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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

Koichiro Kawashima, Yuko Miwa and Kazuko Fujimoto 川島紘一郎,三輪裕子,藤本和子

- 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  $\mu$ g/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.