Localization of Vascular Resistance Changes During Hypertension

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An increase in the vascular resistance of virtually every organ system is a well-known characteristic of hypertension, as modeled by spontaneously hypertensive rats (SHR) and renovascular hypertensive rats. However, the most important issue may be where in the macrovasculature and microvasculature of a given organ the greatest increase in resistance occurs. The relevance of this issue can be considered from two perspectives. First, if a limited region of the vasculature in each organ is responsible for the bulk of the increased resistance, determining which region is predominantly involved would allow definitive evaluation of the abnormal morphological and behavioral characteristics. Second, if the overall arterial vasculature is relatively uniformly involved in elevating the vascular resistance, a general cellular or control system abnormality of the vessel wall potentially can be identified. In effect, the question to be answered is whether an isolated or generalized vascular problem contributes to the development and maintenance of hypertension.

The process of determining the mechanics of increased vascular resistance at the microvascular level during hypertension has taken a variety of approaches. The anatomical approach has been to evaluate morphological characteristics of the vessel wall that might predispose the vasculature to vasoconstriction. In vivo observation of almost every major vascular bed has been used to determine if and where in the vasculature there is an unusual amount of actively or passively induced vasoconstriction. An approach that has received a great deal of attention since described by Hutchins and Darnell is the temporary and permanent closure or rarefaction of arterioles as a means of increasing vascular resistance. All of these approaches have yielded and will continue to yield valuable insight into the hypertensive process. However, unless the percentage of the total resistance occurring at a given level of the microvasculature is known, the importance of a particular morphological or control abnormality is difficult to relate to the increase in vascular resistance.

To assist in the problem of relating vessel morphology and behavior to vascular resistance, measurements of pressures within the microvasculature have been made. This type of data is useful to determine the physical forces placed on the vessel wall as well as the vascular sections associated with an unusually high drop in pressure, which is indicative of a high resistance.

Microvascular pressures in the kidney, various skeletal muscles, small intestine, and cerebral cortex have been measured in SHR. In each of the organs studied, microvascular pressures generally are thought to be grossly elevated in all but the precapillary arterioles, capillaries, and venules. In these latter vascular sections, the microvascular pressures are either close to normal or slightly elevated. In skeletal muscle, small intestine, and cerebral tissue, the fall in pressure from the largest arterioles to the smallest arterioles is typically two to three times greater than normal in SHR. Meininger et al. have also reported somewhat comparable circumstances of near normal pressures in the small arterioles of skeletal muscle in rats with renovascular and deoxycorticosterone-salt hypertension.

Although a higher than normal resistance in the small arterioles may help to minimize increased capillary pressure during hypertension, these small arterioles are not
responsible for the major increase in total organ vascular resistance. In general, mean arterial pressure and vascular resistance in adult SHR are about 50% higher than normal. The small arterioles in the organs just discussed control only 10 to 15% of the total normal resistance and not more than 20 to 25% of the resistance in the hypertensive animal. Therefore, the small arterioles contribute only 10 to 15% of the increase in vascular resistance seen in SHR. To account for the remaining 35 to 40% increase, the resistance of small arteries and larger arterioles must be considered. These vessels control about 60 to 70% of the vascular resistance in every organ tested thus far; consequently, an increase in resistance at this level would have a formidable influence on total vascular resistance. If the blood flow in these vessels is near normal or less than normal at the time the pressure measurements are made, the large pressure drop across the small arteries and larger arterioles predicts a high resistance of these vessels during hypertension.

In vivo measurement of the inner diameter of the larger arterial vessels of the intestine and brain has demonstrated vasoconstriction that would be consistent with a high resistance of these vessels. In the skeletal muscle vasculature of the SHR, however, the largest arterioles available for study typically have been reported to have a diameter equal to or slightly larger than normal. Therefore, the higher than normal resistance of the small arteries and larger arterioles may be caused predominately by vessels upstream from those that can be observed during in vivo studies, or the increased resistance may be caused by factors other than vessel radius.

Borders and Granger have devised a new approach to help support the contention that the primary site of increased resistance in hypertension is at the level of large arterial vessels. This elegant approach is based on determining where the greatest power dissipation, and therefore the highest resistance, occurs in the cremaster muscle vasculature. The calculation of power dissipation is a function of the vessel segment length, blood viscosity, and square of the flow velocity for individual vessels. This method has a variety of benefits because power dissipation takes into account all forms of resistive properties, not just vessel radius. Furthermore, one need not know or make assumptions as to what constitutes the actual resistance properties of cellular and fluid flow through a tapered and branched distribution system. From a technical standpoint, the measurement of power dissipation is much easier and less invasive than is the use of micropipettes needed to measure microvascular pressure because only the vessel length and red blood cell velocity need be measured.

By comparing the power dissipation of selected vessels with the total power dissipation across the entire vasculature, Borders and Granger were able to conclude that the larger arterioles were most influential in increasing resistance. Although their analysis did not include the small arteries that precede the largest arterioles because of anatomical constraints, in terms of the resistance changes within the microvasculature itself, the analysis indicated that as few as 10 vessels could account for most of the increased resistance of the hypertensive microvasculature.

Even though available data are interpreted to indicate that the small arteries and larger arterioles are a major site of increased vascular resistance in SHR, this process may occur by default and may not be due to a particular or unique pathological process or adaptation of these vessels. For example, the resistance of all sections of the arterial vasculature may prove to be elevated proportionately, or all arterioles may prove to have similar forms of adaptation and pathology. However, by virtue of their normally high percentage of the total resistance, the larger vessels will contribute more to the increased resistance than will the smaller vessels. This could occur even if the same set of mechanisms influences all vascular sections equally.

If future studies continue to implicate the small arteries and larger arterioles as the most important functional section of the vasculature during hypertension, it may be advisable for research to be directed to the physiology, anatomy, and pharmacology of these vessels. Furthermore, since the small arteries and larger arterioles of the brain, intestine, and skeletal muscle are about equally implicated in elevation of resistance in each organ, a common mechanism or set of mechanisms for a variety of organ system vasculatures may be responsible for the vascular changes that sustain the peripheral vascular component of hypertension.
References


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