Conditions for secretion of pressor amounts of vasopressin was studied in normal and spontaneously hypertensive rats in the conscious state. The characteristic, almost stepwise lowering of arterial pressure on injection of a vasopressin antagonist was used to detect vasopressin secretion in pressor amounts. In normal rats the necessary and sufficient condition for secretion of vasopressin in pressor amounts was found to be elimination of both pressure and volume receptor impulses, which might take place in nature when the sympathetic nervous system has failed. These impulses have inhibitory influence on a structure in the lower medulla oblongata which sends excitatory signal to vasopressin secreting cells in the hypothalamus. Elimination or marked diminishment of pressoreceptor impulses can be achieved by sinoaortic denervation or spinal transection at C8, which is considered to lower arterial pressure below the threshold of most pressoreceptors. Volume receptor impulses can be eliminated by cervical vagotomy and diminished greatly by ganglion blockade, or alpha or beta adrenoceptor blockade. Alpha blockade is considered to be effective in decreasing venous return. Beta blockade is supposed to lower the sensitivity of cardiac volume receptors by relaxing heart muscles. Ganglion blockade seems to dilate capacitance vessels and relax heart muscles.

In SHR, unlike in normal rats, secretion of vasopressin in pressor amounts could be induced by elimination of pressoreceptor impulses alone, intervention to volume receptors being unnecessary. Thus, for secretion of pressor amounts of vasopressin, sinoaortic denervation + vagotomy or ganglion blockade were necessary for normal rats, while sinoaortic denervation alone was sufficient in SHR. Likewise, spinal transection + ganglion blockade were necessary for vasopressin secretion in pressor amounts in normal rats, while only spinal transection was in SHR.

It may be said that SHR tends to secrete pressor amounts of vasopressin more easily than normal rats. When conditions necessary for secreting pressor amounts of vasopressin in normal rats, i.e. sinoaortic denervation + ganglion blockade or spinal transection + ganglion blockade, were met in SHR, greater amounts of vasopressin were secreted in SHR than in normal rats.

After spinal transection and ganglion blockade, arterial pressure is still higher in SHR than in normal rats (Jpn. J. Physiol. 27: 801, 1977). One might think that this indicates the presence of non-neural hypertensive factors in SHR: spinal transection interrupts the influence of the so-called vasomotor centers on the circulatory system and ganglion blockade is considered to eliminate the effect of the activity of spinal sympathetic cells. However, after further injection of the vasopressin antagonist in addition to spinal transection and ganglion blockade, arterial pressure was equalized between normal and spontaneously hypertensive rats.

In conclusion, since secretion of pressor amounts of vasopressin occurs when the sympathetic nervous system fails and since the secretion is more abundant in SHR than in normal rats, it obscures the result of experiments to observe the effect of elimination of sympathetic activity on the hypertension in SHR. Taking into consideration this interference from the vasopressin secreting mechanism, the hypertension in SHR is after all largely "sympathetic".