, he found the ratio of pulsatile to total external power increased from between 10% and 17% to between 24% and 37%. Milnor et al.⁹ measured pulsatile and steady components of right ventricular power in patients with pulmonary hypertension and found both to be increased compared with control values.

The purpose of this investigation was to study pulsatile pressure-flow relationships and vascular imped-
ance in the proximal aorta of subjects with mild or borderline systemic hypertension and normal peripheral resistance. The findings were compared with measurements made in age-matched subjects with normal arterial pressure.

Methods

Patients. The subjects in this study were adults who were undergoing diagnostic cardiac catheterization studies for a chest pain syndrome. The study was approved by the appropriate institutional committees for clinical investigation, and informed consent was obtained from each patient. Patients with clinical evidence of heart failure or valvular or congenital heart disease were specifically excluded. Mild systemic hypertension was documented in 11 patients, mean age 50 ± 1.9(SEM) years, by multiple measurements of supine diastolic arterial blood pressure of 90 mm Hg or more or systolic pressure of 150 mm Hg or more. Patients were untreated or had discontinued their therapy at least 2 weeks before the study. Patients 12, 14, 16, 17, and 19 had evidence of mild left ventricular hypertrophy. Each hypertensive patient was matched for age (50 ± 2 years) with a subject who was normotensive.

Catheterization technique and instrumentation. Pulsatile ascending aortic blood flow velocity was measured with a No. 8F multisensor catheter (Millar Instruments, model VPC-683D) that was introduced through a brachial artery cutdown; its use and calibration have been described in detail elsewhere. In addition to a velocity sensor located 7 cm from the tip, this catheter contains two micromanometers to measure aortic and left ventricular pressure. The catheter tip (distal micromanometer) was positioned within the ventricular cavity. The region containing proximal pressure and velocity sensors was located near the upper border of the sinus of Valsalva. The velocity probe was energized with a sinewave electromagnetic flowmeter (Biotronex Laboratories, model BL613). The flowmeter was operated at a setting of 50 Hz (3 dB), which gives a constant (±5%) amplitude response from 0 to 23 Hz and a linear phase shift with frequency. The output signal of the flowmeter was calibrated in milliliters per second by reference to a simultaneous determination of cardiac output with dye dilution. Signals from flowmeter and micromanometer control units (TBC-100, Millar instruments) were amplified with an Electronics for Medicine direct-current amplifier and recorded on magnetic tape. These analog signals were later digitized at a sampling interval of 10 msec by an analog-to-digital converter. Data were analyzed on a digital computer (Hewlett-Packard, model 9820A or PDP 11/03) that converted pressure and flow data to Fourier series and computed aortic input impedance and hydraulic power as functions of frequency. The resistive term (impedance modulus at 0 Hz) was calculated by dividing mean flow into mean pressure. Impedance components were not calculated for harmonics in which the pressure modulus was less than 0.6 mm Hg or the flow modulus was less than 1 cm²/sec; these values represent the noise levels of our measurement systems. This procedure eliminated all data above about 12 Hz. To minimize the effects of reflected waves, we estimated characteristic impedance (index of elastance) by averaging impedance moduli greater than 2 Hz. Left ventricular external power was computed by methods previously reported.

Pulsatile aortic flow and pressure signals were recorded during a hemodynamically stable period. Mean values and standard errors of the mean were calculated. Comparisons of hemodynamic data from the hypertensive patients and the age-matched normotensive subjects were made with use of the t test.

Results

No cardiovascular disease was found by hemodynamic measurements, left ventricular cineangiography, or coronary arteriography in the normotensive subjects (table 1). Mean aortic pressure was 102 ± 1.1 mm Hg (average ± SEM), systolic pressure 130 ± 2.1 mm Hg, aortic diastolic pressure 83 ± 2.3 mm Hg, pulse pressure 48 ± 1.6 mm Hg, and cardiac output 6.1 ± 0.3 liters/min. In the hypertensive group three patients (Nos. 12, 14, and 18) had severe coronary artery disease, two (Nos. 15 and 19) had mild

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<th>Ao press (mm Hg)</th>
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HR = heart rate; CO = cardiac output; SV = stroke volume; Ao = aorta; R = input resistance; Zo = characteristic input impedance.

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TABLE 2
Pulsatile hemodynamics in hypertensive patients

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p value <  | NS       | NS             | 0.04       | 0.04    | 0.001                | 0.001    | 0.03       | 0.008 | NS              | 0.002           | 0.001           | 0.001             | 0.001          | 0.001                     |

Abbreviations are as in table 1.

coronary artery disease, and the remaining six had normal coronary arteries (table 2). Mean aortic pressure was 118 ± 2.4 mm Hg (p < .001), systolic pressure 153 ± 3.8 mm Hg (p < .001), aortic diastolic pressure 91 ± 2.4 mm Hg (p < .03), pulse pressure 63 ± 4.8 mm Hg (p < .01), and cardiac output 6.9 ± 0.3 liters/min (p < .04). Heart rate was within normal limits for all patients studied (hypertensive patients 75 ± 3.1 beats/min; normotensive subjects 79 ± 3 beats/min; p = NS).

The resistive (input resistance) and elastic (characteristic impedance) components of left ventricular external load (aortic input impedance) from both groups of patients are displayed in figure 1. Normotensive patients had an input resistance of 1344 ± 55 dyne-sec-cm⁻³ and characteristic impedance of 72 ± 4.5 dyne-sec-cm⁻³. Input resistance was 1386 ± 60 dyne-sec-cm⁻³ in the hypertensive group (p = NS compared with the normotensive group), and characteristic impedance was 142 ± 19 dyne-sec-cm⁻³ (p < .002).

Representative examples of the input impedance spectra obtained from a subject in the normotensive group and a hypertensive patient are shown in figure 2. In both patients input impedance moduli fell steeply from a high value at zero frequency (input resistance) to a minimum between 4.0 and 6.0 Hz, and then increased to a well-defined maximum. In the normotensive subject the minimum occurred at 4.7 Hz, whereas in the hypertensive patient it occurred at 5.5 Hz. Characteristic impedance was 273 dyne-sec-cm⁻³ in the hypertensive patient compared with 60 dyne-sec-cm⁻³ in the normotensive subject. Also, in the hypertensive patient the fluctuation of the impedance moduli was greater. The increased aortic stiffness in the hypertensive patient increased all impedance moduli as pulse-wave velocity increased, shifting the impedance minimum and "zero crossing” of the impedance phase to higher frequencies.

Total left ventricular external hydraulic power (tables 1 and 2) associated with aortic blood flow averaged 1578 ± 72 mW in the normotensive subjects. Average total power (2179 ± 99 mW) was significantly (p < .001) higher in the hypertensive patients compared with normal subjects. The individual values,

![FIGURE 1](http://circ.ahajournals.org/DownloadedFile/10.1161/01.CIR.74.3.612/10.116101.CIR.74.3.612.f1)

**FIGURE 1.** Mean ± SEM aortic input resistance and characteristic impedance values in normotensive subjects (open bars, n = 11) and hypertensive patients (stippled bars, n = 11). Resistance was similar in both groups, but characteristic impedance was higher in the hypertensive patients.
however, overlapped somewhat. The “pulsatile component” of hydraulic power, which expresses the energy expended in pulsations and depends predominantly on the physical properties of the aorta, constituted a larger part of the total power in the hypertensive patients than in the normotensive group (388 ± 32 vs 205 ± 14 mW, p < .001). In addition, the ratio of pulsatile to total power was significantly (p < .001) greater in the hypertensive patients (18 ± 1.1% vs 13 ± 0.8%). Kinetic energy accounted for less than 2% of the total power in both patient groups and therefore was not considered in the calculation of power.

Discussion

Aortic input impedance. Aortic input impedance, the complex relationship of pulsatile pressure and pulsatile flow in the ascending aorta, provides a measure of the dynamic physical properties of the arterial tree.2, 18 This function considers not only the relationship of mean pressure and mean flow (input resistance), but also the elastance (inverse of compliance) of the aortic wall, inertial properties of the blood, and the effects of wave reflections.2, 18 Resistance is determined by the viscous properties of blood and arteriolar caliber and is usually increased in the later stages of hypertension because of a generalized increase in arteriolar tone. Because the heart is a pulsatile pump, resistance describes only one component of the arterial load presented to the heart during ejection.1, 3 The other component (the elastic component) depends on the elastic properties of the aortic wall and is estimated from measurements of characteristic impedance. Reflected pressure and flow waves in the systemic arterial system may also contribute to the load faced by the ventricle during ejection. Use of input impedance to characterize the external arterial load presented to the ventricle was first suggested by McDonald and Taylor18 in 1959 and was described by Patel et al.19 in 1963. Further studies of systemic input impedance in dogs were reported by Noble et al.20 and by O'Rourke and Taylor.3 The use of aortic input impedance to characterize dynamic, external vascular load in humans has been well established.1, 4, 5, 13-16, 21-24 The aortic input impedance spectra are qualitatively the same in both dogs and humans, and conclusions that have been drawn from such spectra on the basis of experiments in dogs apply also to humans.

The characteristic impedance in our normotensive subjects is in agreement with values that we1, 4, 5, 25 and others13, 14 have found in awake unsedated humans.
However, the aortic input impedance spectra were different in the hypertensive patients. The principle findings were (1) an increase in amplitude of moduli at lower harmonics, (2) an increase in characteristic impedance, (3) an increase in amplitude of the fluctuations between maximum and minimum impedance moduli, and (4) a shift in the impedance modulus minimum to a higher frequency.

Figure 2 illustrates the effects of hypertension on the ascending aortic input impedance spectra. The curve on the left indicates impedance spectra in a normotensive subject and that on the right impedance spectra in a hypertensive patient. The latter curve is displaced upward by the impaired elastic properties of the proximal aorta and proximal large arteries and to the right by increased stiffness of the arterial tree (increased pulse-wave velocity). Gabe et al. and O'Rourke and Taylor observed similar changes in normotensive humans and dogs, respectively, during norepinephrine infusion, and we have shown similar alterations in a small group of patients with mild systemic hypertension. Milnor et al. have also shown increases in the main pulmonary artery impedance in patients with pulmonary hypertension. Increases in age are known to increase the elastance of the aortic wall, and experimental stiffening of the aorta by external constraints has been shown to increase characteristic aortic impedance. In the present study average age was not significantly different in the normotensive and hypertensive groups. Thus, age-matching eliminated this as a possible contributing factor to the observed differences in impedance.

Prolonged elevation of arterial pressure induces vascular changes such as increased wall thickness due to hypertrophy of the smooth muscle cells and, possibly, increased deposition of fibrous proteins. The elastic modulus is also increased because the resting tension in the wall is decreased, resulting in a changed variation of diameter for a given pressure variation; that is, the arterial vasculature becomes less distensible. Increased wall thickness (h) and reduced distensibility (E) both increase pulse-wave velocity (c) and characteristic impedance (since from the Moens-Korteweg formula \( c^2 = \frac{Eh}{2pR} \); \( \rho \) = blood density; \( R \) = luminal radius).

Fluctuations in modulus and phase of the input impedance spectra and increases in amplitude at lower harmonics are due to wave reflections at peripheral sites, principally at the arteriolar level. The frequencies at which such fluctuations occur depend on the distance and wave velocity between the heart and peripheral reflecting sites. The fluctuations in the impedance moduli and phase angles that accompany increased blood pressure are attributable to earlier return of reflected waves as a result of increased pulse-wave velocity.

**Effects of arterial pressure.** Transmural pressure is known to have an important influence on the stiffness of the vascular wall, and it has therefore been assumed that changes in arterial pressure affect the characteristic impedance of arteries. The apparent decrease in impedance modulus with increasing pressure reported to occur in dogs is probably an artifact caused by failure of the aorta at the flow transducer to dilate with increased pressure and thus high flow velocity and low impedance modulus is inappropriately registered. Modulus is actually increased when stiffness is increased because of increased characteristic impedance of the proximal aorta and large arteries. When systolic expansion of the dog's upper descending thoracic aorta and proximal brachiocephalic artery was prevented by rigid Lucite ferrules, characteristic impedance increased 47% and stroke volume decreased 29%. The increase in arterial stiffness did not alter mean aortic pressure; however, aortic systolic pressure increased 10% and aortic diastolic pressure decreased 4%. Consideration of the characteristic impedance in relation to mean aortic blood pressure and aortic systolic pressure in our subjects suggests that these factors may be associated with the relatively high characteristic impedance in individuals with hypertension (figure 3). Figure 3, left, shows that the difference in characteristic impedance between the normotensive and hypertensive patients appears to be related, somewhat, to the higher mean pressures in the hypertensive patients because both groups had the same age distribution. The relationship between arterial pressure and characteristic impedance is closer when aortic systolic pressure is used instead of mean pressure. The effect of increased transmural pressure on vascular impedance is far from certain because other factors also influence the impedance. Arteries become stiffer as they are distended, presumably because more and more of the wall stress is borne by collagen as the diameter increases. Vascular impedance is directly proportional to the stiffness (elastic modulus) of the vascular wall, but it is also inversely proportional to the cross-sectional area of the vessel. The net effect of increased transmural pressure on impedance is therefore difficult to predict. Although there are crossovers in our data, patients with elevated arterial pressures (hypertension) had characteristic impedance values greater than those in subjects with normal arterial pressures. Characteristic impedance was 97% higher in the hypertensive
patients than in the normotensive subjects, whereas peripheral resistance was normal. These results indicate that the most important effects of mild hypertension on hemodynamics in large arteries are due to changes in distensibility of the large arteries and that alterations in peripheral resistance per se are of little significance.

Hydraulic power. The ability of the heart to generate external power depends not only on myocardial performance but also on the physical properties of the arterial tree and the contained blood. There are two components of external ventricular hydraulic power. The first is steady power, which is associated with the peripheral resistance and is dissipated in the arterioles. The second is pulsatile power, which is associated with the phasic contraction of the ventricle and depends on large artery distensibility and wave reflections. Normally, pulsatile power accounts for 5% to 15% of total power in dogs and 10% to 17% of that in humans. This is a result of a favorable design of the arterial system and the intrinsic heart frequency, which results in impedance moduli being very low over the frequency range that contains most of the energy of the ventricular ejection wave. In our normotensive subjects pulsatile power averaged 205 mW and accounted for 13% (average) of total left ventricular power, which averaged 1578 mW (table 1 and figure 4). Both components of power were significantly greater in the hypertensive patients than the normotensive subjects (table 2 and figure 4). Pulsatile power (average 388 mW) accounted for 18% of the total power in the hypertensive patients. The magnitude of pulsatile power depends on the relationship between ascending aortic impedance and the harmonic content of the left ventricular ejection wave. Therefore, the stiffer the aorta, the higher the characteristic impedance, and the greater the pulsatile power if mean blood flow is maintained. This condition may be considered as reduced “efficiency” of the arterial system in coupling the inter-

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**FIGURE 3.** Variations in characteristic impedance (Zo) with variations in mean aortic and aortic systolic pressure. Values for the normotensive subjects are indicated by closed circles and those for the hypertensive patients are indicated by open circles.

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**FIGURE 4.** Total external hydraulic power of the left ventricle in normotensive subjects and hypertensive patients. The open area represents the energy needed to pump blood through the systemic resistance (steady power) and the stippled area indicates the energy lost in vascular pulsations (pulsatile power). Both components are significantly higher in the hypertensive patients.
We thank Alice Cullu and Anne Crawford for editing and typing the manuscript.

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mittent ejection from the ventricle to the steady tissue perfusion of the peripheral vasculature.

In the hypertensive patients mean blood flow was significantly greater than that in the normotensive sub jects, in spite of a higher impedance in the former. These patients maintained above-normal cardiac out put in spite of an increased arterial load (increased stiffness and increased wave reflections), in other words, although at the cost of a marked increase in total left ventricular power (figure 5). In other studies in patients with mild or "borderline" hypertension increased cardiac output from a "hyperkinetic" ventricle has also been observed. 31-34 In a follow-up study (average 50 months) the high cardiac output initially found tended to revert toward a normal cardiac output, and total peripheral resistance, which was initially normal, increased.

Thus, in these mild or borderline hypertensive patients with high cardiac output and normal peripheral resistance, aortic distensibility is decreased. This results from the increased arterial pressure, degeneration of the normal components of the arterial wall, or both. Decreased aortic distensibility increases characteristic impedance and through its effects on pulse-wave velocity causes the impedance spectra to shift to higher frequencies. This disturbs the normally favorable relationship between the components of the ventricular ejection wave and the impedance presented to the ventricle (figure 2) and results in increased pulsatile energy losses in vascular pulsation.

FIGURE 5. Relationship between total left ventricular external power and cardiac output in normotensive subjects and hypertensive patients (see text for discussion).

Erratum

In a recent article by Ishida et al. (Circulation 74: 187, 1986) there were errors in one of the equations appearing in the Appendix. The equation at the bottom of the right-hand column on p. 193 should have read: $P_{LV} = E_{l,V_{max}} \cdot e^{-\tau \cdot T} \cdot (V_{LV} - V_{LVO}) + A e^{a V_{LV}} + R_{LV} \cdot M_i F$. 
Ventricular/vascular interaction in patients with mild systemic hypertension and normal peripheral resistance.

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