A Reduced Elastic Modulus of Vascular Wall Components in Hypertension?

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In an elegant study, Hayoz and colleagues1 have demonstrated two features of a conduit artery in the vasculature of essential hypertensive patients. First, that the diameter of the radial artery of essential hypertensive patients is similar to or greater than that of well-matched, normotensive control subjects. Second, if compared under conditions of similar transmural pressure, the compliance and distensibility of the artery (for definitions see "Appendix") are not decreased in the essential hypertensive patients. The finding that the diameter of this conduit artery is normal or increased in essential hypertension confirms a number of previous reports,2 in particular those concerning the brachial artery. However, the results reported by Hayoz et al1 seem particularly clear-cut, for the measurements have been made with a resolution (based on signal averaging) of 1 μm. The finding that compliance and distensibility characteristics of conduit arteries are not decreased in essential hypertension is more controversial.2-5 Most previous measurements in vivo of arterial compliance have, however, been made indirectly on the basis of previous measurements in vivo of arterial compliance have, however, been made indirectly on the basis of measurements of pulse-wave velocity with the classic Moens-Kortewig equation for tubes with perfectly elastic walls.6 By contrast, Hayoz et al1 measured compliance by determining the time course of the diameter change throughout the cardiac cycle and plotting this against the corresponding pressure change, measured with a finger plethysmograph, to obtain a direct assessment of the diameter-pressure relation and thus of compliance and distensibility.7 The experimental basis for the finding seems therefore strong, and Hayoz et al1 obtained further support for their finding by also showing that in the carotid artery of spontaneously hypertensive rats (SHR), compliance and distensibility were similar to or greater than those of arteries from normotensive Wistar-Kyoto (WKY) rats if compared at the same transmural pressure. The implication is therefore that the mechanical properties of conduit arteries are not functionally altered in hypertension; this editorial comment will consider briefly the consequences of this implication regarding vascular development in hypertension.

Given that hypertension is a disease characterized by an increased peripheral resistance, the lack of narrowing in conduit arteries is perhaps surprising. The unexpectedness of the result is, however, dependent on how one views the development of the vasculature in hypertensive individuals. The results would indeed be remarkable if the cause of hypertension were due primarily to an abnormal constriction of the vasculature. If, however, the primary cause of hypertension is external to the vasculature, then the increase in resistance associated with hypertension will correspond to the functional requirements, and only the resistance vessels will be narrowed. By contrast, since the conduit arteries do not contribute substantially to peripheral resistance, one can speculate that the major determinant of conduit artery diameter is the flow requirements.8,9 Thus, since cardiac output is normal in hypertension, a normal conduit artery diameter might actually be expected. To a certain extent, therefore, the finding provides support for the thesis that hypertension is not due primarily to an abnormality in the vasculature, but that the abnormalities reported in the vasculature of hypertensive individuals are secondary responses associated with the increased blood pressure.10,11

The factors determining the conduit artery diameters seen by Hayoz et al1 and other investigators2-4 are not clear, since the relative contributions of smooth muscle activity and arterial structure have not been determined. It would be of interest in future studies to determine the artery diameters under relaxed conditions as, for example, could presumably be done with the technique of Hayoz et al for the radial artery by induction of reactive hyperemia.

The second finding that the compliance and distensibility characteristics of the arteries from the hypertensive individuals were normal is perhaps less expected. Here, Hayoz et al1 make the important point (a point also made by Armentano and colleagues4 in a recent article) that comparison of mechanical properties is meaningful only if the measurements are made under identical conditions, in this case equal transmural pressures. Thus, since vascular compliance and distensibility decrease with increasing intravascular pressure, previous findings that vascular compliance and distensibility are reduced in hypertension are probably due more to hypertensive individuals having higher intravascular pressures than to any difference in the compliance or distensibility characteristics of the vessels. However, although the finding of Hayoz et al1 that conduit artery compliance and distensibility are, at equal transmural pressures, normal in hypertensive individuals, this does not necessarily imply that the intrinsic mechanical properties of the artery are normal. First, as already mentioned, the measurements were made under resting

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conditions, where the level of smooth muscle activity is unknown; the situation could be different, for example, under relaxed conditions. Second, although from a functional point of view the compliance and distensibility of the vessels may be normal, as discussed below, this does not necessarily imply that the mechanical properties of the material in the vessel wall are normal.

The reason why information about vessel compliance and distensibility does not provide direct information about the mechanical properties of the material in the vessel wall is that information is also needed about the wall thickness. The technique used by Hayoz et al. did not permit measurement of the vascular wall thickness. However, given the increased intravascular pressure, it is almost certain that this was increased in the hypertensive individuals. This leads to the perhaps unexpected conclusion that the normal compliance and distensibility can only be achieved if the elastic modulus (a measure of elasticity independent of geometry, here defined as the incremental elastic modulus, see “Appendix”) of the wall components is reduced. One can therefore speculate that the adaptation of the vascular wall to the increased intravascular pressure likely includes not only a thickening of the vascular wall, but also a reduction in the elastic modulus of the wall material.

Support for this possibility comes from animal experiments, in which the mechanical and morphological properties of small arteries have been examined in detail. Baumbach et al. examined cerebral pial arteries in adult stroke-prone SHR (SHRSP). They found that for a given transmural pressure, the diameter of the pial arteries, both under resting conditions and when relaxed, of SHRSP was decreased compared with those of WKY rats. Thus, in contrast to the finding of Hayoz et al. concerning the carotid artery, in these small arteries the lumen diameter for a given transmural pressure was decreased. Nevertheless, the elastic modulus of wall material was decreased. This finding is confirmed by vitro results from my own laboratory concerning a variety of small arteries from SHR under relaxed conditions, including cerebral small arteries, which showed that the elastic modulus of the wall materials for a given wall stress (see “Appendix”) was decreased in the vessels from the hypertensive animals compared with normotensive controls.

Thus, although there may be differences between large and small arteries as to whether the lumen is increased or decreased in hypertension, a reduction in the elastic modulus of the vascular wall materials may be a general abnormality of the hypertensive vasculature. The mechanism involved in this reduction is not known, but it is presumably due to either an alteration in the mechanical properties of the individual wall components or an alteration in the proportions of these components in the wall. It is also possible that the reduction represents a degenerative process. In any event, as has been emphasised by Baumbach et al., this reduction in elastic modulus of the materials constituting the vascular wall would be a means by which the vasculature can functionally maintain its distensibility characteristics despite the relatively increased wall thickness required by the increased intravascular pressure.

It should be emphasized that although the human data of Hayoz et al. imply indirectly that the elastic modulus of the conduit artery wall material may be decreased, the direct experimental data come only from small arteries in the animal studies mentioned. To obtain direct information from the human vasculature, it is to be hoped that further refinement of the technique used by Hayoz et al. will allow determination of vascular wall thickness and thus direct determination of the elastic properties of the wall material. Furthermore, it would clearly be desirable to extend the technique to the analysis of smaller vessels, studies that could be supplemented by in vitro analysis of small arteries from essential hypertensive patients.

Appendix

The literature contains many, often conflicting, definitions of vascular elastic parameters. The following is a brief summary of the terms used here (see References 2, 17, and 18 for further information).

Compliance (C), change in vessel diameter (Δd) for given change in intravascular pressure (Δp), i.e., C=Δd/Δp. Some authors (e.g., Hayoz et al.) define compliance in terms of the change in vessel cross-sectional area, A = πr²/4, i.e., CA/Δp.

Distensibility (D), fractional change in vessel diameter (Δd/Δp) for given change in intravascular pressure (Δp), i.e., D=(1/d)×(Δd/Δp). As for compliance, some authors define distensibility in terms of the fractional change in vessel cross-sectional area, i.e., D=(1/A)×(ΔA/ΔAp).

Wall tension (T), circumferential force in wall per unit vessel length.

Wall stress (σ), wall tension/wall thickness.

Incremental elastic modulus (E), increase in wall stress (Δσ) for given fractional increase in diameter (Δd/Δp), i.e., E=Δσ/Δd. Note that unlike compliance and distensibility the elastic modulus is an intrinsic property of the wall materials, independent of vessel geometry. Note also that, due to viscous losses, the elastic properties of the vascular wall are time dependent. Thus, values of compliance and distensibility will in general be smaller where these are measured at high rates of change of intravascular pressure or diameter. The converse will be the case for elastic modulus.

References

8. Thoma R: Untersuchungen über die Histogenese und Histomechanik des Gefassystems. Stuttgart, Germany, Enke, 1893
17. Dobrin PB: Mechanical properties of arteries. Physiol Rev 1978;58:397–460

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