INTRAVITAL STUDIES ON PIAL VESSELS IN SHR AND WKY

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The hemodynamic consequences of an increased vessel wall thickness in chronic hypertension have been much debated. Whereas some groups consider such changes to be of utmost importance for the increased vascular resistance and for the maintenance of hypertension (see e.g. Folkow 1978) other have claimed that there is little evidence for this hypothesis in vivo. Thus, Hutchins and Danell (1974), Bohlen et al. (1977) and Henrich et al. (1978) reported that the lumen diameter of cremasteric and mesenteric arterial vessels are larger in SHR than NR in vivo and thought that difference in vessel density accounted for the increased vascular resistance. We have earlier reported an increased vessel wall to lumen ratio in pial arteries in SHR (Nordborg & Johansson 1979, 1980) as well as reduced cerebral blood flow during maximal vasodilatation (Johansson & Nilsson 1979). Providing SHR and WKY had the same number of vessels this would indicate a decreased internal radius. The present study was performed to analyze the diameter of pial vessels during normo- and hypercapnia in SHR and WKY and evaluate vessel density.

Rats were initial anesthetized with pentobarbitol and mechanically ventilated with air. A closedcranial window was inserted in the parietal region on the left side. The internal diameters of pial arteries and veins were continuously observed through a Leitz intravital microscope. The fine branches of the middle cerebral artery and of pial veins were measured by aid of a multichannel videangiometer. Single experiments were recorded on a pen-writer and on a videotape. Hypercapnia was induced step-wise until maximal dilatation. At the end of the experiments yohimbine and phenoxybenzamine were given to evaluate the possible sympathoadrenergic influence on vessel tone during hypercapnia.

Initial mean arterial pressure (MAP), MAP during maximum hypercapnic vasodilatation and MAP after yohimbine and phenoxybenzamine were 172 ± 9, 183 ± 9, 180 ± 9 and 147 ± 18 mm Hg in SHR; corresponding values for WKY were 103 ± 5, 127 ± 7, 125 ± 10 and 106 ± 12 mm Hg. Resting pial arterial diameters of SHR (55 ± 1 μm, n 53) were significantly smaller (p<0.001) than in corresponding arteries of WKY (87 ± 1 μm, n 54). During hypercapnia the percentage increase in diameter was 54 % in SHR and 34 % in WKY; however, the absolute lumen diameter during vasodilatation was significantly smaller in SHR (84 ± 2 μm compared to 117 ± 5 μm in WKY; p<0.001). No significant further dilatation was seen in either group after adrenoceptor blockade.

Also veins had smaller lumen diameter in SHR than in WKY (71.6 ± 3.7 μm compared to 97.2 ± 9.7 μm). However, when the material was divided according to vessel sizes the difference for veins was only significant for veins > 100 μm. The response of pial veins to hypercapnia was not significantly different in SHR and WKY.

The present study thus show a smaller lumen diameter in pial vessels of SHR in vivo compared to WKY. As to the arteries, the smaller resting diameter could be related to an autoregulatory vasoconstriction. However, the smaller diameter in SHR during vasodilatation (hypercapnia + alpha adrenoceptor blockade) is consistent with the concept that hypertensive vascular hypertrophy encroaches on the lumen. The prompt response to hypercapnia of pial vessels in SHR indicates that there is no decreased vessel reactivity to hypercapnia. The larger % increase in diameter of pial arteries in SHR is what would be expected out from Folkow's hypothesis as to the hemodynamic consequences of an altered media-lumen ratio in SHR.

A preliminary analysis showed no significant difference between SHR and WKY as to pial vessels density or branching pattern.