Proximal Aortic Diameter and Aortic Pressure-Flow Relationship in Systolic Hypertension

To the Editor...

Mitchell et al promote a controversial view that elevated pulse pressure in systolic hypertension of older subjects is attributable to decreased diameter and elevated characteristic impedance (stiffness) of the ascending aorta. They further propose that drugs such as omapatrilat can reduce aortic stiffness directly and thereby provide effective treatment of systolic hypertension.1,2 The prevailing view is that systolic hypertension is largely due to early return of wave reflection, is associated with degeneration and dilation of the proximal aorta, and is best treated by reduction in peripheral wave reflection by vasodilator drugs.3,4

The authors did not provide measured aortic diameter or aortic pressure/flow relationships, yet they referred to both in the title of their article. They used surrogates of aortic diameter and pressure. Although their protocol entailed ultrasonic measurements of flow velocity and diameter in the ascending aorta for calculation of volumetric flow (their Tables 2 to 5), the actual measured diameter was not stated. Instead, a convoluted process was applied whereby carotid (not aortic) pressure was related to aortic volumetric flow for calculation of “aortic” characteristic impedance; this was then compared with carotid/femoral pulse-wave velocity using the Waterhammer formula to obtain a value of “effective aortic diameter.” The Waterhammer formula is only valid in a reflectionless system and must have pressure and flow measured at the same site and pulse-wave velocity measured locally.3 Given the errors inherent in indirect measurements and the theoretic requirement to measure impedance and wave velocity at the same site, one wonders why such a complex indirect method was applied when aortic diameter had to be measured directly in the first place.

Mitchell et al stressed reduction of effective aortic diameter in hypertensive patients. Their patients (78 male, 50 female) and controls (19 male, 11 female) differed markedly in size. When aortic diameter was expressed in relation to body surface area as in the National Institute of Aging studies of Lakatta;4 values were similar (1.48, 1.41, 1.45, and 1.43 cm/m², respectively) and within the normal range.

We favor the existing view, which is based on a host of direct measurements in large cohorts by different groups, and which conforms to mechanical principles and large epidemiological studies. We also favor scaling of body size by comparing pressure to pulsatile flow velocity so that characteristic impedance can be directly related to pulse-wave velocity as an index of arterial stiffness.3

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Response

We agree with O’Rourke et al that our recent publications1,2 challenge the view that the aorta represents a passive elastic structure that undergoes accelerated, irreversible wall stiffening and dilation due to excessive mechanical fatigue in hypertension. We have shown that aortic stiffness, as assessed by characteristic impedance (Zc), is elevated in hypertensive subjects2 and is reduced after a relatively brief course of treatment with omapatrilat, indicating that aortic stiffening is by no means completely irreversible.1 Our recent paper3 further suggests that accelerated age-related degenerative dilation of the proximal aorta is an unlikely explanation for increased pulse pressure (PP) in systolic hypertension. This important new concept is further supported by two large independent population studies that have shown an inverse relationship between PP and aortic diameter.3,4 We agree that premature wave reflection contributes to elevated central PP in hypertension; however, central augmentation is affected by factors other than reflected wave timing, such as gender, height, heart rate, peripheral resistance, level of ventricular function, and amplitude of the forward wave.

Carotid tonometry provides a close surrogate of pressure in the central aorta, whereas left ventricular outflow tract (LVOT) diameter and flow velocity provide an accurate measure of pulsatile aortic volume flow.3 Therefore, noninvasive estimation of aortic input impedance from carotid pressure and LVOT volume flow provides a valid assessment of the pressure-flow relationship in the proximal aorta. Zc calculated from velocity rather than volume flow is essentially pulse wave velocity (PWV). Such an approach underestimates the dramatic effects of diameter on impedance to pulsatile flow and ignores the fundamental importance of diameter change as an adaptive mechanism in arteries. We focused on “effective diameter” in our article because, like Zc, this variable represents the spatially and temporally averaged properties of the geometrically complex proximal aorta.

We agree that body size must be considered when interpreting Zc and diameter. Therefore, as suggested by O’Rourke et al, we evaluated central PP, measured LVOT diameter, Zc, and carotid-femoral PWV in models adjusted for age, mean arterial pressure, and body surface area. PP and Zc remained higher and LVOT diameter remained lower in hypertensive subjects (and women), whereas PWV did not differ in these models (Figure). These data favor the new view that increased PP in systolic hypertension is associated with an abnormal pressure-flow relationship (Zc) that is partially attributable to a relatively smaller aortic diameter.

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