Arterial and Venous Compliance in Sustained Essential Hypertension

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SUMMARY Arterial and venous compliances are decreased in men with sustained essential hypertension. The reduced arterial compliance acts to maintain systolic pressure and end-systolic stress, thus contributing to the development of cardiac hypertrophy. Since cardiac output remains within the normal range in the hypertrophied hypertensive heart, elevated left ventricular pressures, and therefore increased cardiac filling pressures, are necessary if an adequate stroke volume is to be maintained. In hypertensive persons, reduced venous compliance acts to maintain the filling pressure of the heart in the presence of reduced intravascular volume. In patients with hypertension, even if compliance changes have been initiated by the elevated blood pressure itself, the reduced arterial and venous compliance observed in cross-sectional studies is not simply the mechanical consequence of the elevated blood pressure, but also reflects intrinsic alterations of the vascular wall. Consequently, blood pressure reduction caused by antihypertensive agents is not constantly associated with a reversion of the decreased vascular compliance. Such observations may be of importance in the consideration of cardiovascular morbidity and mortality in patients treated for hypertension.

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KEY WORDS • essential hypertension • vascular compliance • antihypertensive therapy

FOR many years the hemodynamics of hypertension have been analyzed in terms of a comprehensive linear model.1 In this model, the driving pressure of the cardiovascular system (i.e., the difference between mean arterial pressure and central venous pressure) reflects a steady flow. Since cardiac output remains within the normal range, application of the Poiseuille law implies that the resistive function of arteries is modified in hypertension, principally at the arteriolar level.

A more realistic approach to the hemodynamics of hypertension takes account of the fact that flow is pulsatile in the arterial part of the cardiovascular system.2 Accordingly, blood pressure may be composed of two components: a steady component, represented by mean arterial pressure, and a pulsatile component, represented by pulse pressure, which is the extent of the pressure oscillation around the mean value. This model implies that the damping function of large vessels is modified in hypertension, principally within the large arteries and veins.

It has been noted that the elasticity of arteries and veins is reduced in hypertension.2,3 However, the general hemodynamic consequences of this alteration have not been widely considered. In the present review, the importance of this reduced arterial and venous compliance is assessed in the systemic circulation of patients with essential hypertension. Because of methodological difficulties in hypertensive humans, information about regional circulations will be limited to the forearm. The implications of compliance changes for blood pressure and cardiac output regulation are analyzed, with particular emphasis on the consequences of antihypertensive therapy.

Basic Concepts for the Definition of Vascular Compliance

In experimental animals, the compliance of the systemic circulation is evaluated by momentarily stopping the circulation and rapidly equalizing the arterial and
venous pressures by pumping blood from arteries to veins. The intravascular pressure measured when arterial and venous pressures are equal is called mean systemic pressure (MSP). By studying the relationship between blood volume and MSP, an index of the elasticity of the system, vascular compliance, can be defined. Vascular compliance, the change in volume divided by the change in pressure, is the slope of the pressure-volume relationship.

Figure 1 illustrates a linearized model of the pressure-volume curves in the arterial and venous systems. As previously reported, these curves have been derived from values measured in dogs and have been extrapolated to normal humans. As far as the arterial tree is concerned, the arterial pressure remains at zero until the volume of blood rises to approximately 0.5 L. The volume at which the pressure begins to rise above the zero level is called the unstressed volume of the arterial system. At the critical level of 0.5 L, any further increase in volume causes a marked and rapid rise in pressure, so that when the volume increases to 0.85 L, the arterial pressure rises to 100 mm Hg. In the venous system, both unstressed volume and compliance are far greater than in the arterial system. The unstressed volume approximates 2.95 L. Any increase in venous volume above 2.95 L causes the pressure to rise above zero. However, at the normal venous volume of 3.25 L, the average pressure in the entire venous system is 3.7 mm Hg. This example illustrates the basic difference between the volume-pressure relationship of the arterial and venous systems, which is related to anatomical differences between elastic structures in the two vascular compartments. These differences have a profound influence on overall circulatory mechanics.

In studying pressure-volume curves in clinical hypertension research, several points must be assessed. It is not possible to determine unstressed volume since linear extrapolations of pressure-volume curves are difficult to accept in humans. On the other hand, the simple model presented in Figure 1 is linearized. In fact, pressure-volume relationships are curvilinear, due to pressure-dependent active changes in vasomotor tone as well as pressure-dependent mechanical effects on the passive wall elements. In the arterial system in particular, arterial compliance increases when pressure decreases. Thus, compliance may be defined only at a given pressure. Consequently, when compliance in hypertensive subjects is compared with that in normal subjects, modifications reflect intrinsic alterations of the vascular system only if the two groups are compared at the same pressure.

**Reduced Arterial and Venous Compliance in Sustained Essential Hypertension**

In normal humans with intact cardiac performance, systemic vascular compliance may be evaluated using a rapid iso-osmotic dextran infusion. Under such conditions, a linear relationship between changes in blood volume and central venous pressure can be established in humans, and the slope of this relationship has the dimension of vascular compliance. The methodology, which has been described and validated in detail elsewhere, is derived from experimental evidence that the central venous pressure is modified in the same way as MSP—increasing with an increase in blood volume and decreasing with a decrease in blood volume. However, to obtain an accurate determination, several prerequisites must be observed. The infusion must be performed within 3 minutes in order to minimize delayed compliance and adaptive reflexes. Indeed, infusion over a 3-minute period does not seem to be rapid enough to prevent some participation of baroreceptor reflexes (occurring within seconds) and autoregulatory adjustments of vascular tone (occurring within 1 minute). However, even if these factors affect estimation of arterial compliance, their influence on total compliance may be considered negligible. On the other hand, the findings can be interpreted only if the functional capacity of the heart is preserved. Indeed, in patients with uncomplicated sustained essential hypertension, it has been widely shown that the curves relating central venous pressure to cardiac output either are similar to those of normal subjects or exhibit a slight rightward shift of otherwise normally shaped curves. In the latter case, the "pump" operates normally but at a higher "basic pressure level" and, for the same rapid volume expansion, cardiac output increases even more in essential hypertensive subjects than in controls.

Following rapid dextran infusion, central venous pressure increases more in hypertensive than in age-matched normal subjects. This result is observed even when the two populations have the same baseline central venous pressure. Systemic vascular compliance, defined as the ratio between the change in volume and the change in pressure during dextran infusion, has been shown to be significantly reduced in hypertensive
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Reduced systemic vascular compliance in hypertensive persons reflects a decrease in the elasticity of the cardiovascular system involving the venous circulation, the arterial circulation, and the left ventricle in diastole. Our knowledge about this problem remains limited. In particular, it is difficult in hypertensive humans to 1) indicate how compliance is partitioned within the arterial and the venous system and 2) ascertain whether only certain vessel sizes are preferentially affected or if all vessels are affected equally. However, some insight can be provided about each of the compartments involved in the decreased systemic vascular compliance. As has been shown using invasive hemodynamic methods, left ventricular compliance is decreased in hypertensive subjects. The values were 0.04 ± 0.10 ml/mm Hg/10³ in hypertensive and 0.07 ± 0.16 ml/mm Hg/10³ in normotensive subjects (p < 0.001). Systemic arterial compliance, evaluated from a Windkessel first-order model of the arterial system, is also reduced; the corresponding values are 0.025 ± 0.001 ml/mm Hg/kg in hypertensive and 0.044 ± 0.004 ml/mm Hg/kg in normotensive subjects (p < 0.001). The reduction in hypertensive subjects is observed even when mean arterial pressure equals that of normal subjects, as demonstrated in the forearm and the systemic circulations. These findings show that left ventricular and systemic arterial compliance are very low in relation to total systemic vascular compliance (<5% of total compliance). Consequently, the reduction in total systemic vascular compliance occurs mainly in the venous system, as would be expected from basic physiology (see Figure 1). With the use of separate determinations of cardiopulmonary and total blood volume in humans, it is possible to deduce the value of intrathoracic vascular compliance. Since this parameter remains within the normal range in hypertensive subjects, the decreased venous compliance principally results from a reduction in the peripheral (extrathoracic) compartment of the venous system.6-9

In conclusion, clinical studies performed in the last few years have shown that systemic vascular compliance is significantly reduced in hypertensive patients, reflecting a reduction in the compliance of the peripheral venous system, the arterial system, and the left ventricle in diastole. Except in the forearm, there is no available information indicating that the compliance of the various regional circulations might be altered to different extents. However, since the reductions in compliance are observed even for the same baseline pressure as in normal subjects, decreased vascular compliance clearly cannot be considered the simple mechanical consequence of the elevated blood pressure; it also reflects intrinsic alterations of the vascular (arterial and venous) wall. Since clinical studies are cross-sectional in nature, it is difficult to establish time-dependent relationships in patients with essential hypertension. Thus, the compliance changes may be initiated by elevated pressure, and over time the intrinsic alterations of the arterial wall may become permanent. As an alternative possibility, if pressure is not the precipitating event, neurohumoral or genetic factors or an associated atherosclerotic process could be involved in the mechanism of the vascular alterations.

Arterial and Venous Compliance and the Pressure-Volume Relationship

Since vascular compliance is the reciprocal of the slope of the pressure (ordinate)-volume (abscissa) relationship, it seems likely that reduced compliance acts to maintain or to increase pressure at any given value of volume. In patients with hypertension, this effect may be observed in both the arterial and venous systems.

In human studies, the pressure considered as most representative of the low pressure system is right atrial pressure (central venous pressure). Central venous pressure, measured in patients with sustained essential hypertension, has been found to be slightly but significantly increased, even in the absence of congestive heart failure or with normal pumping ability of the heart. Since intravascular volume is normal or decreased in patients with hypertension,11 it is clear that decreased venous compliance acts to maintain or increase central venous pressure. In patients with hypertension, this interpretation is strengthened by two findings: 1) intravascular volume and central venous pressure are inversely related, and 2) blood volume and venous compliance are positively correlated, both thus participating to maintain the filling pressure of the heart. The increase in "low-pressure system" filling pressure, caused by decreased systemic capacitance, probably explains the previously observed elevation in renal venous pressure in the hypertensive human kidney. Such an increase of hydrostatic forces within the venous system could compensate for the increased peritubular oncotic forces observed in the hypertensive kidney and participate in maintaining normal sodium balance. Indeed, recent data obtained in our laboratory have shown that 1) systemic vascular compliance is negatively correlated with renal filtration fraction and peritubular oncotic pressure, and 2) central venous pressure and postglomerular oncotic pressure are positively correlated.

In arterial circulation the role of decreased compliance in maintaining pressure is more difficult to assess because of the pulsatile nature of the pressure oscillation. Whereas mean arterial pressure is exclusively

subjects (1.49 ± 0.06 vs. 2.08 ± 0.09 ml/mm Hg/kg; p < 0.01). Total vascular distensibility, defined as the ratio between vascular compliance and initial vascular volume, is also significantly reduced. As measurements are performed in the presence of a beating heart, the decrease in vascular compliance has been considered "effective." However, clinical data obtained by rapid dextran infusion have been introduced in sophisticated models of the cardiovascular system. From the mathematical interpretation of the results, it was shown that the reduction in "effective" vascular compliance clearly reflected a "true" decreased compliance in the systemic circulation, a finding that has been further demonstrated in animal hypertension using a similar methodology. Reduced systemic vascular compliance in hypertensive persons reflects a decrease in the elasticity of the cardiovascular system involving the venous circulation, the arterial circulation, and the left ventricle in diastole. Our knowledge about this problem remains limited. In particular, it is difficult in hypertensive humans to 1) indicate how compliance is partitioned within the arterial and the venous system and 2) ascertain whether only certain vessel sizes are preferentially affected or if all vessels are affected equally. However, some insight can be provided about each of the compartments involved in the decreased systemic vascular compliance. As has been shown using invasive hemodynamic methods, left ventricular compliance is decreased in hypertensive subjects. The values were 0.04 ± 0.10 ml/mm Hg/10³ in hypertensive and 0.07 ± 0.16 ml/mm Hg/10³ in normotensive subjects (p < 0.001). Systemic arterial compliance, evaluated from a Windkessel first-order model of the arterial system, is also reduced; the corresponding values are 0.025 ± 0.001 ml/mm Hg/kg in hypertensive and 0.044 ± 0.004 ml/mm Hg/kg in normotensive subjects (p < 0.001). The reduction in hypertensive subjects is observed even when mean arterial pressure equals that of normal subjects, as demonstrated in the forearm and the systemic circulations. These findings show that left ventricular and systemic arterial compliance are very low in relation to total systemic vascular compliance (<5% of total compliance). Consequently, the reduction in total systemic vascular compliance occurs mainly in the venous system, as would be expected from basic physiology (see Figure 1). With the use of separate determinations of cardiopulmonary and total blood volume in humans, it is possible to deduce the value of intrathoracic vascular compliance. Since this parameter remains within the normal range in hypertensive subjects, the decreased venous compliance principally results from a reduction in the peripheral (extrathoracic) compartment of the venous system.6-9

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determined by the level of cardiac output and vascular resistance, pulse pressure is influenced by three independent hemodynamic mechanisms: the pattern of ventricular ejection, the timing of reflected waves, and the level of arterial compliance. Indeed, for the same mean arterial pressure, a given population of subjects may have different values of pulse pressure depending on the combined values of these parameters, the most important of which is arterial compliance. Thus, reduced arterial compliance may change the amplitude of the pulse pressure and thereby increase the level of systolic pressure, without any change in mean arterial pressure. In isolated systolic hypertension in the elderly, reduced systemic arterial compliance has been shown to be the major factor underlying the predominant or exclusive increase in systolic pressure. In sustained essential systolodiastolic hypertension of middle age, systolic pressure is often disproportionately increased in relation to diastolic pressure. It has been shown that, compared with age-matched and sex-matched normal subjects with the same mean arterial pressure, systolic pressure in this group is slightly but significantly elevated. In this condition, the level of systolic pressure is negatively correlated with the reduction in systemic and forearm compliances. Thus, in patients with hypertension, the reduction in arterial compliance acts to increase blood pressure, but exclusively through its pulsatile component, involving mainly the systolic pressure.

Vascular Compliance and the Structure and Function of the Heart

In patients with hypertension, a normal cardiac output is achieved through cardiac hypertrophy as a result of the application of Laplace's law in the presence of increased afterload. Consequently, a positive correlation would be expected to exist between the degree of cardiac hypertrophy and the level of blood pressure and vascular resistance. However, in patients with sustained essential hypertension, the correlation coefficient of the relationship between cardiac mass and blood pressure has been shown to be relatively low, indicating that other factors may play an important role in modulating left ventricular hypertrophy. Studies of arterial impedance indicate that, in addition to vascular resistance, aortic distensibility markedly contributes to the afterload imposed on the heart and that this parameter should be taken into account in studying the development of cardiac hypertrophy. Indeed, we have found that left ventricular mass is strongly and positively correlated with carotid-femoral pulse-wave velocity, and hence with the reduction in aortic distensibility observed in patients with sustained essential hypertension. Thus, decreased aortic distensibility clearly plays an important role in the increased load of the hypertensive heart and, through increased systolic pressure, influences the degree of end-systolic stress and cardiac hypertrophy.

Cardiac hypertrophy, which is responsible for decreased left ventricular compliance, acts to maintain a normal stroke volume and cardiac output through increased intraventricular pressure. This process ensures an adequate fiber length within the context of a stiffer myocardium. However, established cardiac hypertrophy leads to a reduction in stroke volume in the absence of effective compensation. Such compensation can be found in the form of increased cardiac filling pressure. In patients with essential hypertension, an increase in capillary wedge pressure is observed, indirectly reflecting an increase in left atrial pressure. Right atrial pressure is also slightly but significantly increased. In the presence of normal or decreased blood volume, this increase could result in decreased venous compliance in order to produce an adequate driving pressure for the hypertrophied heart. Ulych et al. showed that the increase in cardiac output following an intravenous volume load was exaggerated in hypertensive patients, suggesting a reduction in peripheral vascular capacity. More direct evidence results from the observation that an inverse correlation exists between cardiac output and vascular compliance in hypertensive subjects: the greater the reduction in the compliance, the higher the cardiac output. Indeed, such a mechanism requires a redistribution of intravascular volume in favor of the intrathoracic compartment, as previously described in hypertensive subjects.

In conclusion, in hypertension, the reduction in both arterial and venous compliances plays a part in the adaptive process of normalizing cardiac output in the presence of myocardial hypertrophy. It seems likely that reduced arterial compliance modulates the systolic function of the heart, whereas reduced venous compliance participates in the changes in diastolic function.

Structural and Functional Components of Decreased Vascular Compliance in Essential Hypertension

As well as smooth muscle cells, the vascular wall contains elastin and collagen fibers. The functioning of the vascular system can be altered either by smooth muscle activity or by structural modifications of the vascular wall itself, such as changes in the relative proportions of the different elements of which it is composed. Changes in the sodium, calcium, and water content of smooth muscle cells also participate in modifications of the vascular wall. Unlike resistance vessels, which alter their caliber in response to local mechanisms of vasoregulation, arterial and venous vessels are regulated through the sympathetic nervous system. In addition to this dominant sympathetic control, biologically active substances, especially those derived from the renin-angiotensin and prostaglandin systems, may affect smooth muscle activity. For this reason, the problem in elucidating the mechanism of decreased vascular compliance in hypertension lies not so much in evaluating the role of functional factors as in determining the contribution of the structural component. Indeed, any increase in the thickness of the vessel wall acts to reduce elasticity.

Both venous wall hypertrophy and modification in tissue water and electrolyte content have been suggest-
ed as possible structural abnormalities in different models of animal hypertension. These modifications have long been considered as occurring in a venous system in which venous pressure remains strictly normal. As discussed, venous pressure is slightly elevated in patients with hypertension. Therefore, the possibility that Laplace's law is operating under these conditions cannot be completely excluded. In addition, physiological and pharmacological changes in venous tone in hypertensive humans provide indirect arguments in favor of structural modifications of the venous wall. In the forearm circulation, the venous pressure-volume curves obtained from hypertensive humans are curvilinear, with the convexity toward the volume axis. This hemodynamic pattern is observed when active venoconstriction is eliminated by venodilator agents or by prolonged stretch followed by relaxation. Even with the use of forearm venous plethysmography, it has proved difficult to demonstrate venous dilatation induced by head-down tilt in hypertensive subjects, whereas this finding has been commonly observed in normal subjects. Interestingly, minor changes in venous tone following short-term or long-term administration of adrenergic blocking agents have been observed in hypertensive subjects.

The role of Laplace's law is evident in the arterial system, since it is precisely the increase in blood pressure within the large arteries that defines clinical hyper- tension. However, direct evidence in favor of increased thickness of the arterial wall in hypertensive humans remains scarce and results mainly from hemodynamic and histopathological observations in the carotid circulation. In patients with sustained uncomplicated essential hypertension, the internal diameter of the common carotid artery remains within the normal range despite the elevated distending pressure, indicating a decrease in arterial compliance. Morphological carotid artery studies have revealed tetraploid smooth muscle cells in both hypertensive rats and hypertensive humans. As many as 50% of the cells in these vessels may have a tetraploid state and, consequently, a higher DNA content. When the DNA content of the wall is corrected for ploidy, endoreplication appears to be associated with an increase in the mass (not in the number) of arterial smooth muscle fibers. Thus, both clinical and morphological changes favor the theory that structural modifications of the arterial wall occur in hypertensive humans. The recent finding that the hypertensive brachial artery shows increased responsiveness to various vasoactive substances, such as norepinephrine, angiotensin, and sodium nitroprusside, is consistent with this interpretation.

Finally, in evaluating the structural and functional components of decreased vascular compliance in hypertensive humans, the role of age must also be considered. Recent studies have shown that, as arterial stiffness and blood pressure increase with age, venous compliance falls, the slope of the curve being steeper in hypertensive subjects. In other words, the possibility exists that, in patients with hypertension, modifications of the arterial and venous systems may evolve in parallel over the course of the disease, with or without an alteration in the time course between the different parts of the vascular system.

**Applications to Cardiovascular Pharmacology and Therapeutics**

From the findings reviewed thus far, it would seem that both arterial and venous compliance are reduced in hypertension, but that these reductions are not entirely related to the level of blood pressure and, instead, reflect intrinsic alterations of the arterial wall. Thus, the blood pressure reduction induced by antihypertensive drugs may be associated with unexpected changes in vascular compliance. Venous tone is only slightly influenced by antihypertensive therapy; furthermore, the arterial system does not appear to respond homogeneously to antihypertensive drugs. The first observation in this regard resulted from measurements of brachial artery diameter following short-term administration of vasodilators. Whereas dihydralazine caused arterial constriction, diltiazem increased vessel diameter for an equivalent reduction in blood pressure. This concept was further extended to changes in aortic distensibility, using carotidofemoral pulse-wave velocity as an index of arterial stiffness. Whereas the dihydralazine-like substance cadsalazine was unable to modify pulse-wave velocity, calcium channel blockers (nicardipine, nifedipine) and converting enzyme inhibitors (captopril) decreased it for an equivalent reduction in blood pressure. Thus, the blood pressure reduction caused by antihypertensive drugs is not consistently associated with improvements in arterial and venous compliance; such improvements depend on the type of antihypertensive compounds administered.

These observations have several consequences for antihypertensive therapy. First, there is no parallelism between the changes in arterial and venous compliance with long-term treatment, a finding that might indirectly suggest that the mechanism of compliance changes differs in the various compartments. For instance, enalapril improves brachial artery compliance but does not change forearm venous tone, while, for the same antihypertensive effect, propranolol does not modify arterial compliance but increases venous tone. Second, the modifications in the arterial and venous systems induced by drugs may influence the load on the heart and hence the extent of reversion of cardiac hypertrophy. Dihydraldazine and related compounds are unable to modify arterial and venous compliance and are known to have only minor effects on cardiac hypertrophy. On the other hand, for the same blood pressure reduction, calcium channel blockers and converting enzyme inhibitors cause both an improvement in arterial compliance and a reversion of cardiac hypertension. Third, the effects of long-term antihypertensive therapy on the structural component of the reduced arterial and venous compliance may be of importance when cardiac hypertrophy is reduced in parallel. Indeed, experimental studies have demon-
stated that cardiac hypertrophy may be reversed with antihypertensive drugs, whereas aortic structural changes remain unmodified, reflecting a dissociation between cardiac and vascular effects.43

Conclusion

The present review has emphasized the role played by decreased arterial and venous compliance in the mechanisms of essential hypertension. This arterial and venous involvement implies that all components of the cardiovascular system are altered in patients with essential hypertension: not only the small arteries and the heart, but also the major arteries and the venous system. In the present report, this assumption was based on analyses of hemodynamic factors. However, it is important to recall that many of the neurohumoral control mechanisms involved in cardiovascular regulation originate from mechanoreceptors located in the arterial system (aortic and carotid receptors) and the low pressure system (atria), with possible consequences for the control of vascular resistance, fluid volume, and kidney function.44 From these observations, it would appear that the role of reduced arterial and venous compliance in the neurohumoral changes observed in hypertension remains an important field for research.

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