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Possible Involvement of an Impaired Baroreflex Mechanism but not the Renin-Angiotensin System and Vasopressin in the Enhanced Pressor Responsiveness to Physostigmine in Spontaneously Hypertensive Rats

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1. Effects of physostigmine on heart rate, mean arterial pressure (MAP), plasma renin concentration (PRC) and vasopressin (AVP) release were investigated in spontaneously hypertensive (SHR) and Wistar-Kyoto (WKY) rats.

2. Physostigmine (100 μg/kg, i.a.) produced a greater and prolonged hypertensive response in the SHR than in the WKY.

3. Heart rate was increased by physostigmine in SHR rats while it was unchanged in the WKY.

4. PRC was unchanged or even slightly decreased in these animals when MAP was increased by physostigmine.

5. An AVP pressor antagonist did not attenuate the pressor and cardiac effects of physostigmine in these animals.

6. These data indicate that an impaired baroreflex mechanism or a different mode of sympathetic neuronal activation by physostigmine through the central mechanism appears to be contributory, at least in part, to the enhanced pressor responsiveness in the SHR.

7. The renin-angiotensin system and AVP do not appear to be involved in the enhanced pressor responsiveness to physostigmine in SHR rats.

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