Is Pulse Pressure a Stimulus for Altered Vascular Structure in Chronic Hypertension?

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Until recently, investigators in the field of hypertension have focused primarily on the role played by mean arterial pressure in alterations of vascular structure during chronic hypertension. In this issue of *Hypertension*, Christensen considers the possible role of pulse pressure. The author measured mean arterial pressure, systolic pressure, diastolic pressure, pulse pressure, and heart rate in awake spontaneously hypertensive rats (SHR) that had been treated with perindopril or captopril (angiotensin I converting enzyme inhibitors), isradipine (a calcium antagonist), hydralazine (a vasodilator), or metoprolol (a β-blocker). Structural characteristics were determined in small mesenteric arteries using an in vitro approach. Care was taken to standardize both the location from which arteries were obtained and the conditions under which the cross-sectional area of the wall and luminal circumference were determined. All parameters of blood pressure and media/lumen ratio of small arteries were greater in untreated SHR than in Wistar-Kyoto (WKY) rats. Whereas each of the antihypertensive treatments significantly reduced all parameters of blood pressure in SHR (except for metoprolol, which did not lower pulse pressure), only perindopril and isradipine significantly reduced the media/lumen ratio. The correlation between individual parameters of blood pressure and media/lumen ratio was stronger for pulse pressure than for systolic blood pressure, mean blood pressure, or diastolic blood pressure. These findings led Christensen to speculate that reduction of pulse pressure during antihypertensive treatment may be a factor in prevention of altered vascular structure during chronic hypertension.

Christensen’s findings have important implications in relation to determinants of vascular structure. Alterations of vascular structure during chronic hypertension have been linked to a variety of determinants including intravascular pressure, neural factors, humoral agents, and genetic factors. Of the various factors that may contribute to vascular structure, one might assume that arterial pressure per se would play an especially important role. With respect to mean arterial pressure, however, the evidence has not been convincing. Although treatment of hypertension has been shown to reverse medial hypertrophy in several vascular beds including aorta, mesentery, kidney, cerebrum, and muscle, the degree of reversal often has not matched the degree of reduction in mean arterial pressure.

In contrast to mean arterial pressure, there is a growing body of evidence that pulse pressure may play an important role in alterations of vascular structure during hypertension. For example, both hydralazine and cilazapril, an angiotensin converting enzyme inhibitor, prevent hypertrophy of cerebral arterioles in stroke-prone SHR (SHRSP). Hydralazine and cilazapril also are equally effective in reducing pulse pressure in cerebral arterioles of SHRSP, although hydralazine is less effective in reducing mean arterial pressure.

Ligation of the internal carotid artery has similar effects to those of hydralazine on pressure and wall mass of cerebral arterioles. Carotid clipping prevents hypertension and normalizes pulse pressure, but not systolic pressure and mean pressure, in pial arterioles of SHRSP. Furthermore, there is not a significant correlation among cross-sectional area of the vessel wall and systolic pressure and mean pressure in sham and clipped pial arterioles of WKY rats and SHRSP. There is, on the other hand, a strong correlation between cross-sectional area and pulse pressure.

The possibility that pulse pressure may be a determinant of vascular structure is supported by studies of large arteries and vascular cells in tissue culture. Coarctation of the thoracic aorta in monkey and dog reduces pulse pressure, but not mean arterial pressure, distal to the coarctation. The reduction in pulse pressure is associated with decreased motion of the aortic wall and a reduction in DNA and collagen content. In vascular smooth muscle that is grown in culture, DNA synthesis and rate of growth are greater in cells that are subjected to cyclic stretching than in cells that are grown under static conditions.
conditions. These findings suggest that alterations of cyclic stretching of smooth muscle may influence cellular and extracellular components in the vessel wall and affect vascular growth.

The results presented by Christensen support the hypothesis that pulse pressure may be a determinant of vascular structure. His findings provide an important step forward in our efforts to understand factors that influence vascular structure. In addition, the findings serve to emphasize the need for further work in this new area to elucidate the mechanism by which pulse pressure influences vascular growth and to clarify the extent to which it acts as a stimulus of altered vascular structure during chronic hypertension.

References

Key Words: chronic hypertension, pulse pressure, blood pressure, hypertrophy
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Hypertension. 1991;18:728-729
doi: 10.1161/01.HYP.18.6.728

Hypertension is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0194-911X. Online ISSN: 1524-4563

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://hyper.ahajournals.org/content/18/6/728.citation

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